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Composed and Presented by

DAVID DALE LOGAN

M. B., Ch. B. Glasg.,

1900.

Auchterhall,
NEWMAINS,
Lanarkshire.

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A STUDY OF PUERPERAL ECLAMPSIA - ITS MOST RECENT ETIOLOGY

AND TREATMENT.

P R O L O G U E.

The study of puerperal eclampsia is a most interesting one to the medical practitioner - not only because it is one of the most formidable complications of pregnancy and labour that he has at times to face,- threatening the life of the mother and her child and alarming the onlookers by the hideousness of its manifestations,- but on account of the modern conception of its etiology, which regards it in the light of a toxæmia, and also in view of the fact that a study of the causes raises the attractive hypothesis that the so-called minor symptoms of pregnancy,- vomiting, salivation, constipation, irritability of temper, etc.,- which are so common as to be considered almost normal, are nothing more nor less than evidences of a slight toxæmia.

Notwithstanding the fact that an enormous amount of work has been done, regarding the chemical and microscopical aspects of the subject, within recent years, analysis of the urine and blood by more correct and more scientific methods, and investigation of the pathological changes found in the organs of the mother and child,- there is still a considerable divergence of opinion concerning its essential etiology; and, although since it was recognised that eclampsia was due to a condition of toxæmia, we have made considerable strides towards the adoption of a rational mode of treatment, nevertheless, there is perhaps no condition, the treatment of which has produced such a bewildering array of divergent and conflicting opinions, as has the disease under consideration.

D E F I N I T I O N.

Eclampsia is a condition which appears during pregnancy, labour, or the puerperal state, characterised by disturbances of the nervous system,- the most prominent of which are the epileptiform convulsions,- which are attended by rise of temperature, loss of consciousness, and the development of coma, as well as a certain amount of injury to the kidneys (caused by the action of certain toxic substances, of unknown nature and somewhat uncertain origin), the injury in question being manifested by the appearance in the urine of albumen.

This definition may be taken as a good working one for a typical case of eclampsia, but it must not be taken as covering all cases of eclampsia; for, as we shall see later on, there are distinct varieties of this disease, and its signs and symptoms, moreover, are by no means constant in their appearance.

FREQUENCY OF ECLAMPSIA AND THE PERIODS IN WHICH IT IS FOUND.

In arriving at an estimation regarding the frequency of eclampsia, no author's statistics should be taken into consideration, unless he produces a sufficient number of cases, which cover a certain number of years.

We find that its frequency is variously estimated by writers, some putting it as low as 1 in 500,- e.g., Veit, who, curiously, in 1896, in his own cases, placed it as high as 1 in 166. Vinay put it at 1 in 250 or 260; Anvard in 330; while Leishman, taking the average of English and Continental practice, considers that it

appears about 1 in 350. Charpentier, in an analysis of 259,000 labours in France, found 730 cases of eclampsia, or 1 in 350; and Schreiber, among 42,600 in Vienna, found 137 cases, or 1 in 311. In all probability, however, about 1 : 300 or 1 : 350 is the usual proportion. According to Norris and Dickinson, the frequency, as reported in the Philadelphia Board of Health Returns, was variable, some years producing as many as 1 in 500, while in other years only 1 in 500 was the number found; and this is what is generally found in medical practice in the country - some obstetricians having a run of eclamptic cases in a short period, and then perhaps not seeing another case for years.

In looking over the literature of eclampsia, I find a greater percentage of this disease in hospital than in private practice. This would be explained by the fact that (1) many primiparae, among whom the disease is very much more common than in multiparae, find their way into hospital; (2) that many multiparae of the poorer classes when they find themselves in that alarming state of health, as frequently happens before the superintention of the fit, would apply to the hospital for admittance; while (3) many cases, where it developed, would be sent to hospital for treatment.

Several observers have drawn attention to the fact that eclampsia is more frequent at certain times than at others; that it is more often found in certain districts than in others; and that it also greatly varies in the severity of its attacks. For example, we find that in two districts where the same treatment is adopted the death-rate in the one is very much higher than in the other. To prove that great variations may be found in different parts of a country, Schmitt has published statistics, comprising the number of cases of eclampsia seen in the various lying-in institutions of seven large towns in Germany, in which he shows that the death-rate in one town was as high as 1 in 49, while in another it was as low as 1 in 1700. In Tübingen, where the latter figures were observed, one of the physicians to the institution affirmed that he had not seen a single case during twelve years. It is very remarkable that in Wurtemberg eclampsia is very rarely seen. Doderlein gives 1 case in 3561, these figures being taken from 644,567 births. The principal beverages in this country are their sour wines, which contain a considerable quantity of tartrates, citrates, etc., and it is suggested that these, which we know are turned into carbonates in the blood and act as excellent diuretics, may be the reason for this remarkable immunity against eclampsia. Buttner, in his statistics of this disease in the province of Mecklenberg Schwerin, found 1 case of eclampsia in 600 - 650 births.

TABLE I.

Author.	No. of Births.	Eclampsia Cases.	Frequency.
Doderlein.	644,567	181	1 in 3561
Galabin.	23,000	27	1 " 833
Braun.	24,000	52	1 " 461
Schanta.	134,345	342	1 " 380
Charpentier.	259,000	730	1 " 350
Schreiber.	42,600	137	1 " 311
Herzfeld.	96,000	463	1 " 200
Bidder.	60,515	455	1 " 133
Goldberg (Dresden).	10,718	81	1 " 133
Braun.			
Veit (Germany).	149,336	905	1 " 166
Jelett (Dublin).	20,000	56	1 " 357
Löhlein (Germany).	15,328	325	1 " 160
Green (Boston).	2,300	38	
British and Continental Collections by Jelett.	227,000	635	1 " 357.5

The older observers (Ramsbotham, Lusk, etc.) held that puerperal convulsions were much more frequent in hot weather than in cold, and especially during those periods when the atmosphere was charged with electricity. This latter condition seems to specially weigh with them, for we find one author stating that "the electrical state of the air on the approach of a storm has often served to bring on a convulsive fit". Perhaps the only reason why they laid so much stress on the electrical condition of the air was because they looked upon eclampsia

in those days as essentially a nervous disorder, and there was also in their minds a close relationship between nerve energy and electricity. Oldhausen has also pointed out the tendency for eclampsia to appear at special periods of the year. In Berlin, for example, he found more between the months of September and February than at any other time, and more between September and May than May to September; sometimes the cases were so frequent as almost to suggest an epidemic. Schroeber, in fact, holds that one may have an epidemic of puerperal eclampsia during raw damp weather, but the consensus of opinion is altogether against this view. Buttner, in contradistinction to Oldhausen, says that he found most of his cases occur between May and October. Furthermore, Schreiber found that most cases were encountered in August and July, and fewest in November and February; while Bidder found most between March and May, and least from September to November.

It is recognised by all that cold damp weather and chills are certainly factors in the incidence of renal disease, but as yet we know very little regarding the relation of the weather to eclampsia. One cannot doubt, however, that any thing which, by interfering with the proper function of the skin, throws an extra load on the principal excretory organs, - the kidneys, etc., will have a certain influence in predisposing the patient to eclampsia. In Madras, Sturmer has observed that a larger number of the eclamptics are brought into hospital on dull and cloudy days, and he regards this as due to the skin not acting so well on these days.

FREQUENCY IN PRIMIPARAE AND MULTIPARAE.

All are agreed that eclampsia is much more frequently seen in primiparae. The percentage among these has been given by Durhssen as 84%; Schanta as 82.75%; Schreiber as 79.5%; and Oldhausen as 74%. Again, it is relatively more frequent in very young primiparae (that is in those over 34 years of age) than it is in women between these ages, although Winkel, however, found that old primiparae were only very slightly liable. Out of 195 cases, Durhssen found that 40.5% were either below 20 or over 30 years of age; and Oldhausen found 25% over 28 years. Two reasons for this increased liability to eclampsia of the old and very young primiparae have been given, - first, the small space in the abdominal and pelvic cavities causing increased intra-abdominal tension, and, secondly, the undue rigidity and resistance of the abdominal muscles and other parts. This may account for the fact, which is generally recognised, that eclampsia brings on labour more frequently in a primipara than in a multipara.

With regard to the frequency of plural pregnancy in eclampsia, Oldhausen in his cases found that 8% were plural, Winkel 11%, Durhssen 4.5%, and Schreiber 8.7%.

When we next consider the period in which eclampsia is most frequently encountered, we find that while most are agreed that eclampsia coming on after labour has been completed is comparatively infrequent, opinion is still divided concerning the frequency of its appearance before or during labour. It must be borne in mind, when considering their relative frequency, that the attack, often sudden and unexpected, occurs frequently in the last few weeks of pregnancy and that eclampsia very frequently induces labour, the onset of the convulsions being so closely followed by labour that they have been looked upon as synchronous. This question has considerable interest in relation to treatment, under which it will be further discussed later on. Kaltenbach says that it may appear towards the end of pregnancy, but that it is by far the most frequently found during labour; and Goldberg recently, in a careful scrutiny of 1120 cases, found that in 21% the disease appeared in pregnancy, in 55.5% in labour, and in 22.5% after it. Braun and Bailly, again, state that the order of frequency is pregnancy, labour, and after delivery. The amount of discrepancy of opinion regarding their relative frequency may be shown by stating that the number of cases occurring before labour has been put by well-known observers as low as 8%; while other, equally distinguished, - e. g., Oldhausen, have put it as high as 40%. The probability is that the number is generally put too low as regards the incidence of the disease before labour, and that certain of the cases, noted as occurring during labour, ought more properly to be looked upon as occurring before labour.

Reviewing the statistics of different observers, one may, roughly speaking, affirm that post-partum eclampsia occurs in 18.20%.

TABLE II.

Author.	Pregnancy.	Labour.	Puerperium.
Wiegert.	24%	52%	24%
Schanta.	13.5%	57%	29.5%
Schroeder.	19.62%	60.87%	20.25%
Winckel.	23%	60%	17%
Schreiber.	16.78%	62.04%	21.16%
Oldhausen.	40%	46%	14%
Goldberg.	25.9%	57.14%	16.8%
Moran.	49%	31%	20%
Green.	36%	22%	42%
Herman.	75%	16%	8%

It will be noticed, in table II., that Green puts it at 42%, but we must regard his experience as being exceptional.

While eclampsia may be found at any period during pregnancy, cases being on record as early as the twelfth day, by far the greater number occur after the sixth month, and especially between the seventh and ninth months. A case has recently been reported by Hirschmann where eclampsia was found in a girl who had not menstruated for four and a half months, while the uterus had attained the average size of the seventh month. The uterus was emptied of a large vesicular mole, in which there was no trace of a foetus. Oldhausen also has on record two cases of eclampsia associated with vesicular mole. Spiegelberg relates a case of eclampsia in extra-uterine pregnancy: in this case the fits ceased after the death of the foetus; and Mygrier has also reported an instance of eclampsia coming on in the false labour accompanying extra-uterine gestation.

In these cases of eclampsia occurring during labour it is generally found that the first stage ushers in the fits, although sometimes, when the first fit occurs, the head may be found bulging out the perineum.

In Tarnier's 52 cases -

1 case occurred at the 5th month. 14 cases occurred at the 8th month.
 5 cases " " 6th " 5 " " " 8½ "
 4 " " " 7th " 16 " " " 9th "

In one of my cases of post-partum eclampsia the fits came on ten hours after labour had terminated; and the rule as regards the puerperium seems to be that eclampsia very rarely develops after the fourth day, but usually within a very few hours of labour. Some cases have been reported where days elapsed before the fits occurred - although such cases as reported by Bailly, where the fits came on 25 days after labour, must be looked on as very exceptional.

SYMPTOMATOLOGY.

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The symptoms and signs of an eclamptic seizure present such a well-known and impressive picture to the trained observer that I shall not seek to portray it in detail here, contenting myself to briefly remarking upon a few of the more salient features, especially those which have a practical bearing on treatment. I may say here that the most vivid and best description of an eclamptic seizure I have read is that of Bailly, the distinguished French observer.

THE ECLAMPTIC SEIZURE.

The convulsion is epileptiform in character, and sudden in onset, although there are signs and symptoms often present, - for example, extreme restlessness, twitching of the face, which I shall discuss more fully presently, - which point to a convulsion being imminent. The fit is very rarely preceded by an aura such as we find so commonly in epilepsy. Cases have been reported where the patient has emitted a scream of terror before the fit came on, or has put up her hands to the head as if to shield it from the threatened blow. Oldhausen has reported a case where a patient uttered her husband's name, and then fell into a fit; and also two other cases where there was a distinct aura. Ramsbotham tells of a patient in whom the fit was preceded by an exclamation that the room was studded with diamonds.

In the convulsion there is first a tonic stage, wherein the face is white and contorted, and the muscles of the whole system ~~perhaps~~ in a state of spasm, and in which respiration is also suspended. This stage is very short in duration; and it is followed by the violent clonic contractions beginning with the twitchings of the face, which latter soon becomes livid and hideously altered in expression, while the muscles of the trunk and limbs are also violently convulsed. Great care must now be taken lest the tongue, which is forcibly protuded, be not lacerated by the violent grinding of the teeth. The breathing, after its temporary arrest in the tonic stage, becomes irregular and hurried, and is accompanied by laboured hissing sounds. Insensibility is complete during an attack, which generally lasts from one to three minutes. This is followed by the comatose stage with loud stertorous breathing. This may last for a longer or shorter period, depending on the violence of the attack. During a fit, involuntary passage of urine and faeces may occur.

PREMONITORY SIGNS AND SYMPTOMS OF ECLAMPSIA.

These require most careful consideration, as their early recognition permits of a treatment which may have considerable influence on this stage, and which, moreover, may so modify it that the patient may be able to reach full term and escape the dangers which the confinement incurs, without an eclamptic attack.

It is necessary in every case of pregnancy, especially in primiparae, to see that the various organs, - the liver, kidneys, etc., which have to do with the protection of the body against the invasion of toxins, from whatever source, - and also other organs, such as the kidneys, bowels, skin, etc., which have to do with the elimination of waste products and the like, - are in full working order. Undoubtedly, the best method of effecting this (if we could manage it always in general practice) would be the systematic examination of the urine of every pregnant woman in the last few months of pregnancy, - especially primiparae, - as it is in the urine that we often find the first danger signals of a break-down in both ~~our~~ lines of defence - the one which protects against, and the other which eliminates, the poisons - in the first place, by a quantitative estimation of the urea excreted by the kidneys in the 24 hours, and, in the second place, the presence or absence of albumen in the renal secretion. I shall deal with these two prodromal signs - the fall in the percentage of urea and the presence of albumen in the urine - in detail later on.

In considering any case of eclampsia, it will be found that, in nearly every instance, premonitory symptoms have been present, ^{but in some cases} to such a slight degree as not to have attracted attention from the patient, their existence only being ascertained ^{afterwards} by close-questioning the latter or her friends. Again, these signs and symptoms may be recognised hours, or perhaps days or weeks, before an attack develops. In some cases, however, the fit has developed without the slightest warning; and ^{where} ~~there~~ have ^{been} ~~reported~~ cases where the fit occurred shortly after the examination of the urine revealed not the slightest trace of albumen, and while the patients were not complaining in any way. Handfield-Jones reported a case where labour came on thirty minutes after the urine was found to normal in its contents; delivery being effected, the patient had fits, and the urine was distinctly albuminous.

One of the most constant signs of a prodromal character in puerperal eclampsia is headache. This may be frontal or unilateral, or even more localised to a certain spot, the patient describing it as like a nail in the head; or it may be occipital; while, again, the pain may radiate over the whole of the head. In one of my cases, the patient complained most of the pain in the back of the head, and this darted and shot into the neck. The pain complained of may be dull and aching in character, throbbing, or darting and shooting. It is usually worse on stooping or walking. The headache is often accompanied by attacks of giddiness, ringing in the ears, and nausea and vomiting; spots before the eyes, flashes of light, disturbances of vision, and, in some cases, even blindness.

Disturbances of vision may be complained of days, or even weeks, before the attack of eclampsia. This usually takes the form of blurred or indistinct sight, or the patient complains of not being able to use the eyes for any length of time, or declares that

objects appear to be of peculiar colours; or, again, there may be hallucinations of sight. Photophobia is sometimes met with, while diplopia is frequently encountered. Strabismus may develop, or colour blindness and temporary blindness are by no means uncommonly observed before a convulsion. Albuminuric retinitis is sometimes met with, and it is usually the result of acute nephritis. It does not follow in these cases that eclampsia will develop; but, where eclampsia has occurred, the course of the disease has often been more severe - some observers, indeed, holding that in every case almost of eclampsia where it is prominent the prognosis must be very grave.

Besides these, other anomalies of special sense may be met with, such as deafness and ringing in the ears; but some observers have noted, instead of this, a remarkably increased acuteness of hearing. Loss of memory or temporary aphasia are sometimes met with shortly before the appearance of the convulsion.

The patient complains of feeling ill, is very restless, sleeps badly at night, has terrifying dreams and nightmares, is exceedingly irritable and easily disturbed, while the slightest exertion gives rise to exhaustion. She may complain of numbness of the lower limbs, while in some cases dyspnoea is a prominent symptom, and this is always aggravated by the slightest exertion. In others, one may find great mental excitement, and, in a few, dread of impending trouble. A few cases have been reported where there was great pain in the back, shooting into the belly and down the legs. All these phenomena, however, occur in great variation in different patients; and, although we may find one or two of them present, in a pregnant woman, it does not follow that eclampsia must necessarily develop.

Another very important manifestation, and one which was complained of in four of my cases, is epigastric pain, or it may be better termed in certain cases epigastric distress. With regard to its appearance, Dührssen seems to consider it a very rare symptom; but this is quite different from the bulk of observers, who look upon it as being pretty general in its occurrence: Oldhausen lays great stress upon its importance. This pain may bring on an attack of vomiting, the vomited matter being frequently bilious in character. In some the pain is of a vague nature, and felt in and around the pit of the stomach; while in one of my patients the pain, which came on in paroxysms, was described by her as agonising, causing her to double up and cry aloud. It is interesting to note that this pain seems to be not unlike that complained of before an attack of diabetic coma. I have had two such cases in young men during the last three years, in both of whom the complaint was of excessive pain in the epigastrium just before the development of the characteristic coma.

Vomiting is seen, in the great majority of cases, at some time before the development of the fit: it is sometimes excessive. It was present in all my cases. Vinay declares that the patient is sure to have an attack of eclampsia if she complains of headache, with flashes of light, giddiness, and ringing in the ears, as well as numbness and tingling in the lower extremities.

Jaundice may be encountered in a few cases, and with it enlargement of the liver, which in these cases is often found to be tender on pressure or ~~hard~~ ^{hard} on percussion. Splenic enlargement has also been noted.

Albuminuria. Perhaps the most valuable premonitory sign we have clinically is the presence of albumen in the urine, in considerable and increasing quantities. In a certain proportion of cases, eclampsia has been noted without albuminuria; but, in reading the observations of the most experienced observers, I find this type of eclampsia to be far from common. When any of the above-mentioned premonitory signs and symptoms are found, on examination of the urine one may be almost certain of finding albumen; and the amount of the latter, moreover, forms a capital index of the severity of the toxicæmic disturbances.

Decrease in the Amount of Urea Excreted. This is one of the most reliable of the premonitory signs of eclampsia, and one that should never be ignored. It shows that not only the kidneys are not carrying on their work of elimination properly, but also that proteid metabolism is not being perfected by the liver. I shall have more to say about this sign when considering the chemistry of the urine in eclampsia.

Oedema. This frequent accompaniment of the pregnant condition is usually limited to the lower limbs: here it is probably due to pressure of the gravid uterus upon the veins returning from the legs. Sometimes the vulva may be greatly oedematous. Again, oedema may be general, and involve the whole body. Particularly significant are a swollen, dusky, bloated condition of the face and oedema of the upper arms. In these cases, examination of the urine will generally proclaim some renal disturbance; but, as a matter of routine, however, in all cases of oedema, - even if it only appears in the legs, - an examination of the urine should be made. Frequently the oedema of the face is only transitory, appearing in the morning after the woman has been asleep, and disappearing as the day wears on. Oedema of the face, limbs, or trunk, then, is a grave sign; and if, on examination of the urine, - which is very scanty in amount and high-coloured, - we find albumen, tube casts, and perhaps blood, and with it the patient complaining of headache and epigastric pain, she will certainly develop convulsions, unless prompt measures are forthwith adopted for its prevention.

The condition of the pulse is also a valuable indication of the state of the renal circulation. Should it be rapid, bounding, full, and accompanied by high arterial tension, an examination ought at once to be made of the urine.

It is rare, therefore, to find convulsions unheralded by some prodromal sign or symptom, slight though the same may be; and if, on finding these, frequent examination of the urine reveals the presence of albumen, or that the amount of urea is decreased to a marked degree, we ought to be on our guard.

CONDITION OF THE PATIENT AFTER AN ATTACK.

At the end of an eclamptic seizure the stage of coma develops; this may be slight and last only for a very short time, or it may be profound in character and prolonged in duration. This comatose condition is produced by the muscular spasms interfering with the circulation in the veins of the neck, which become swollen and turgid - resulting in cerebral congestion. After very slight attacks, the patient may be only in a state of semi-unconsciousness and ^{conscious} clear mentally, perhaps complaining of headache. Deep and incessant sighing is a condition sometimes met with between the fits, and it is nature's way of trying to aerate the toxic blood. Except in very bad cases, the coma is rarely complete, as the patient may be partially roused by shouting or shaking her; while, should the convulsions appear during labour, she may groan during the pains. But, if the fits recur frequently, it will be found that the patient usually continues in a state of more or less profound coma. The symptoms which are found in the premonitory stage, - such as headache and visual disturbances, - may be encountered after an attack. Even after only one or two convulsions, the patient is usually very dazed and bewildered-looking, and the return to consciousness is slow indeed; and, if in the interval labour has been terminated successfully, she has no recollection of anything that has happened meanwhile. Even after the patient has completely recovered from the attack and is convalescent, we generally find that the period succeeding the fit has been completely blotted from her memory. This temporary loss of memory is a marked feature of most cases. In ~~some~~ my cases of post-partum eclampsia, - when the patient regained her memory and senses completely, which was not until ~~six~~ or eight weeks after delivery, - I found that she had no recollection of anything that had happened ^{not of her confinement} six weeks before her confinement, although she spoke ^{to me} quite sensibly after it. The loss of memory in some cases may last for many months, or it may continue indefinitely after an attack of eclampsia. In most cases this loss of memory relates only to the most recent events or, as in my case, to a certain period before labour; but, in other cases, lengthened periods - perhaps years - have been erased from the memory of the patient.

Mental derangements are also found ~~after~~ an attack - these may be very slight, the patient becoming "peculiar", as her friends express it, or, again, insanity may be found, which also may last indefinitely. In other cases, where the coma was very marked and profound, the patient developed out of this condition maniacal symptoms.

RECURRENCE.

Eclampsia recurs in very few cases, and I have never observed it. Leopold has on record one case where it developed in three successive pregnancies; and, lately, Caraccio has published a case where, out of seven pregnancies, six were accompanied by eclampsia - the last ending fatally. In this case the eclampsia came on very early in pregnancy, and in all cases coma was a marked feature. The post-mortem examination showed chronic nephritis of an advanced type in the left kidney, and acute inflammation of the right. In Oldhausen's cases, eclampsia was found recurring in only 1%; and Dürhssen, out of all his cases, only met with it in three patients. Where it does recur, it is often not in the immediately succeeding pregnancy, but in an interval of one or two ordinary labours. *Sturmer* has lately reported a case where eclampsia appeared ~~in~~ three different pregnancies, in this case the attacks were only slightly affected during the attacks differing markedly from above case of Caraccio in this respect.

NUMBER OF CONVULSIONS AND DURATION OF ATTACKS.

The number of convulsions seen in different cases is very variable. There are usually more than one, occurring at varying intervals; although cases are on record where there was only one attack; and, recently, a death after a single convulsion has been reported. Again, the number of convulsions may be very great, as many as 125 in the 24 hours having been noted. In the great majority of cases, there is a return of the convulsions in fifteen to twenty minutes after the first; and they continue with greater or less severity, and at longer or shorter intervals, according to the severity of the attack. In some cases, however, the patient may not have another fit for hours; or, again, the attacks may recur so often that the patient seems to pass from one fit into another. In the milder forms of eclampsia, the patient may only have seven or eight seizures; while, in the severe, Vinay and others have counted over one hundred.

As regards the duration of the attack, we find that most of the cases either get better, or become very much worse during the first 30 hours of the attack: we rarely find them lasting longer than ~~the~~ or, perhaps, three days.

INFLUENCE OF ECLAMPSIA ON THE UTERUS.

It is my opinion that a good deal of work still requires to be done regarding the condition and behaviour of the uterus during an eclamptic seizure. Indeed, research up to now gives one the impression that its behaviour has been almost ignored, receiving very scant attention, and being dismissed by many observers in a few general sentences. Furthermore, in most cases, in the hurry and anxiety to end labour or combat the convulsions, the behaviour of the uterus is overlooked.

In looking carefully through the literature of the subject, and from my own observations, I think one may legitimately conclude that the uterus during an eclamptic seizure does not always behave in the same way. For example, in some cases, - and one is bound to add a small percentage of cases, - it remains dormant, taking no part in the general muscular storm going on, "as if it were," the famous Tarnier very happily puts it, "astonished at the universal disorder." Again, some observers (e.g., Spiegelberg) declare that in eclampsia, when labour occurs, sometimes the period of dilatation was slower than usual under like circumstances. Further, many observers declare that when eclampsia occurs at the onset of labour, after a few convulsions, the cervical canal is obliterated. In my own cases, I found that labour went on very slowly indeed till full dilatation of the cervix, that it was practically stationary, and that I had to artificially dilate the os; but I found also that, in 2 cases after the os was pretty well dilated, labour was rapidly terminated. On the other hand, we find generally, as we would naturally expect, that owing to the contraction of the abdominal muscles the labour is very generally hastened; in some cases it may be precipitately finished, and the physician is astonished to find the child appear when nothing a short time before suggested such an occurrence. This takes place more often in multiparae. It is also probable that in many cases the uterus is involved in the general muscular excitement and that the labour pains become intensified. In a case closely studied by such a careful observer as Braxton Hicks, the uterus contracted gently every 10 or 15 minutes, as it does during pregnancy and early labour, relaxing after a minute or so, and becoming quite soft, the foetal form being readily felt. When the attack of convulsions came on, he found that the uterus became intensely firm and hard, and remained in that condition for 10 or 15 minutes,

and then slowly relaxed to its normal condition.

Cases are on record where the convulsions ceased for a time after the escape of the liquor amnii; while, again, it is well known that uterine contractions may determine a fit.

When eclampsia occurs in pregnancy it is not surprising, in view of the profound shock to the system and the general disturbances of the muscles, to find very frequently an abrupt termination of labour. Although, then, the general rule is that the eclampsia brings on labour sooner or later or that labour is induced to save the patient's life, a good many cases have been recorded of late where convulsions during pregnancy have been cured by the treatment adopted; while, again, they may occur without the uterus being involved and disappear spontaneously without any treatment being adopted - the patient going on the full term without a return of the convulsions. But I think that more usually the convulsions recur immediately the labour pains commence. In one of my cases of eclampsia, in an epileptic, the patient had three typical eclamptic seizures when the disease was arrested, and bore her pregnancy for nearly two months before the occurrence of labour at the eighth month when the fits reappeared. In most of the cases where eclampsia has been arrested, the usual thing is for the child to be still-born: in some cases it is even macerated. In the same cases, the child survived three fits, but was killed by the return of the convulsions during labour. Different writers have reported cases in which the convulsions appeared during pregnancy and caused the death of the foetus; then the fits ceased, and the patient went on to full term without the superintention of convulsions. The same thing, moreover, has been reported in the albuminuria of pregnancy: on the death of the child there was cessation of the threatening symptoms. But Schreiber relates four instances of eclampsia, where on delivery the foetuses were found to be macerated, and remained in the uterus dead for a long time before the superintention of the eclampsia. At other times it is found that, where eclampsia occurs and results in the induction of labour, the convulsions cease when there is dilatation of the os - to recommence, however, as soon as delivery is effected. Nevertheless, this is unusual the commoner experience being that the convulsions are accentuated during labour, sometimes each contraction bringing on a fresh fit. On this account, some observers have gone the length of declaring that there can be no convulsions without uterine contractions, that the two are intimately connected, the one being the reflex action of the other; the first convulsions, though it happens early in pregnancy, being associated with a contraction of the uterus, no matter how small; determining the attack; and that at any time these little pains may be overlooked.

Some hold that the uterus is stimulated to contract by the defective aëration of the blood, for Marshal found that he could induce contractions in involuntary muscle by irritating it with CO_2 , and that in an eclamptic seizure there must be an excess of that gas in the blood; while other observers hold that it is the toxins in the blood which irritate the uterine nerve endings, and determine and intensify the contractions.

In two of my cases, notwithstanding strong uterine contractions, during fits, no difference in the size of the os was noticeable; but, once the os was mechanically dilated to the size of a five shilling piece, labour was speedily terminated. In one, I managed to dilate the os to the size of a florin; it was then soft and patulous and dilated easily; but, with the accession of two fits, it became contracted and rigid. In none of my cases, kneading the uterus after delivery (to check haemorrhage, which was becoming alarmingly severe) brought on a fit: this has been frequently confirmed by various observers.

I think it will be proved from further observation that - notwithstanding the views of several prominent authors, who say that the involuntary muscular organs take no part in the general convulsive seizures, in many cases the uterus does become ^{more} powerfully contracted whenever a convulsion appears, and that the pain lasts longer than it would otherwise have done.

THE EFFECT OF DELIVERY ON ECLAMPSIA.

This has a most important bearing upon obstetric treatment, as we shall see when we discuss it. Opinion is sharply divided on this point, - the members of ~~the~~ school, who favour the early evacuation of the uterus, basing their treatment on the fact that, in the great majority of cases, this is followed by the cessation of the fits; while their opponents deny this, holding that, in many cases, the convulsions continue after delivery has been effected, and consequently point to there being no necessity for active obstetric interference.

CONDITION OF THE UTERUS AFTER DELIVERY.

According to many observers, post-partum haemorrhage is a frequent occurrence after eclampsia. It is usually caused by uterine inertia; but it may follow prolonged anaesthesia, or it may be due to the hastening of labour giving rise to lacerations. The inertia of the uterus, and this tendency to haemorrhage, may be due to the albuminuria or to the ^{poorly} aerated condition of the blood caused by the attack. Some writers, however, have found the very opposite condition, namely, that the involution of the uterus was unusually rapid; and others also, who have had many cases of eclampsia, say that post-partum haemorrhage is very seldom found.

EFFECT OF ECLAMPSIA ON THE CHILD.

The ordinary movements of the foetus in utero are sometimes exaggerated, and ~~the child may develop~~ ^{it may develop} convulsions. The prognosis for the child in eclampsia is very bad, death probably occurring in about 50% of the cases, - although some writers estimate differently. Tarnier put it at 60-70%; Winckel at 77% (in marked contradistinction to his 7% in the case of the mother); Dührssen at 49%; Schreiber at 26.1%; and Oldhausen at 28%.

In considering the statistics published by different observers, we must remember that the method of treatment must have an important bearing on the life of the child. Those who belong to the "expectant school of treatment" certainly show a larger mortality (e.g., Winckel's 77%) than those who believe in emptying the uterus as soon as convulsions develop. The opponents of the morphia treatment hold that in some cases it has a bad action upon the child: this is admitted by some of its warmest supporters. This question, however, shall be more fully considered when I come to the subject of treatment.

The age of the foetus, the number, frequency, and severity of the fits, and the depth of coma have all an important influence on the prognosis as regards the child. According to Dührssen, should eclampsia develop in the seventh month, or earlier, the foetal mortality is 100%, and in the eighth month 93%.

A great many cases have been reported where the child, after surviving labour, developed convulsions - these being caused probably by the action of the toxic substances which were absorbed when in utero. Some authors have demonstrated the presence of albumen in the child's urine shortly after birth. It is not difficult to understand that the vitality of a child born of an eclamptic mother is very much below the normal, and that the former frequently succumbs during the first 36 hours of its existence. Some hold, moreover, that in the bulk of cases where the foetus is born alive it is not properly nourished, and is also below its normal weight. Fitzgerald has recently reported a case where a child had 30 fits during the first few days and, strange to say, recovered. Winckel and others have shown that, if the foetus is killed by a convulsion and pregnancy goes on uninterruptedly, labour may be quite free from fits. In twin pregnancies sometimes one child dies, while the other one survives.

As regards the cause of the death of the foetus during an eclamptic seizure, asphyxia is perhaps the commonest. The vitiated blood of the mother, caused by the convulsions, interferes with the respiration, and consequently gives rise to deficient aeration of the blood, and allows of a too small supply of oxygen to reach the foetus. Again, the toxins which have caused the convulsions in the mother play a very important part in the death of the foetus. The continuous and violent compression of the uterus upon the child must also be taken into consideration, as often the meconium is seen to

trickle forth with the liquor amnii when the head presents.

Placental haemorrhages and infarctions are very frequently found, and as a result of this, of course, many villi are destroyed, and this, too, will interfere with the oxygenation of the foetal blood.

THE TEMPERATURE IN ECLAMPSIA.

Winckel was the first to draw attention to the fact that in eclampsia we have a progressive rise in temperature, which may reach a very high degree in those cases in which the termination is fatal.

Bourneville made more extended observations on the temperature. He found that, during a convulsion, the temperature rises from the beginning to the end; and that, in the intervals between the attacks, it remains elevated, rising with each successive convulsion. In cases where death occurred, he found that the temperature progressively rose till it reached a very high degree at death (101° F. being common and very often exceeded), while it might be much higher after death. Again, if the case is to end in recovery, the temperature falls progressively till it reaches the normal. The temperature, then, in his opinion was a valuable prognostic sign. Furthermore, it is common to find the temperature fall and become subnormal in uraemia, so that it was also a great aid to diagnosis.

But many experienced observers since then have shown that eclampsia may occur without any considerable rise in temperature, while in some cases there may be no pyrexia at all. Regarding its value in prognosis, it has also been pointed out that the case may be very grave without great increase in temperature; while in certain cases the temperature has become subnormal at death, and in cases, too, where there was no history of previous renal mischief.

With regard to its diagnostic value, Charpentier states that Bouchard has ascertained that if uraemia, taken in general, produces in the majority of cases a slowing of calorification sufficient to produce a subnormal temperature, nevertheless, it might break out again, and give rise to an increase in the body-heat. Charpentier also shows - in the charts accompanying his publication - that, in conditions of mal epileptica, the curve of temperature is the same as that of eclampsia, but there is no albuminuria; and, on the other hand, after the commencing elevation of temperature a depression is produced, succeeded by a sudden rise: when death occurred a very high temperature might be reached.

One is forced to admit that this question is still in a most unsettled position; for, apparently, we may have great variations in the temperature, and also many fits without materially affecting the hyperthermia. Cases are encountered frequently where profound coma succeeded the cessation of the fits, and yet the temperature rose.

The rise in temperature, therefore, must be caused by something else than the number and severity of the convulsions. Various theories have been advanced to explain this hyperthermia. One condition appears to favour the theory that the convulsions themselves are the cause of the pyrexia, and that is the status epilepticus, - a condition in which one fit follows another in rapid succession without consciousness intervening, and in which there is a marked and progressive rise of temperature. Oldhausen affirms that the rise in temperature is brought about by the poison which causes eclampsia stimulating the thermal centres; Zweifel that the fever is nearly always of an infectious order; and Stroganoff that, as the disease itself is an acute infectious fever, we would naturally look for a rise in temperature during the time of its existence.

Of the modern writers on this subject, Herman has done excellent work. In a most careful examination of 12 cases, of which I give a short summary, he regards the temperature as being neither a help in prognosis or in diagnosis. In 4 fatal cases, where the patient practically went from one convulsion into another, the temperature was subnormal in one. In another, where death was due to a pulmonary complication, there was a moderate rise of temperature (102° 8 F.) before death. In the third, where there was no pulmonary complication, death occurred in the midst of coma, with the temperature steadily rising; while, in the last case, where death resulted from haemorrhage, into the lungs and brain, the temperature rose to 104° F. up to an hour from death, but again fell immediately before the patient expired. In one case, which recovered, great and sudden variations of temperature were found, but without any relation to the fits. He holds that a temperature over 103° is exceptional, its common range

being between 100° and 102°F.

One cannot generalise, then, regarding the temperature in eclampsia, for we at present have no proper knowledge of the condition which leads to this increase of temperature found in the disease.

In all my cases there was a rise in temperature, although not very marked: this rise was most pronounced in the post-partum case, where ~~whereas in one~~ renal changes were slight, and everything pointed to the liver as the organ at fault. We know that there are different clinical varieties of eclampsia: may not the progress and height of the temperature depend upon the clinical type encountered?

One must also remember, in considering this subject, that the different methods of treatment adopted must have considerable influence on the temperature. It has often been pointed out, for example, that chloroform, bleeding, and chloral have a considerable effect on the temperature, lowering it considerably; also that in cases where treatment had not been adopted till well on in the disease, as where medical aid was long in being procured, a high temperature is very often found. A good deal of investigation, however, still requires to be made as regards the temperature in eclampsia, and ^{also} as to the effects of the different lines of treatment upon it.

THE PULSE IN ECLAMPSIA.

In the premonitory stage of eclampsia, the ~~phase~~ is one of high tension, hard, bounding, and rapid. During an attack, the arterial tension is markedly increased, especially during the tonic spasms, the pulse becomes more rapid and loses its regularity. As soon as the attack passes off, the pulse becomes slower and more regular, while the tension becomes less. In severe cases, however, the pulse loses its high tension and becomes much softer, more rapid, and irregular; and this increases as the condition intensifies.

To obtain sphygmographic tracings in a case of eclampsia is a matter, needless to say, of considerable difficulty; but Ballantyne has managed to obtain them from three cases at different stages. He found that, during the first stage of the labour, where eclampsia supervened, the tracing gave a small pulse of high tension and regular, not unlike that seen in the rigor of acute fevers and peritonitis. Also, when a series of attacks occurred, or where the first stage was prolonged, the blood pressure fell in a remarkable manner - the pulse becoming dicrotic, or hyperdicrotic even, and very rapid, resembling that seen in cases of severe haemorrhages, etc.

Galabin has frequently taken sphygmographic tracings of the pulse, and he declares that it is not dicrotic and of low tension, as is found in fever, but one of abnormally high tension like that observed in Bright's disease.

The old observers laid great stress on the high tension of the pulse, and all their efforts in treatment were directed towards its reduction. Herman has drawn attention to the fact that this high tension may not be a bad feature, and that, in fact, it may serve a good purpose.

TERMINATIONS OF ECLAMPSIA.

The outcome of the eclamptic condition usually depends upon the severity of the attacks. It may end in death, as is too often the case; or the attack may leave behind it a mind permanently deranged - insanity, as one would naturally expect, being more frequently found after the eclampsia than after a normal delivery. Charpentier observed it in 9.2% of his cases, and Knapp in 13%.

Other nervous disorders, following in the train of eclampsia, are hemiplegia, - as a result of haemorrhage into the brain, - rarely aphasia, - which may be only temporary, - disturbances of vision, - e.g., amaurosis continuing for some time, - and deafness. Or, the kidneys may never recover from the damage done them, and so become chronically affected. Fortunately, however, we find that, in the bulk of cases which ~~recover~~, the cure is complete.

It is rare for death to occur during a paroxysm: when it does happen, it is due to asphyxia (caused by the long-continued tetanic contraction of the respiratory muscles occasioning cessation of respiration) or to spasm of the glottis. More usually, however, the patient dies from a more gradual asphyxiation, the result of interference with the proper aëration of the blood by the convulsions. Again, death may result from oedema of the lungs, which is the result of serous effusion from the overcharged capillaries; or it may be

due to ^{gradual} ~~general~~ asphyxia caused by the accumulation of secretions in the bronchial tubes. A frequent cause of death is exhaustion; while, again, the respiratory centre in the brain may be directly affected by the toxic substances in the circulation and become gradually paralysed. One may have the same action in the heart, either by the poison acting directly on the nerve endings in the heart, or on the heart muscle, or indirectly by the interference with the function of the lungs. Cerebral and meningeal haemorrhages cause death in some cases, while cerebral congestion or oedema may produce a fatal termination in others. Cases have been again and again reported where the patient passed into a state of profound coma with the temperature rising to a great height, even after the disappearance of the fit, the patient practically dying of pyrexia; while, again, others may die in deepening coma with the temperature subnormal. Dyspnoea may come on suddenly and be very marked, gradually increasing till death occurs.

Many patients after an attack of eclampsia suffer for a while from bronchitis, which is the result of the congestion of the lungs so frequent during the fit; or acute capillary bronchitis or pneumonia may develop and the patient perish forthwith.

Some patients succumb, after they have recovered from the eclamptic attacks, of deglutition pneumonia, which either takes the form of capillary bronchitis or lobar pneumonia; it is caused by the entrance of secretions from the pharynx and mouth into the lungs, or by nourishment or drugs, administered by the mouth, gaining access to the bronchial tubes.

Munro, Karr, and others have reported cases where death was caused by a ruptured duodenal ulcer giving rise to septic peritonitis; and Leicester, of Calcutta, has lately reported a case where death occurred, on the tenth day after delivery, from general septic peritonitis caused by the rupture of an abscess in the spleen.

My friend and namesake, Dr. James Logan, of Wishaw, has told me of a case of eclampsia, which he recently attended, the history and termination of which was rather unusual. This patient developed a marked petechial rash in the last month of pregnancy, and her labour was accompanied by eclamptic fits which continued for a time after delivery. On the fourth day after delivery, severe vomiting of blood set in; the blood first vomited was like coffee grounds, but later on, blood unacted upon by gastric juice was vomited up in large quantities. The patient died. There was no pulmonary complication, no rise in temperature, and no signs of peritonitis.

Cazeau declares that death may occur from rupture of the uterus when eclampsia occurs at the commencement of labour, owing to the uterus participating in the violent contractions of the muscular wall in the presence of an insufficiently dilated os.

Baillly has reported two remarkable cases - the first where the tongue, during a convulsion, was so severely bitten as to necessitate ligature of the lingual artery, otherwise the patient would have bled to death; and the second where such great swelling of the tongue followed injury by being bitten as to cause suffocation.

D I A G N O S I S.

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I. DIAGNOSIS IN THE PRE-ECLAMPTIC STAGE.

In describing the various prodromal manifestations of eclampsia, I showed how it is now recognised by most observers that the condition of toxæmia, which may lead to an eclamptic seizure, can in many cases be diagnosed long before the occurrence of the convulsions. These premonitory phenomena, again, may be so slight as not to force themselves upon the patient's notice; or, again, even if unnoticed, they may be misinterpreted by the bulk of women, who seem to look forward to the pregnant condition as one bringing with it untold misery, so that they come to regard many signs and symptoms as the natural sequence of parturient state. Furthermore, they may even be misinterpreted by the medical attendant, who often too carelessly brushes aside the various complaints of the pregnant woman without stopping to investigate their causation, or what may be responsible for the production of these signs of ill-health; or, if he does trouble himself, in many cases it is merely to treat the

most obtrusive of the symptoms themselves, leaving the condition which has given rise to these symptoms to look after itself. The warning, ~~then~~, conveyed by the rapid pulse of high tension, persistent headache, ocular disturbances, obstinate constipation, etc., may be entirely overlooked. Diminution in the amount of urine passed, a fall in the percentage of urea, with complaints of severe headache, flashes of light, feeling very ill, and so forth, are danger signals which ought to force themselves on the notice of every obstetrician, and which, moreover, should be regarded as augurs of the near approach of eclampsia; while the appearance of albumen in the urine, the occurrence of oedema, - especially of the face, - and epigastric pain are still more significant signs of impending danger, and signs which ought by no possibility to be misconstrued.

II. DIAGNOSIS DURING THE CONVULSION.

One would think that a typical attack of eclampsia could not present any formidable difficulties in effecting a correct diagnosis - nevertheless, care is necessary in interpreting any form of convulsion that may make its appearance during pregnancy, in labour, or during the puerperium. It does not follow that because we find convulsions during these periods, the case is necessarily one of eclampsia, although it is perhaps the best plan to regard all such cases as eclamptic till the contrary is proved. Hysteria, for example, is a condition which is frequently seen; it certainly sometimes simulates an eclamptic seizure, and it may be mistaken for the latter by a careless observer; while, again, epilepsy - which perhaps bears the most striking resemblance to eclampsia and which may occur in pregnancy, labour, or during the puerperium - might also be mistaken for it. Let us first consider:

EPILEPSY.

Here we have the same loss of consciousness and sensation, tonic and clonic convulsions, with the same relation to each other, followed by the development of coma. As epileptic fits sometimes develop in pregnancy and during the puerperium in women who have never previously experienced them, Barnes declares that "gestation has the faculty of evoking a latent organic or functional disposition to epilepsy". When epilepsy occurs in pregnancy, it is not nearly so liable to bring on abortion or premature labour. In epilepsy one has usually, however, the history of previous attacks, which may guide one to a proper diagnosis; but, again, eclampsia may develop in an epileptic who has become pregnant.

This happened in a most interesting case of my own, where the patient had two separate attacks of eclampsia - one at the sixth month, which disappeared after she had three fits, reappearing at labour, which occurred prematurely at ~~eight~~ and a half months, and who, besides four separate epileptic fits at different periods of her pregnancy, had one eight days after the termination of her labour. I saw her after two of these epileptic fits: in each there was the characteristic cry of the epileptic, as well as the sudden fall to the ground. Her first epileptic fit occurred when she was one month pregnant, the second when two and a half months, the third when four months, the fourth when five and a half months pregnant, and the which appeared the first day she rose after confinement. I saw the fit which occurred at five and a half months, as well as the one after delivery. The urine was normal in quantity and contained a trace of albumen after the epileptic fit, although the former was ~~teeming~~ ^{teeming} with the ~~latter~~ ^{latter} a month after ~~the~~ ^{during} the eclamptic seizure. Following the post-partum fit there was ~~also~~ ^{no} albumen present, but the urine had never properly cleared (I had examined it every day) since the eclamptic fits during labour, when it was loaded with albumen. Before each of the epileptic fits she complained of giddiness and tingling in the right arm, and this she said was what nearly always occurred before her epileptic fits. Sometimes, I may add here, albumen has been found in the urine after an epileptic fit, but it is usually only a trace and is not accompanied by tube casts, blood, etc. ^{and suppressed during the day.} One may look upon its appearance as the result of the excessive muscular efforts in the fit, just as we may find albumen in the urine of athletes after strenuous exertion. None of the premonitory signs and symptoms which were present before her attack ^{of eclampsia} were seen prior to the occurrence of the epileptic fits. I took her temperature after the latter and found it normal; while, during her attacks of eclampsia, after she had had two fits, I found that it was 102.8°F.

We find, then, that the aura, which is so often found ushering in

the epileptic fit, is a rarity before eclampsia, as is also the epileptic's shrill cry; that the attack is usually more sudden in epilepsy, the patient generally falling to the ground; that albumin~~ia~~ is very rarely found in the urine of epileptics, unless there is present some other condition producing it or some lesion of the kidney; and that the temperature, which is so often found in eclampsia to be progressively raised, is normal, or only very slightly raised, for a short period, in epilepsy. In the status epilepticus, where the patient goes from one attack into another without regaining consciousness, the temperature may be greatly raised. The coma after the epileptic attack is never so prolonged, and consciousness returns more quickly and the intervals are longer, than in eclampsia. The pulse, too, will greatly aid in diagnosis, for it is quite different in eclampsia from epilepsy where, indeed, it may be normal or but slightly affected.

CEREBRAL APOPLEXY.

Cerebral apoplexy rarely occurs in pregnancy. If it does, as a rule, it generally occurs suddenly without any prodromal signs being present. There is the same rapid development of coma, but convulsions are very rarely seen. One also finds paralysis setting in. It should be borne in mind, of course, that cerebral haemorrhage may develop during an eclamptic seizure, as evidenced by paralysis, etc., setting in.

MENINGITIS.

There are rare cases on record where meningitis has simulated eclampsia. The history of the case here would be of the utmost assistance. The spasms, too, are more localised than in eclampsia where they are general; and, again, they usually increase gradually in severity. Delirium is very often present at one stage, and we also find fever before the occurrence of the convulsions; moreover, the pulse, which is slow, should serve as a good guide in clearing up the case. In connection with this disease, a most remarkable case has been reported by Dr. Wilson, where the patient was seven months pregnant, and in whom the development of severe continuous headache, the occurrence of epileptiform convulsions with coma, along with the presence in the urine of albumen, in considerable quantity, appeared to make the diagnosis of eclampsia absolutely certain. The urine likewise contained sugar in abundance; and, as this could not have arisen from absorption from the breasts, it must have been produced by the cerebral condition. The patient died, and the post-mortem examination demonstrated that it was a case of primary pneumococcus meningitis.

HYSTERIA.

I once had a very interesting case where hysteria, in a multipara (tenth child), developed during labour and simulated eclampsia. She was at full term, but had not "felt life" (to use her own words) for two months. The labour was a very difficult one, as I had practically to take the dead foetus away in pieces: it was a breech presentation. The patient fell into a semi-unconscious condition soon after labour began, but could be roused by roughly shaking her; and there were at intervals convulsive movements of the face, limbs, and body. She remained in this condition for two days, the convulsive movements occurring at intervals during that time; and, on recovery, she stoutly maintained that she had no recollection of the condition she had been in, nor even of the labour and its completion. I drew off a large quantity of urine with the catheter, but found nothing abnormal therein on examination. There was no particular rise in temperature, except eight hours after labour, when the temperature was 99.6°F. Next day it was normal, and she made an excellent recovery. The movements were not of the general nature observed in eclampsia, being more disordered and irregular, and no coma developed at any time. She was the wife of a miner who had frequent outbursts of drinking, during one of which, occurring the night before the confinement, he had abused her; and this she thought might explain her condition during labour and for three days after it. She had never had such an attack before, and was not what one would usually term an emotional woman.

The history of this case may be taken as showing the different

and distinctive features in which an attack of hysteria - occurring during pregnancy, labour, or in the puerperium - differs from eclampsia. I may say, however, that, unlike this case, one can frequently obtain a history of a previous attack, and that there is often marked excitement present with frequent outbursts of laughing, crying, or screaming. Furthermore, it usually develops in nervous, excitable, and irritable women; and there are often complaints of a sensation of globus, palpitation of the heart, oppression, and a feeling of choking - none of which phenomena were present in my case.

CONVULSIONS OF FATAL HAEMORRHAGE.

It has been noted that in cases of fatal haemorrhage one of the signs immediately preceding death is a convulsion: this is probably due to cerebral anaemia. Barnes says that here death is preceded by general tremor, - a kind of universal shuddering, - consciousness is not always abolished, and there is no congestion of the face, of course. Again, there is often vomiting, the pulse is rapid, thready, and almost imperceptible, and there are the usual signs of acute anaemia.

Spiegelberg has a case where the convulsions, - which preceded death in a woman whose uterus was enormously distended after delivery through internal bleeding, - simulated eclampsia.

PUERPERAL CHOREA.

I had a very severe case of puerperal chorea in a primipara where, at certain times while the patient was lying, one would actually have thought that she was in a convulsion. There was albuminuria as well in this case; but, as eclampsia developed with labour, I shall describe it in detail later on.

ALCOHOLISM.

Where the patient is seen by the obstetrician in the comatose condition, - especially as the friends of the patient may have administered alcohol to her for the complaints which usually precede the manifestation of the disease, - the case may be mistaken for alcoholism. The examination of the urine, however, would solve the difficulty.

CEREBRAL TUMOUR OR ABSCESS.

Where fits develop here, they may resemble eclampsia; but the history of the case, the headache, vomiting, condition of the pulse, and the gradual development of the illness; while in these cases optic neuritis is frequently present, and paralysis in certain part is sometimes seen.

CONVULSIONS IN LEAD-POISONING.

In saturnine intoxication the symptoms are not unlike those of eclampsia: here there are often convulsions, coma, and albuminuria produced by nephritis. Depaul has recorded a remarkable case of this kind. The muscles of the face are usually not convulsed to the extent seen in eclampsia, and the convulsions - tonic or clonic - are incomplete. Furthermore, coma is not so profound, and the blue line on the gums will help considerably towards a correct diagnosis.

Regarding lead-poisoning, attention has been drawn by various observers - more especially by Albutt - to the fact that the manifestations thereof - symptoms, signs, pathology - are not unlike those of eclampsia; and Albutt uses this fact in furthering his argument for the toxæmic cause of the albuminuria of pregnancy.

When in doubt about any case, the examination of the urine for sugar, albumen, tube casts, and blood will usually help to throw light upon the subject; together with, of course, a careful investigation of the history and the other symptoms and features of the case.

P R O G N O S I S .

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THE MORTALITY OF ECLAMPSIA.

Although it is agreed that during recent years there has been a considerable fall in the mortality of eclampsia, nevertheless, it is still one of the most formidable conditions which we have to deal with in pregnancy, labour, or the puerperium.

The mortality is very variously estimated. Among the older observers, it was common to find writers giving a mortality of over 50%; Pajot has placed it at 48%, and Bailly at 42%; but all these are higher figures than now recognised as average.

Treatment has undoubtedly done a great deal to lessen the mortality; but, even when the same treatment has been adopted in different districts, and considering great numbers of cases, we find that the death-rate is very much higher in one place than another. ~~Tarnier~~ Tarnier put his mortality at 30%; Charpentier 28%; and Oldhausen at 25%.

It is interesting to compare the statistics of the great pioneers of the different lines of treatment. Veit, who did so much to popularise the morphia treatment, had 60 cases with 3% of deaths. Winckel, the great advocate of chloroform in this disease, had a mortality of only 7%, but he had a very high foetal death-rate. Both these observers favoured the "expectant" treatment, and administered their favourite remedy in heroic doses.

Few, however, have been able to point to such good results as the above. Mangiagalli has lately published 18 cases, in which he used veratrum viride, all of which recovered; Parvin, who also enthusiastically vouches for the efficacy of this drug, had the low death-rate of 8%. Stroganoff, who used chloral and morphia, had a death-rate of 5.31 per cent. in 113 cases; and Porack, one of the first advocates of the use of saline injections, a mortality of 6.38%. The average death-rate in America is about 25%; and Dührssen, the great advocate of active interference, gives 20% as his mortality; and Zweifel, a follower of his, 22%. Fehling who, like Dührssen, advises active interference, but in a modified form, had a death-rate of 17%; and Bidder one of 17.3%.

Recently, a number of observers have reported series of cases where the death-rate was as low as 5%, while some obstetricians - in a limited number of cases up to 24 - report not a single fatality.

It is agreed by the great majority of observers that the mortality is higher among multiparae than among primiparae - the highest death-rate being found where the fits developed during pregnancy in the former class. Oldhausen, however, declares that there is little difference between the two; while, again, Zweifel affirms that the mortality in primiparae is nearly three times greater than in multiparae. The following table of the observations of various obstetricians gives the relative mortality in the two classes:

TABLE III.

THE MORTALITY IN PRIMIPARAE AND MULTIPARAE COMPARED.

<u>Author.</u>	<u>Death-rate in Primiparae.</u>	<u>Death-rate in Multiparae.</u>
Dührssen.	19.5%	28%
Goldberg.	21%	45%
Veit.	14.3%	19.5%
Zweifel.	16.6%	3.5%
Oldhausen.	25%	25%
Schanta.	37.3%	44.9%

The earlier the eclamptic symptoms appear during pregnancy the graver must be the prognosis, and the same is true as regards their early appearance in labour, while, should eclampsia develop when the latter is well advanced, we may reasonably expect a more favourable issue. As a rule, we may say that eclampsia, if it appear after the termination of labour, is least likely to cause death; but Tarnier found that the most fatal period was after delivery, 42% of his deaths being recorded then. Oldhausen, in his mortality list, found that the period in which eclampsia developed made no difference in the death-rate, for he recorded the same number of deaths in the three periods. Charpentier, in an analysis of the statistics

of the German authors, found that the mortality of post-partum eclampsia was 12.5%. The following table gives a variety of estimations in this particular:

TABLE IV.

MORTALITY OF ECLAMPSIA ACCORDING TO TIME OF ONSET OF THE SYMPTOMS.

Author.	Pregnancy.	Labour.	Puerperium.
Tarnier.	27%	31%	42%
Dührssen.	38%	19%	12%
Oldhausen.	25%	25%	25%
Boldberg.	65%	11%	0%
Schreiber.	30.5%	19%	13.5%
Zweifel.			9.5%
Green.			6.5%
Schanta.	52.5%	40.2%	27%

THE BEARING OF THE FREQUENCY, SEVERITY, AND NUMBER OF FITS ON PROGNOSIS.

In general, we may say that the greater the number of the convulsions the graver should be our prognosis. Although no hard and fast rule can be drawn up, we may take it that, if the patient has more than from 14 to 18 fits, the outlook is bad. Bailly has reported a case where recovery took place after 100 fits, but this is quite an exceptional experience. Other things being equal, it is a fact that the prognosis becomes graver with each successive fit. One must remember, of course, that death may take place after but a very few fits; and one case, at least, is on record where death occurred after a single convulsion. Whenever a fatal result occurred after but a few convulsions, it has been found that the comatose condition developed early and was very profound.

The more violent the fits, and the more prolonged they are, especially in the tonic stage, the more serious would be our prognosis; while, should signs of mania develop, the case must be looked upon as almost hopeless. I think, then, it will be found that the frequency and severity of the attacks are of more account than their actual number.

Attention has lately been drawn to the fact that should death of the foetus occur, as a result of eclampsia, the chances of the mother's recovery are considerably increased.

THE URINE AS AN AID TO PROGNOSIS.

Most observers are agreed that the prognosis is more serious when the amount of urine passed is small. Where there is rapid diminution of urine, or complete anuria lasting even for a short time, the outlook is even worse. Herman, however, has reported two cases, in one of which there was polyuria and in the other no diminution of urine: the first one died, and the other developed permanent renal disease. Nevertheless, one of the best prognostic features we can have, in the vast majority of instances, is where the urinary secretion becomes increased.

Several observers (Davis, etc.) have demonstrated that the amount of urea excreted is a very valuable aid to prognosis: should the urea be diminished, the signs and symptoms of toxaemia are more marked, and the prognosis is consequently graver.

As regards the quantity of albumen present in the urine and prognosis, it has often been found, - in cases which ended fatally after delivery, where eclampsia appeared in pregnancy or labour, - that the albumen, instead of becoming less, persisted or even increased in amount. Again, it is generally held that the smaller the amount of albumen present the better the prognosis. But, although this may obtain in certain cases, a great number of the latter have been recorded which ended fatally in the absence of any marked urinary change.

In my case of post-partum eclampsia, there was only a very slight trace of albumen during the whole of the attack, and the case was much severer than the others where albumen was present in abundance.

THE TEMPERATURE AS AN AID TO PROGNOSIS.

Recent observations on the temperature in eclampsia have made it an open question as to the influence of the temperature upon a given case. Formerly, obstetricians laid very great stress on the rise and progress of the temperature; and many modern observers also have been too prone to accept the views of the older physicians, who - especially the Continental teachers - were very dogmatic in their assertions regarding the temperature and its bearing upon prognosis. Herman has made a very careful study of the temperature in eclampsia, and he places very little reliance upon its value as a prognostic sign.

As a rule, however, one may say that in most cases, where the temperature is steadily rising to a higher figure, the prognosis would be grave; again, that where the temperature was found to be gradually coming down, one would look for a favourable termination; and also in those cases where it never reached a higher degree. Nevertheless, cases are on record where the temperature was only slightly elevated and which ended fatally, and also instances in which the temperature was subnormal before death.

PRESENCE OF OEDEMA AND ITS BEARING UPON PROGNOSIS.

Formerly, it was held that one of the worst signs in eclampsia was the appearance of marked oedema. Wieger was one of the first, however, to prove from his mortality figures that it was not such a serious symptom as had been supposed, for he found that the death-rate in eclampsia with oedema was 36%, while the mortality in eclampsia where it did not appear was no less than 63%. He also regarded with alarm the absorption of oedema during pregnancy.

Clinicians have long held that uraemic convulsions are more frequently encountered in Bright's disease, among those who are not dropsical, than in those where dropsy is a prominent symptom; and in eclampsia the view that oedema often points to safety is now accepted by the most modern observers. Leopold showed that in 40 cases in which it appeared, it was severe in 10, all of which recovered; not very marked in 17, of whom 5 died; while, again, in 13, in whom it was not very marked, 5 died.

Carstairs Douglas has lately suggested that oedema may be looked upon as a safety-valve for serous exudation: if it occurred in the subcutaneous tissue it would augur better for the patient's recovery than where it was intracranial, or where oedema of the lungs appeared.

THE VALUE OF THE PULSE IN PROGNOSIS.

In reading the literature of the subject, I have been struck by the fact that the pulse in eclampsia has but seldom received the systematic and careful attention which it deserves, the bulk of observers being content to generalise thereon. It is, however, a very important factor in guiding one to a correct prognosis. If it recover volume and strength between the attacks and remain fairly regular and of good volume, even though it may be rapid, one would necessarily give a much better prognosis than where one found it, not only very rapid, but small, very compressible, or thready and very irregular.

ICTERUS.

Audebert has recently called attention to the value of icterus as a prognostic sign; for, in 34 cases of eclampsia, he found that, out of 4 who had icterus, 3 died; and Tarnier states that if icterus is present with the temperature running high, urine scanty, and the appearance of suncutaneous ecchymoses, the patient would rarely recover; but if the icterus alone were present without these other signs, the prognosis would be much more favourable.

OTHER FAVOURABLE SIGNS are profuse sweating early in the disease, and a rapid return to consciousness between the attacks.

The prognosis would be affected by the appearance of any MECHANICAL OBSTRUCTION to labour.

So many figures have been given lately, by capable observers, to prove that the disease is more fatal in CERTAIN DISTRICTS than in others (notwithstanding the fact that the same methods of treatment have been adopted in each) that we may hold the point established.

It is questionable, too, whether the SEASON OF THE YEAR has any bearing on prognosis, as some observers have tried to make out.

The prognosis may be made much more serious by DELAY IN TREATMENT: it happens not infrequently that when the obstetrician arrives he finds his patient in a moribund condition.

Again, the TREATMENT ADOPTED has undoubtedly a very important bearing on prognosis. When, for example, the obstetrician, in his haste and anxiety to bring the labour to a termination, causes extensive lacerations (with the consequent haemorrhage and risk of septic infection), or the development of post-partum haemorrhage follows the too rapid evacuation of the uterine contents, the attendant haemorrhage and shock in both cases must have a direct bearing upon the prognosis. Again sepsis may set in after the eclamptic seizure has been successfully combated, and cause the death of the patient. The faulty position of the patient, and the administration of drugs and nutriment to her when in a semi-unconscious condition, may, by ~~means of~~ the secretions of the mouth or materials placed there, find their way into the bronchi, and cause the patient to fall a prey to deglutition pneumonia.

Where old CARDIAC DISEASE is known to exist, the patient's chances of weathering a storm which throws such demands on the heart's resources would be greatly minimised.

Old RENAL DISEASE would likewise greatly prejudice the patient's chances of recovery.

ETIOLOGY.

.....

(A) GENERAL ETIOLOGY.PREDISPOSING CAUSES.

In considering the predisposing causes of eclampsia, and remembering the large percentage (75 - 78%) of cases occurring in primiparae as compared with multiparae, one might almost say that primiparity was itself a predisposing cause. In looking for a reason to explain this increased liability to eclampsia in such persons, we must admit that no clear scientific evidence has yet been brought forward. The greater intra-abdominal pressure, - caused partly by the undue rigidity and resistance of the abdominal muscles, - the dread of the approaching confinement accentuating the already considerable nervous excitement, the more frequent occurrence of albuminuria - all these have been advanced to show undoubted preference of eclampsia for the primiparous woman.

Again, eclampsia is found in primiparae more frequently when they are very young, that is to say, in those under 20; and in the aged, that is to say, in those over thirty years. Dührssen found that, out of 195 cases, 40.5% were either below 20 or over 30.

There are no statistics to prove that eclampsia is found more frequently in unmarried women who have the misfortune to become pregnant, although the shame and anxiety caused by their condition might so act upon their nervous system, and so lower their general vitality, as to predispose them to a certain extent to an attack of eclampsia.

Renal Disease. - All the work on eclampsia points to defective elimination as a paramount predisposing cause. We know that, notwithstanding the presence of grave toxæmia, symptoms of an alarming nature will seldom arise, provided the kidneys are sound and are actively eliminating toxic material. Again, local conditions of the kidney - such as acute and chronic diseases, which handicap it in its work - are bound to predispose the patient to attacks of eclampsia. These conditions will be discussed in detail presently.

From the writings of certain observers, one would think that kidney disease in no way predisposed the patient to eclampsia. One must remember, however, that where we have marked renal mischief, abortion and miscarriage are not infrequently encountered, as well as early premature death of the foetus - which latter, we have already seen, exercises a favourable influence on prognosis. There can be no doubt whatever that renal insufficiency does play an important part as a predisposing factor.

Retention of Urine, caused by constriction of the ureters, may be put down as a very likely predisposing cause. This might be produced by other conditions, which are also regarded as factors in the production of eclampsia - such as, an excessive enlargement of the uterus by hydramnios; and it has been noticed, in certain conditions of this kind, that, after the rupture of the membranes and the escape of the liquor amnii, the fits in some cases have stopped for a time or become less violent.

Multiple pregnancy may also be regarded as a predisposing element: we must remember that in this condition we have not only the increased size of the uterus, but also the fact that additional work must necessarily be thrown upon the mother's organs of elimination. An abnormally large foetus has also been given a place as a predisposing cause.

Although I have placed hydramnios, multiple pregnancy, and other conditions of excessive size of the uterus as predisposing ~~factors~~ ^{factors} in the production of eclampsia, the latter occurring in cases of great distension is far from being constant.

Painful and tedious labour (as in very young or aged primiparae),

and the existence of mechanical obstruction (e.g., by contracted pelvis or tumours), may also be regarded as predisposing causes.

There is nothing to show that the presentation of the foetus has anything to do with the occurrence of eclampsia.

Again, in cases where there is diminution in the size of the thyroid gland, or where it fails to enlarge as it normally does in pregnancy, and where in consequence we have a diminished amount of thyroid secretion, Nicholson says there may be a marked predisposition towards eclampsia.

Hereditary or Acquired Hepatic Insufficiency.- As we shall see in dealing with ~~the~~ special etiological considerations, the liver plays an important part in defending the organism against the inroads of toxic substances; while, again, should it be thrown out of gear, it not only adds to the general toxic condition (by failure in its defensive rôle) but may, by not functioning properly and perfecting proteid metabolism, allow of additional poisons being thrown into the system. Pregnant women are peculiarly susceptible to hepatic disturbances (the so-called bilious turns), tenderness over the liver (which organ is often found enlarged), and the development of icterus (which sometimes occurs). Insufficiency of this organ, as of the kidney, may be regarded as predisposing the patient to eclampsia.

Constipation and defective action of the skin are most important predisposing causes.

Heredity.- A few cases are on record where marked predisposition to the development of eclampsia was hereditary. The most remarkable instance of this kind was reported by Elliot. A woman has four daughters and died, at the birth of her son, of eclampsia. All four daughters in after life developed eclampsia, only one of them recovering from the attack.

In pregnancy we must always bear in mind that we have increased cerebral and reflex irritability; and if this nervous instability be marked (and Herff has recently drawn attention to this condition, which he says is too frequently hereditary), one would naturally consider that in such a person convulsions would be more easily produced.

As regards this nervous irritability, it has been suggested that, - in those cases of eclampsia where careful examination of the urine, ~~carefully performed~~, failed to show much kidney mischief, - it might be explained, in the presence of an increased cortical excitability, on the ground that very little renal insufficiency might be sufficient to bring about convulsions; also that where there was slight alteration in the function of the heart, liver, etc., this alteration, with increased irritability of the cortical region of the brain, might occasion the outbreak of eclampsia.

EXCITING CAUSES.

In some cases it is practically impossible to find any exciting cause, the eclamptic seizure coming on during sleep or after the patient has awakened therefrom; or she may be attacked whilst she is following her customary household avocations.

Again, the patient may be in such a condition, - actually saturated, one might say, with the poison, whatever it may be, - that very little will excite an attack - for example, a convulsion may be brought on by merely touching the os, or by the pressure of the hand on the abdomen, by the movement of the child in utero, or by long-continued uterine contractions. Thus, it is a well-known fact that sometimes when a frog is drugged with strychnine, its system is in such a condition that, if it is left alone, it will remain motionless; but the slightest external stimulus of irritation - e.g., merely a slight touch of the hand - will forthwith send it into convulsions.

Abrupt suppression of urine may be regarded, in a few cases, as

an exciting cause: in these cases it is frequently noted, on passing the catheter, that the bladder is hard and firmly contracted; while, again, cases are on record where eclampsia disappeared after the bladder had been emptied of an excessive quantity of urine (in one case the latter was ammoniacal). Irritation of the bladder may therefore be looked upon as an exciting cause of the convulsions.

Distension of the Bowel and Obstinate Constipation. - We have already seen how this may act as a predisposing cause; but the hardened faeces may also, by reflex action on the bowel, precipitate an attack. An unusual case of eclampsia is on record where, on the removal of foul-smelling faeces from the bowel by irrigation, the convulsions ceased.

The attack may be determined, as in one of my cases, by excessive exercise, or too hard work; or, again, it may follow upon the ingestion of a hearty meal, rich in nitrogenous substances, or of indigestible food.

Some observers lay great stress upon chill as the immediate exciting cause of eclampsia. Jaccoud - who introduced the rigid milk regimen in albuminuria - declares, in writing upon the subject of prophylaxis in eclampsia, that all treatment may prove of no avail, unless the patient be sedulously guarded against chill. The older observers laid great stress on atmospheric conditions; and we can readily understand that, where the kidneys are in any way incompetent, anything that would throw extra work on them might readily precipitate an attack.

In considering the predisposing and exciting causes of eclampsia, we must take into consideration the quantity of the waste products circulating in the blood, which will be influenced by multiple pregnancy - e.g., how long these products have been in accumulating, and the time taken in their elimination, will depend upon the condition of the excretory organs, and the conditions of defense which have to do with the destruction of toxins and the elaboration of metabolic waste products.

(B) ESSENTIAL ETIOLOGY.

Many of the numerous theories as to the causation of eclampsia have been abandoned - theories, some of them, which held undisputed possession of the medical world for years.

No one would contend nowadays that the disease is the result of uraemic poisoning, just as no one would now affirm that uraemia is due to the retention of UREA in the blood, for we now know that large quantities of urea may be injected into the blood stream without producing convulsions: in fact, its presence acts as a good diuretic, and Bouchard, recognising this, advised Pinard to employ it hypodermically in cases of anuria.

Still, as some of the older theories have contributed a good deal to our knowledge of this terrible disease by stimulating observation and research, and as modified forms of some of them yet find support, I think it will not be out of place here to devote some space to a consideration of some of the more important.

RAYER AND LEVER'S THEORY.

After Bright's great work on nephritis, Rayer in 1840, and Lever in 1843, demonstrated how frequently albuminuria was to be found in cases of eclampsia. The convulsions of that affection were looked upon as the same as those of uraemia, and as the direct result of interference of the renal function, caused by pressure on the renal veins, giving rise to inflammation of the kidneys - urea being regarded as the principal poison.

THE THEORY OF LABOUR PAINS.

Another theory was that the convulsions were produced by the labour pains. The increase in arterial pressure may be caused merely by the renal incompetency; where that is present, every pain

would increase it. When the uterus contracts, the result will be that the blood will be prevented from getting into the uterus. Some of the arteries supplying the latter arise from the aorta near the renal artery; so it was supposed that, owing to pressure on them, the circulation in the kidneys would be interfered with. The greater the pains, the greater would be the pressure in the vessels. Again, we find that eclampsia is very rare before the twenty-second or twenty-third week - at which time we usually begin to have intermittent uterine contractions. The nearer full term, the greater is the possibility of eclampsia developing; and all are agreed that eclampsia is most often seen during labour.

Against this theory, however, is the fact that in Bright's disease, where we have very high arterial tension, the quantity of albumen is often very slight in the urine. Again, experimental proof is against it - for, on dividing the renal and splanchnic nerves, the blood pressure in the kidneys is greatly increased, but there is no albuminuria.

FRIERICH'S THEORY.

Frierich was the next observer of note to give special attention to this disease. He also held the view that eclampsia was practically identical with uraemia; but, as he was not able to demonstrate the presence of urea in the blood, he affirmed that the poison at work was ammonium carbonate, - one of the decomposition products of urea, - which he found capable of producing convulsions when injected into the veins of the lower animals. The decomposition of urea into ammonium carbonate he regarded as the result of the action of some ferment developed in the blood.

Spägelberg, from the careful examination of the blood of an eclamptic (in which he was able to demonstrate the presence of ammonia) supported the view advanced by Frierich. But the popularity of this theory owed a good deal to the work of that brilliant writer and acute observer Braun.

It is interesting to note at this stage that Simpson suggested that the reason why chloroform was so valuable in the treatment of this disease was because it prevented the decomposition of urea into ammonium carbonate. He held that the inhalation of the drug produced a temporary diabetes; and he pointed out that if a little sugar be added to urine (out of the body), it prevented for a time this decomposition change.

Frierich's theory held the field until Richardson proved, from his special researches, that ammonia could be detected in the expired gases; and, as it could be shown to be present in the healthy subject, that one might look upon it as a normal constituent of the blood.

Hammond also did a lot of painstaking work on this subject. In some subjects he injected urea, in others he removed the kidneys, and in other some he, after removal of the kidneys, injected urea and urine into the veins. From an examination of the amount of ammonia in the blood and breath, before and after the operations, he found that there was no increase in the carbonate of ammonia, nor did the urea when injected change into that substance.

Many chemists have demonstrated ~~since~~ then that it is impossible that the changes described by Frierich can take place in the blood; and ammonium carbonate has never been detected in the blood of eclamptics in greater quantity than normal (Bernard).

Some observers state that in eclampsia we have, as a result of the kidneys not allowing the unsynthesised antecedents of urea and saline matter to pass from the blood into the urine, an accumulation of these resulting in an increase of the ionic concentration of the blood, and convulsions. They point to the fact that uraemia may be experimentally produced by the infusion into the blood of concentrated saline solutions.

REASONS AGAINST ACCEPTING THE URAEMIC THEORY.

These are numerous. Thus, eclampsia is known to occur in a small percentage of cases without the appearance of albumen in the urine; also, albumen is found to appear in the urine in some cases of eclampsia after convulsions have occurred, and, consequently, both the albumen and the convulsion have probably been produced by the same cause. Braun, and other supporters of the uraemic theory

admitted that these cases occurred; but they held that they were caused by reflex stimulation of the vasomotor and convulsive centres, and that they ought to be put in a class by themselves under the name of acute epilepsy or eclamptiform attack. Such observers declared that these cases were much milder than the typical eclampsia; but this has not been borne out by modern observers, many fatal cases being reported where the symptoms and signs of kidney mischief and the post-mortem lesions found in these organs were very slight.

In most cases of eclampsia there has been no previous renal disease; again, many patients suffering from Bright's disease who ~~became pregnant~~ never develop eclampsia; recently, cases have been reported where nephrotomy was performed successfully and the patient afterwards became pregnant, but did not develop eclampsia; the other day a case was reported where a cystic kidney was removed during pregnancy and the patient went successfully through her labour at the full term; and, lastly, eclampsia is found only in a very small proportion of pregnant women whose urine reveals the presence of albumen.

Experiments have been carried out where urea was injected into the veins of dogs whose ureters had been ligatured, and it was found that death did not follow nearly so quickly as when all the solid constituents were injected under similar conditions.

Nowadays, urea is regarded as the result of proteid metabolism; and its appearance must be taken as pointing to that production being carried out under normal circumstances.

THE TRAUBE-ROSENSTEIN THEORY.

The next theory of importance is that which goes by the above name: it was suggested by Traube to account for the convulsions of uraemia in general, and adapted by Rosenstein to explain those of puerperal eclampsia in particular.

The observer in question believed that eclampsia was produced by a condition of cerebral anaemia which results from the changes produced in the blood by the pregnant condition. In pregnancy the blood is in a more hydraemic state than ordinarily, and this condition would be aggravated by the appearance of albumen in the urine. Along with this, is found increased arterial tension, - due partly to hypertrophy of the left ventricle during pregnancy, - and this would naturally be aggravated by labour. The combined result of this would be a state of temporary hyperaemia, - to be quickly followed by serous effusion, - and the swollen and oedematous brain tissue, by pressing on the small vessels, would cause anaemia. Convulsions were the result of anaemia of the motor centres, and coma arose from anaemia of the cerebrum.

But against this theory we have the following facts: (1) Eclampsia is infrequent in the serous cachexia; (2) hydraemia is not a special feature of eclampsia; (3) as an hydraemic condition is not an infrequent phenomenon of pregnancy, we would expect to find eclampsia developing much more frequently than we do; (4) Oldhausen never saw oedema in any of his cases, and also observed that hyperaemia was much more frequent than anaemia; (5) post-mortem examination does not constantly show oedema or anaemia; (6) the clinical evidences afforded by the pulse and pupils are not those of oedema (Spiegelberg); while experiments upon animals have demonstrated that to produce convulsions an enormous amount of fluid must be injected; the autopsies of these animals showing neither oedema nor anaemia, but only congestion of the brain; (8) also one would expect to find, in those cases where oedema was marked, that the fits would be more severe - but these cases are usually found to be the least severe, and many clinicians have found that in uraemia from Bright's disease, fits are most usually met with where the oedema is least marked: in other words, that the tendency to convulsions is in no way proportionate to the degree of oedema present.

Angus MacDonald, in 1878, - as the result of two careful post-mortem examinations of the brains of two patients who had died from eclampsia and in which he found anaemia of the cerebral substance, especially of the central parts, congestion of the meninges, serum in the ventricles, but no oedema or flattening, also a limited extravasation of blood in the anterior portion of the right corpus striatum where it drops down to form the lenticular nucleus, - believed that eclampsia was the result of an anaemia of matter, caused by the circulation in the blood of excrementitious (the result of altered renal function) - which, by irritating

the vasomotor centre, would cause contraction of the arteries and consequently anaemia of the brain substance, with a simultaneous congestion of the meninges and the collection of blood in the venous channels of the body. Experimental proof of this may be found in tying the carotids of dogs, or letting them bleed to death, when slight convulsions will be seen. Convulsions may also be seen in a person dying from excessive haemorrhage, and in certain cases of heart disease where there is a deficient supply of blood to the brain.

Herman declares that it altogether depends on whether the chest or skull is opened first on post-mortem examination whether the brain looks congested or anaemia. If the skull is opened first, the brain looks congested; while, if the chest is opened first and the great veins cut, the brain will be found to be anaemic.

NERVOUS THEORIES OF ECLAMPSIA.

There can be no doubt that pregnancy makes great demands on the nervous system, slight mental changes being so common that the public look upon them as peculiar to that condition. This nervous irritability or instability, as we shall presently see may be regarded in itself as evidence of a toxæmic condition. As one might expect in eclampsia, there have been numerous theories advanced in which an altered state of the nervous system was regarded as directly contributing directly to the eclamptic state. It is my intention to briefly describe a few of the more important.

~~The Neurotic Theory.~~

~~One of the earliest theories of eclampsia was that it is due to a disorder of the nervous system absolutely peculiar to the pregnant condition and seen under certain circumstances.~~

The Neurotic Theory.

There can be no doubt whatever, as we have already seen, that a neurotic temperament will predispose a patient to eclampsia; and in these cases where very little change is to be seen in the liver and kidneys, it is quite possible (as Herff holds) that, where there is an abnormally irritable cortical psychomotoric centre, very little toxic material might precipitate an attack. The nervous system is in many women during pregnancy in a state of high tension, and very unstable - as shown by the nervous disorders which make their appearance at this time. Blumreich and Zuntz have lately carried out experiments on rabbits, which they trephined over the motor area, and irritated the cortex by the application of creatin. They discovered that in pregnant rabbits convulsions were much more easily produced than in non-pregnant ones.

The Reflex Nervous Theory.

A theory, which received much support in its day and which in a modified form still has its advocates, is that during pregnancy, owing to the enlargement of the uterus, and its rhythmical contractions, which are known to occur during the later months of gestation, the uterine nerves are in a state of constant irritation, which, acting reflexly on the renal sympathetic nerves, gives rise to albuminuria and eclampsia. But if we regard these phenomena as the result of reflex action, how are we to explain the pathological lesions found in fatal cases? We also find that this disease, far from being more frequently seen in neurotic and highly-strung nervous women, is more often encountered in robust, strong, healthy females who are far from being what one might term nervous.

The Reflex Neurosis Theory.

In pregnancy, owing to the enlargement of the uterus and its contraction and retraction, the uterine nerves are kept in a state of continual irritation and tension. There is a close relationship between the nerve supply of the kidneys and the pelvic organs, and the supporters of this theory hold that the irritation of the uterine nerves, by reflex action on the renal and sympathetic systems, causes albuminuria and eclampsia.

But such theories as this all fail to explain the pathological lesions found in the various organs. While, again, one would look for the more frequent appearance of eclampsia in nervous and highly-strung women, it is very often in the robust and full-blooded healthy women that it actually appears.

Barnes's Theory of Exalted Nervous Tension.

Dr. Barnes has advanced a theory which, owing to his power as a writer and the lucidity and acuity of argumentation which he displayed in presenting it, has received considerable attention at the time. I give it here more as a curiosity than on account of any real value that it now possesses.

He begins by saying that "emotions take a large part in every act of the generative function. In short, emotional affectability is the measure of convulsive liability". According to him, nature provides against the period of labour by storing up a special supply of nerve force; and in pregnancy there is an increased irritability of the nerve centres, and with it a corresponding development of the spinal cord; this excess supply of nerve force, under certain circumstances breaks out into convulsions. The factors which lead to this explosion are the hydraemic state of the blood in pregnancy causing defective nutrition of the nerve centres with increase of vascular tension, and, owing to imperfect elimination of waste products by the kidneys and other excretories, the collection of poisonous substances in the blood.

Herff's Theory.

The theory of the nervous origin of eclampsia has lately been revived by Herff, who declares that if we compare the convulsive attacks in epilepsy, eclampsia, and uraemia, without regard to their etiology, we must admit that they are practically identical; also that it follows from this that the origin of the symptoms should be sought for in a special alteration in degree of the psychomotoric brain centres, and in the ganglion cells. This alteration, which he calls "degrees of eclamptic excitability", may be inherited or acquired by blood-poisoning, urate-poisoning, infection, and different pathological conditions (as maladies of the blood-vessels, etc.), or, finally, in consequence of the physiological irritation of gestation. He held, then, that the tendency to eclampsia was produced by certain unhealthy conditions of the nervous system, or by faulty development of the same - both of which might be hereditary. In these cases, as one would naturally expect, the disease would be more easily produced than in healthy women. Should this tendency to eclampsia exist in any woman, the irritation of pregnancy is sufficient to produce eclampsia.

THE ALBUMINURIA OF PREGNANCY.

There is hardly any condition which has given rise to so much discussion and investigation as the changes which occur in the kidneys during pregnancy. This question has a very important bearing upon the etiology of eclampsia; and I think that, after the discussion of albuminuria of pregnancy and its cause, one may say that it is almost impossible to draw any boundary line between it, - be the case of albuminuria severe or slight, - and eclampsia, and that they are probably due to the same cause.

I shall here describe shortly the most important renal changes found, and the most prominent views relating to the production of these, discussing at the same time one or two of the theories of the etiology of eclampsia, which will, in the course of that discussion, present themselves for criticism.

FREQUENCY OF ALBUMINURIA IN PREGNANCY.

There is an extraordinary difference of opinion regarding the frequency of appearance of albumen in the urine of pregnant women. Ingerslew collected 100 cases, comprising compilations from various sources, where there was no trace of albumen in the urine. The cases of Oldhausen and Dührssen showed albuminuria to the extent of 98%. German observers put the frequency of albuminuria at from 1.5 to 12%, while one French observer states that, in 50% of pregnant women, a slight degree of it was found during the latter half of pregnancy, while it was nearly always present during labour. Some recent observers, too, found it of such frequent appearance as to justify them in saying that its appearance was the rule.

In those statistics, where it is represented as appearing in a high percentage, it will be found that the trace of albumen present was due to the contamination of the specimen by vaginal or urethral discharge. The statistics, then, of those workers who have made

observations on the frequency of the appearance of albumen in pregnancy, when they did not collect their specimens by catheterisation, should be rejected.

A good deal of painstaking research, carried through on thoroughly scientific principles, must be done before we can yet come to a distinct conclusion regarding this frequency. In looking over the literature of the subject, I find that many authors declare in a general way that they have found albumen in the urine in a certain percentage of the cases which they have examined; and they only mention in a very general way that it was frequently found during labour - at which period, one might add, the chances of the specimen being contaminated would be greater. They do not go ~~any~~ more definitely into the periods in which it is found; while they do not even consider the presence and, if present, the characters of the tube casts. As regards, too, the relation of the size of the uterus, etc., on the amount of albumen present in the urine, the same silence is usually observed.

Bonn has lately published the results of his investigations into 54,010 pregnancies: he believes that albumen is present in the urine in fully 80% of normal gestations. Albumen and casts, according to him, are present in at least 30%.

The most careful work on this subject, however, is that done by Ingerslew, who was very careful to guard against any contamination of his specimens. He tested the urine of 600 pregnant women, in 29 of whom he was able to demonstrate the presence of albumen - that is, in 4.8%. As regards the presence of tube casts, he found them in only 6 of the cases - that is, in 1%. Out of 153 women in labour, he was unable to demonstrate the presence of albumen in 50.

Саft, who only considered cases where a considerable amount of albumen could be shown, found in all his cases a percentage of 5.41, in labour in primiparae 32%, and in multiparae 22.6%.

The bulk of the French writers put the frequency of albuminuria at from 10 - 25%. Others point to its occurrence in 2% of all pregnant women who are free from organic disease of the kidneys at the commencement of pregnancy, and affirm that it occurs in nearly 50% during labour; the reason for this general appearance at that period, they say, is that there is increased arterial and intra-abdominal tension; that renal anaemia is brought about by reflex vasomotor spasm of the renal arteries, which results from the contraction of the uterus (Spiegelberg). Again, some hold that it is the result of the renal stasis which is occasioned by the act of labour. However, it may appear before there is any possibility of renal venous stagnation from pressure; and it may be then the result purely of reflex irritation, the intimate relationship of the nerve ganglia of the pelvis and the nerve supply of the kidney explaining this. Other theories will presently be considered.

That we have an hypertrophy of the kidneys during pregnancy is agreed on by most observers. The quantity of urine passed per diem is increased, while its specific gravity is decreased, being about 1014. These changes are the result of increased arterial tension. As the result of increased work (due to augmentation in the number and size of the vascular channels in pregnancy, and a rise in intra-abdominal pressure brought about by increase in size of the pregnant uterus and insufficient expansion of the thorax and compression of the lungs), we have slight hypertrophy of the heart, and this helps to bring about the increased arterial tension of the pregnant state.

The chlorides in the urine of the pregnant woman are slightly diminished, and the phosphates and sulphates more so, owing to their being used in the development of the foetus. Generally, after the termination of normal pregnancy, there is an abundant diuresis which continues for two or three days - the urine for the first day or two having a specific gravity below 1020, but, after the third day, it is usually above 1020. In cases where there is marked intra-abdominal pressure, this diuresis may be even more marked: probably in these cases the abnormal pressure has interfered with the function of the kidneys; but, when this is relieved, the renal organs act even more vigorously till the normal condition is restored. Acute nephritis may, of course, occur during pregnancy.

All are agreed that the albuminuria of pregnancy is only a temporary condition, as it disappears rapidly, as a rule, after delivery:

in a small number, however, the kidneys do not recover, but pass into a state of chronic nephritis. Oldhausen has described a case of eclampsia where the urine showed much albumen and tube casts at labour; five days after delivery the patient died; and on examining the kidneys at the autopsy he was unable to detect any lesions there. It must therefore be the result of some condition which acts temporarily, and which is benefited by emptying the uterus. Albuminuria, again, is found in many women who go through pregnancy and get over their confinement without the development of eclampsia.

RELATION OF THE ALBUMINURIA OF PREGNANCY TO ECLAMPSIA.

Dührssen reported that, out of his 195 cases, albumen was found in 189, or 96%; present in large quantity in 121 cases, or 62%; while casts were observed in 121 cases, or 65%.

Again, Hoffmann, out of 5,000 cases, found 137 where albumen was present in considerable quantity, and out of that number 104 developed eclampsia.

PHYSIOLOGICAL ALBUMINURIA.

Some look upon the albuminuria of pregnancy as a physiological process, and as allied to those cases in which, under certain circumstances, albuminuria develops in healthy men and women. In the normal condition of the kidney, only the water and salts are allowed to filter through from the blood. If albuminous material appears, it is certain that there are certain changes going on in the nutrition of the epithelium of the capillaries of the glomeruli or in the cells surrounding the latter. This change, however, may be slight and only transitory; and it is probably caused by differences of blood pressure, irritating articles of diet, and other factors. This transitory or temporary presence of albumen in the urine is called "functional or physiological albuminuria", and it may be seen in healthy men and women as the result of increased work or exercise, changes of climate, after cold baths, and following the ingestion of food rich in proteids. We know that we may meet with this transitory albuminuria in adolescence: here it is only found at certain periods of the day, or after exertion or a hearty meal. It also appears in pyrexia, even in some cases of tonsillitis, as well as in dyspeptics. Some cases of albuminuria in pregnancy may be explained in such a manner; but we must look for other causes, especially as we find evidences of changes in the cortex of the kidney, with oedema and the appearance of certain tube casts in the urine, which are seldom found in a typical case of functional albuminuria, where the tube casts are usually of the hyaline variety.

THE MECHANICAL THEORIES OF ALBUMINURIA.

The Pressure Theory.

The first theory advanced was that the gravid uterus pressed upon the inferior vena cava, the iliac veins, and upon the kidneys - the vessels of the latter causing passive congestion of these organs. In some cases of ovarian tumour the pressure exerted on the renal vein gave rise to a congestive condition of the kidney, with the appearance of albumen in the urine - which condition disappeared after operation. This theory at one time had the support of those who regarded it as very convenient to explain the causation of eclampsia. But against this is the fact that in valvular disease of the heart, and in other conditions which may give rise to great venous congestion, no convulsions appear. Further, in cases where pregnancy was complicated by the presence of a tumour, and where the combined pressure resulting would be far greater than that (e.g., in twins or severe cases of hydramnios), eclampsia, although frequently met with in the latter conditions, is seldom met with in the first. Again, the changes seen in pregnancy and eclampsia are not congestive in type, but more those of degeneration.

Halbertsma's Theory.

The last-mentioned theory was gradually set aside, by those who believed in the mechanical causation of albuminuria and eclampsia, in favour of that of Halbertsma. From a study of eclampsia he found that the gravid uterus, by increasing intra-abdominal pressure, presses upon the ureters, forcing them upwards, distending and distorting and compressing them, and, by interfering with their function, occasioning urinary stasis which would lead to alterations in the structure of the kidneys. The urine, however, does not contain albumen if the obstruction of the ureters is not complete from the onset.

Albuminuria is seldom found before the twenty-third week, that is to say, before the uterus attains sufficient size to cause pressure.

Albuminuria is found more frequently in primiparae than in multiparae - especially in very young or elderly primiparae. It is also more frequently observed in multiple pregnancies than in single: here, of course there is, besides increased size of the uterus causing increased pressure, increase of metabolism throwing more work upon the kidneys.

Albuminuria is found so frequently in hydramnios that some observers see in it the cause of that condition. Halbertsma declared that if other abdominal tumours rarely cause kidney changes, it is because they are not developed as the uterus is between the ureters. His statement that dilatation of the ureters is found has not been proved by other observers. He declares, however, that all that is wanted to interfere with the flow of urine is a paresis of the ureters causing decreased peristaltic action. Large fibroid tumours of the uterus have been known sometimes to cause changes in the urine, kidneys, and ureters almost identical with those produced by the pregnant uterus. Halbertsma found it difficult to reply to those critics who asked for an explanation of the appearance of albuminuria and eclampsia during the puerperium. More recent supporters of this theory, however, have advanced very plausible theories regarding this difficulty; and these will receive due attention later on.

An argument against this theory is that under appropriate treatment albumen may disappear from the urine, or the patient may develop eclampsia and recover, and, along with her child, eventually reach full term. In both cases the uterus continues to grow larger, and still no pressure symptoms arise. Again, albuminuria and eclampsia may occur in pluriparae who are quite free from either in their first confinement. Although it is probable that the pressure of the gravid uterus on the ureters interferes with their function and causes renal changes, thus contributing to the production of albuminuria, one must admit that there are other conditions at work.

With regard to the production of eclampsia, we may admit that it may determine an attack, but that it is the essential cause of this disease no clear proof has been advanced. The symptoms of the latter are in no way related to an attack of uraemia. In the uraemic attack the coma increases, and there are no intervals in which consciousness returns; while, again, the temperature in the great majority of cases, instead of progressively rising, as it usually does in eclampsia, falls and may become subnormal.

Many cases have been recorded since Winckel first drew attention to the fact that, on the death of the foetus in eclampsia, the fits disappeared notwithstanding that the amount of pressure would remain the same.

Fehling explains the amelioration of the condition of the eclamptic patient on the death of the foetus by the fact that on this happening, which is really due to a lack of oxygen and placental infarction, there is a lessened quantity of liquor amni, the placental circulation stops, giving rise to a lessening of pressure, and allowing the kidneys to recover.

Again, Spiegelberg had a case in which eclampsia developed in extra-uterine pregnancy; when the foetus died no more convulsions appeared.

Cornil and Ranvier pointed out a fact, which has received abundant confirmation by modern observers, that in a large proportion of the women who have died from uterine cancer, - although obstruction of the ureters is present to a marked degree and accompanied very often by hydronephrosis, - uraemic convulsions were never encountered.

As regards the influence of pressure on the production of albuminuria and eclampsia, a most interesting case has been reported

by Haultain where he removed an enormous ovarian tumour, which was larger than the product of a full term pregnancy. In this case no albumen appeared in the urine. The other ovary was left, and the patient afterwards became pregnant. She then developed severe albuminuria, and eclampsia supervened during the seventh month. She recovered, and again became pregnant, her pregnancy and labour this time, however, being normal.

Lastly, in post-mortem examinations of patients dying from eclampsia, dilatation of the ureters is absent in the great majority of instances; and Schmorl declared that he found the ureters in eclampsia not more often dilated than in normal pregnancy; while, again, they have been found markedly dilated in women who have never had eclampsia.

The reasons why I have given this extended description of this theory with the arguments in favour of it are, first, that in doing so I think I have disposed of the urinaemic theories of the production of the disease; and, secondly, because there has lately been a revival of Halbertsma's hypothesis, in a modified form, by certain writers, as satisfactorily explaining the causation of the eclamptic condition.

Webster's Theory.

Webster, in frozen sections, found that the pregnant uterus moulds itself accurately along either side of the spine, more especially at the brim of the pelvis; and that when the intra-abdominal pressure is greatly increased, the general pressure on the uterus must also be increased. In the last weeks of pregnancy, the foetal head in primiparae lies in the pelvic cavity; and, if the pelvis be a justo-minor or funnel-shaped in type, or the head be abnormally enlarged or ossified, it is nearly impossible for the ureters to escape being compressed against the pelvic walls; and, even if this did not cause a local interference, there still might be a dangerous pressure resulting in paresis. Webster's answer to Halbertsma's critics - who declared that it was impossible to explain the occurrence of renal disturbances and eclampsia in the puerperium by his theory - is that his frozen sections, after the birth of the child, also showed that the post-partum uterus, in its contracted and retracted state, fills the greater part of the normal pelvic cavity so firmly as to form a ball-plug, which compresses all the extra-uterine tissues against the pelvic wall, and this more especially for the first two or three days of the puerperium, after which eclampsia is rarely found. Halbertsma and others have shown that pelvic inflammatory exudates might, by compressing the ureters, cause renal disturbances in the puerperium.

Kundrat's Theory.

Other modern supporters of Halbertsma are Herzfield and Kundrat.

The latter pointed out that if the aorta bifurcated either higher or lower than normally, it would displace the ureters, and thus expose them to pressure by the foetal head as they cross the brim of the pelvis. Normally the ureters are protected, especially by the bifurcation of the aorta; again, the right ureter is more liable to pressure than the left as it crosses the external iliac artery at a lower level and enters the pelvis at a greater angle. The fact that in primiparae the pressure of the hard foetal parts in the pelvis is longer in duration, would explain the more frequent occurrence of dilated ureters in this case. In regard to Kundrat's theory, we have to add that the ureters are usually only dilated so far as the common iliacs, and that in primiparae the foetal head has sunk into the pelvis some time before labour.

Herzfield's Theory.

All Herzfield's cases of eclampsia showed changes in the renal system, most of them also oedema of the brain, and changes in the liver, spleen, and cardiac muscle. In 18 cases (i.e., in 22%) he found dilatation of both ureters during their whole length, as well as a dilatation of the pelves of the kidney - this was in primiparae where the fits came on during labour. Such change was, however, never found in fatal cases in multiparae, nor in primiparae where eclampsia occurred in pregnancy, or in the puerperium. This condition, Herzfield declared to be due to a gradual compression of the ureters by the

growing uterus and the foetal head - at the point where they cross the pelvic brim at the division of the common into the internal and external iliac arteries. From this spot downwards the ureters are normal in size. In the normal state this would be impossible; but where, as was pointed out by Kundrat, the bifurcation of the aorta occurred higher or lower than normal, the ureters would be more exposed to pressure.

In these theories of the mechanical causation of eclampsia and albuminuria of pregnancy, the head of the child is regarded as being of great account in exercising pressure; and some observers have vaunted the opinion that it was only when the head presented one could have eclampsia develop; but we know that eclampsia may occur in transverse and breech presentations. Dr. Neil has reported a remarkable case of eclampsia occurring where the presentation was of the former variety: as soon as the malposition was rectified, the convulsions ceased.

Although very few now believe that pressure on the ureters is the sole cause of eclampsia, I think we must admit that in a certain percentage of cases it may produce albuminuria, and also that it plays a very important part in embarrassing these organs - at least in a large number of instances.

ALBUMINURIA THE RESULT OF TOXAEMIA.

It is now generally agreed that the albuminuria of pregnancy is the result of the circulation in the blood of some poison, or poisons, acting injuriously upon the kidneys and rendering them incapable of performing their actual functions of elimination in a satisfactory fashion - the kidneys being at the same time handicapped by the increased intra-abdominal pressure, which itself may be looked upon as a contributory cause of that condition.

Before going any further in this discussion, I think it would be advisable at this juncture to have a clear idea of the changes most usually found in the KIDNEYS and in the LIVER in pregnancy, in order that the modern theory of albuminuria and eclampsia may be the more readily comprehended. A great deal of the modern work on this subject owes its inception to the researches of Virchow, in 1848. He showed that albuminuria and the renal and hepatic changes in pregnancy were the result of altered maternal metabolism. Most observers are agreed, moreover, that there is a slight hypertrophy of the kidneys during pregnancy in pregnancy, owing to increased work.

Leyden, in 1886, showed that the kidney of pregnancy was quite distinctive in character; and, instead of showing inflammatory changes, - as was formerly thought, - he showed that the lesions were of a degenerative type. In three fatal cases this observer found the kidneys large and pale, the cortex yellowish and dull. On microscopic examination, fat, present in large drops, was seen in the kidneys - especially in the convoluted tubules, and to a less extent in the glomeruli and the Malpighian capsules. This fat was to a great extent dissolved when the kidneys were placed for some time in alcohol; and, when sections were cut and microscopically examined, it was found that the kidney was practically normal.

We have already observed that, in the great number of cases where albuminuria is found during pregnancy or where recovery takes place after eclampsia develops, the kidney condition rapidly disappears, while only in very few cases does the damage done last permanently. The toxins (we shall discuss these and their sources of origin later on) may act directly on the cells of the tubules, or they may so irritate the arteries that they are constricted, and consequently the nutrition of the tubules is interfered with. The changes found are almost identical with those produced by certain poisons.

LESIONS OF THE LIVER.

These are constantly found, and vary greatly in both severity and extent. A certain degree of fatty degeneration is said to be the rule in pregnancy, and this in a few cases may pass into a condition of acute parenchymatous hepatitis. Necrosis may also be seen in minute areas; while, again, this may be so general as to occasion the development of acute yellow atrophy of the organ. Lower (Ueber die Bedeutung der Icterus Gravidarum, Zeit.f.Geb.u.Gyn., 1886, xiii.,

169 - 185) found that out of 143 cases of fatal acute yellow atrophy no less than 30 were in pregnant women. It was this condition that gave rise to the idea that eclampsia was the result of a toxic condition, and also because icterus gravis was so frequently found in pregnant women. It is a well-known fact that epidemic jaundice is peculiarly fatal to the pregnant: on that account Vinay believed the liver to be specially vulnerable in such persons. A great many observers have shown that in pregnant women disturbances of the liver are of common occurrence, the natural condition of the liver at that time being one of congestion; and Tarnier has shown that in nearly all pregnant women who die, changes will be found in this organ - mostly of the nature of a fatty infiltration of the hepatic parenchyma. The liver is frequently found enlarged and tender on pressure during pregnancy; but, in cases where toxæmia is very virulent, we find instead that the liver is much reduced and destroyed. We sometimes find jaundice develop: sometimes it appears well on in pregnancy, probably as the result of greatly increased intra-abdominal pressure, or as a sequel of duodenal catarrh. Ahlfeld reported a case where icterus was noted four times during different pregnancies in one of his patients (Lehrbuch der Geburtshilfe, ii. Aufl., 1898, 239 - 242).

Although the functions of the liver are still but somewhat imperfectly understood, we know sufficient to recognise that it is not only the most efficient scavenger that the human organism possesses, - purifying the blood and rendering highly toxic substances inert, and thus defending the system against the inroads of toxins actually manufactured within our bodies, - but that it is also intimately connected with the elaboration and renewal of the blood, and in the rebuilding of tissue. It has been well said that this organ "presides over metabolism". In the pregnant woman, the duties of the liver would be enormously increased. In the first place, its energy would be taxed to the utmost to build up the body of a foetus so as ultimately to reach seven pounds, or more, in weight. It would be handicapped more and more as pregnancy advanced, on account of the increased intra-abdominal pressure interfering with its function; and perhaps owing to the constipated condition of the bowels so frequently met with in pregnancy, the absorption of putrefactive material would occur with its consequent increase of work for the liver to do.

I think that nothing demonstrates so well the importance of the liver to the growing organism as the fact that in young children, - during the early period when growth is most frequent, and the chemical changes occurring in its body most active, we find it attaining a disproportionately large size. In pregnancy, too, it is not only the growth of the foetus which adds to the work of the liver, but the growth of the uterine tissue as well. It has been stated by Charcot that hepatic insufficiency is hereditary; and observers have shown that women of the bilious temperament are more liable to develop bilious vomiting, albuminuria, excessive pigmentation - all of which are symptoms of hepatic insufficiency.

Saint-Blaise has done a great deal of excellent work on this condition, which he calls HEPATOTOXAEMIA. The following are some of the signs pointing to the existence of that affection: Diminution in the amount of urea excreted, and increase in uric acid; presence in the urine of urobilin, albumen, indican, and peptones; presence of extractives, such as leucin, tyrosin, and xanthin; and glycosuria, due to the liver not being functionally active enough to perform its glycogenic function. Attention was drawn by him to the fact that the symptoms of hepatic insufficiency as given by Hanot, and the conditions found in pregnancy are very similar - viz., ptialism; nausea and vomiting; constipation and meteorism; disturbances of vision; headache; somnolence or insomnia; polyneuritis; pigmentation, epistaxis, diminished urea, etc., as above; pruritus; icterus; lassitude; alteration of character; and mania.

THE TOXAEMIA OF PREGNANCY.

The modern conception of the toxæmia of pregnancy, as bearing upon the etiology of eclampsia, is founded upon the work of Bouchard. As I shall refer to this again in detail later on, I shall touch upon it here only very briefly.

Toxic materials are constantly being eliminated from each cell

of the organism owing to the changes going on therein, and these are discharged into the fluids of the body, with the result that the system is threatened with auto-intoxication. But it has various lines of defence to guard it against that. To one line belong those organs which have to do with elimination - the intestines, skin, lungs, and kidneys. The liver has a double rôle to play - it reduces poisons floating in the body to an innocuous state, and at the same time excretes bile to prevent decomposition in the intestines, and aid in the digestion which goes on therein. Other organs of defence are the spleen, thyroid, suprarenal capsules, and various glands: we shall consider these more fully later on.

In pregnancy, as a result of the increased chemical changes going on, the effete products of metabolism would be greatly increased, owing to the building up of the foetus and of increased uterine tissue. Again, the toxins may be derived from the foetus, or be even placental in origin. The poison and its source, or sources, will likewise be considered at a later stage.

All this leads up to a short description of the symptoms and causation of a subject which is creating a vast amount of interest at the present time, and which, although not believed in by all observers, nevertheless, has had a bearing already on the essential etiology of eclampsia and its rational treatment; and has at the same time received such support and testimony from the clinical experience of numerous practitioners that it cannot be ignored. I refer to the view that in pregnancy we have a condition of TOXAEMIA, and that the so-called "minor symptoms" of pregnancy, - such as vomiting, salivation, neuralgia, constipation, and changes in the temperament, - are nothing more nor less than evidences of slight toxæmia. Although these symptoms are very common, it does not follow that they are the normal conditions of pregnancy, for we meet many women who go through pregnancy without complaining in any way. With the advance of pregnancy these minor manifestations usually pass away - we find, indeed, that compensation has been established, the organs which have either to do with the destruction of the toxins (e.g., the liver), or those which have to do with their elimination (e.g., the kidneys) doing more efficient work. Should any of the organs of elimination fail, then, of course, toxic products increase in the blood, and more work is thrown on the liver and other defensive structures. If these organs are not able to cope with this increase of toxins, one might break down, and we would have symptoms of poisoning appear. For example, the kidneys may break down from the inefficiency of the liver throwing more work on it than it is capable of doing, with the result that we have the appearance of albuminuria, or even of eclampsia. Vomiting, salivation, dyspepsia, and constipation are all minor manifestations of toxæmia. The liver may become enlarged and tender, and jaundice sometimes develops. If the poison is more violent, we have the more serious conditions produced - e.g., acute hepatic toxæmia and acute yellow atrophy of the liver.

The skin is often the seat of pigmentary deposits, sometimes so intense as to simulate Addison's disease. Pruritus is common, and so is herpes. Probably all these cutaneous disturbances are the result of the action of the poison on the nervous system, as it is there we find symptoms most characteristic of the toxic state. An increase in excitability, restlessness, insomnia, delirium, or, again, mania or melancholia may be seen, or even somnolence and stupor. Peripheral neuritis (polyneuritis) may be met with, and cases of myelitis have been described. It is generally agreed that the excretion of urea is a very good guide to the intensity of the toxicity. It is low at first, but increases with pregnancy. Should toxicity increase, the percentage of urea eliminated will fall. Glycosuria also is often seen.

The vomiting of pregnancy was really the condition which led observers to regard it as the result of a toxic condition; and what led more directly to this conclusion was the fact that in some cases of hyperemesis gravidarum multiple neuritis would develop; and we have seen that the nervous system is peculiarly susceptible to toxins.

That there was some relation between pernicious vomiting and disturbed hepatic function was suggested by the fact that not a few women, in the last stages of the disease, showed an icteric hue of the skin, or developed marked jaundice. Stone and Ewing, two American

observers, declare that, from the results of their post-mortem examination of patients who had succumbed to pernicious vomiting, it is their belief that the vomiting of pregnancy and eclampsia are manifestations of the same toxæmia; and the latter they attribute to primary disturbance in the hepatic system. In a certain proportion of cases of hyperemesis gravidarum, lesions are found identical to those seen in acute yellow atrophy and icterus gravis: these we have already described. Williams, again, believes that there are at least two toxæmias of pregnancy - probably one giving rise to the vomiting of pregnancy and acute yellow atrophy, and the other to eclampsia. He bases these conclusions more especially on what he found at his post-mortem examinations. In eclampsia the lesions begin in the portal spaces, and invade the lobule towards the centre; whilst in hyperemesis gravidarum the necrosis begins in the centre of the lobule and spreads peripherally, never involving the portal spaces. Again, in eclampsia the total amount of nitrogen is greatly diminished, while the ammonia co-efficient remains practically normal. In vomiting, on the contrary, in spite of the scanty urine, the amount of the total nitrogen remains nearly normal, while the ammonia co-efficient is greatly increased. Generally speaking, a high ammonia output is a favourable prognostic sign in eclampsia, but a very ominous one in vomiting. It has not been proved that all cases of vomiting of pregnancy are toxæmic in origin, for it is undoubtedly true that some are reflex, - arising from some such abnormal condition as retroflexion of the uterus or the presence of ovarian tumours, etc., - while others are neurotic in origin. The bulk of the cases, looking through recent literature, I must conclude are toxæmic.

Numerous theories have been advanced to explain the origin and nature of the toxic process. One of these we have already described - the hepatotoxæmia of Saint-Blaise and Pinard. Its intimate connection with eclampsia and acute yellow atrophy of the liver was pointed out above. Others, again, for example, absorption from the intestines of toxic substances, and the syncytio-toxin theory, - where we have an invasion of the maternal organism by certain foetal elements causing the toxæmic condition, - and the other theories of its causation we shall describe more particularly when dealing with the modern hypotheses of the etiology of eclampsia.

Regarding the theory of the toxæmia of pregnancy, might it not also explain not only the development of eclampsia in labour in a patient suffering from chorea gravidarum, but also the chorea itself? The chorea in one of my patients, at the 5½ month, got gradually worse, notwithstanding treatment, till the 7th month, when it suddenly disappeared - compensation had become established. It recurred just before labour came on prematurely at the 8½ month; while labour was in progression the patient had two severe convulsions. While the chorea lasted, I examined the urine frequently, and always found albumen present. During the early months of pregnancy she suffered severely from vomiting. After her next pregnancy she became melancholic but had no return of the chorea.

Very grave cases of the toxæmia of pregnancy have been reported where the patient rapidly passed into a comatose condition and died; and at the post-mortem examination the lesions found were almost identical with those observed in eclampsia. Also, a few cases, which have been termed "puerperal ~~eclampsia~~ without convulsions", where the clinical picture was identical with that of eclampsia, except that the convulsions did not appear.

Much work, however, still requires to be done before this theory can leave the domain of conjecture and become an established fact. When we come across a case of ~~the~~ albuminuria of pregnancy or of eclampsia, more attention I think ought to be paid to the history of the earlier stages of pregnancy, and also to that of the previous pregnancies, if there have been any. If we are to regard serious gastric and hepatic disturbances, and other conditions, such as marked constipation, headache, excessive pigmentation, etc., as evidences of the presence of the toxæmic state, it behoves us to be on our guard to pay close attention to these organs which protect the body against their attacks, or help in their elimination, lest eclampsia supervene.

In one of my cases of eclampsia, in an epileptic in the early months of pregnancy, she was under the treatment of a doctor in ~~Ticon~~ for severe vomiting: he tried many remedies to stop it, but failed;

and he was on the point of procuring abortion,- as the patient was rapidly losing ground,- when the vomiting suddenly ceased - compensation, as we saw above, had been established; the organs of the body now making a better stand against the toxic invasion. In ~~my~~ ~~my~~ my cases of post-partum eclampsia, obstinate constipation, with severe headache, and excessive pigmentation of the skin were marked features during pregnancy - the pigmentation, indeed, was so marked as to suggest Addison's disease. These phenomena, occurring in ~~the~~ patients who eventually developed eclampsia, are suggestive; and the theory of toxæmia of pregnancy, were we to accept such, undoubtedly presents a very plausible solution for their appearance and for the supervention of eclampsia. Although this theory of the toxæmia of pregnancy has not been accepted by all, most are agreed that the immediate cause of eclampsia is the presence of some toxic agent, whatever it may be, in the blood; but the bulk of evidence points to changes found in the nitrogenous metabolism.

This discussion may seem an absolute digression from the subject in hand; but the evidence I have brought forward in support of this suppositious toxæmia of pregnancy, and evidence, I may add, which has received support of the strongest kind from pathological and clinical observations, has a very close relation to the modern etiology of eclampsia.

LESIONS OF ORGANS IN ECLAMPSIA.

As a knowledge of the changes found in the post-mortem examinations of those patients who have died from eclampsia is most essential for the proper understanding of the etiology of this disease, I shall here briefly describe the most important of their number.

THE KIDNEYS IN ECLAMPSIA.

Although in certain cases very little is to be found post-mortem in the kidneys, yet, on looking over the reports of different observers, we find that in the vast majority of instances these organs are found to be injured by the action of the poisons which are circulating in the blood: consequently, we shall first of all describe the changes in this organ.

The PATHOLOGICAL CHANGES in the kidneys are by no means constant; for, as we have already noted, some cases of a very severe type leave the kidneys practically uninjured; while, again, cases have been seen where there was little or no albumen in the urine, although on post-mortem examination renal lesions of a very marked degree were observed. Charpentier collected 143 cases from the literature of this subject where there was no albumen in the urine at the time of the attack; and many observers have placed on record series of similar observations. The Germans lay much more stress on the lesions found in the kidneys than the French, who regard these in the liver as the more important: nevertheless, I think that all are agreed that in the bulk of the cases (nearly 95%) changes, of a more or less marked degree, are met with in the kidneys.

It was formerly supposed that the changes produced were inflammatory in character, but we now know that they are of the degenerative type described under the "kidney of pregnancy". This degeneration may go on to actual necrosis, so that we may find in a given case all changes from a cloudy swelling of the epithelium, through fatty changes, to actual necrosis, or even severe general infarction. These changes are perhaps more marked in the epithelium of the convoluted tubules, although they may also be found in other parts of these structures. Sometimes these changes are particularly well-marked in the cortex. The epithelium is often seen in a finely granular condition - also a degenerative change. The lumen of the uriniferous tubules is often found filled with debris composed of degenerated epithelial cells, red blood-corpuscles, and casts of various kinds. Haemorrhages into the substance of the kidney have also been noticed - only, however, very seldom. Still, blood is frequently found

in the interstitial tissue and in the tubules; and it was this perhaps which led the older observers to regard the changes found as being of an inflammatory type.

The microscopical changes found in the kidney, in fact, are, as St. Blaise first pointed out, very like those seen in infectious diseases, where a coagulation necrosis of the epithelium is the characteristic lesion. Again, the whole organ may be in a state of acute nephritis. Fibrinous and hyaline thrombi are occasionally found in the glomeruli, while the vessels of the latter may show signs of commencing hyaline change. Thromboses of the capillaries and of the smaller vessels are frequently met with; and areas of infarction and necrosis - either microscopical in size or visible to the naked eye where they have coalesced to form large areas, and similar to those found in the liver - are also observed in the kidney. Their origin and appearance will be fully described when we come to treat of the hepatic lesions.

As regards the naked-eye appearances, the kidneys may be normal, but in these cases the microscope generally reveals what I have described above; and petechiae may be noticed on the surface. Again, the kidneys may be found enlarged and congested, and in a state of acute parenchymatous nephritis; and in severe cases haemorrhagic inflammation, with infarction and extravasations of blood. Furthermore, there may be, in old-standing cases, established lesions found, - which, by handicapping these organs, have predisposed the patient to eclamptic attacks, - such as cystic kidney, chronic interstitial or parenchymatous nephritis; hydronephrosis is either alone or associated with what Halbertsma and other observers regard as a common lesion - dilatation of the ureters. In one case of eclampsia, where there was complete anuria, Sippel found one of the kidneys very much enlarged with the capsule stretched very tightly over it. On cutting through the capsule, the parenchyma sprang out in a manner showing that it had been retained within the capsule by great tension. He regarded the case as one of renal glaucoma, and as due to pressure of the ureter at the brim of the pelvis.

The lesions found in the kidneys are in all probability part of the many changes produced by the poison of eclampsia - although other conditions, such as we described under the mechanical theories of albuminuria and eclampsia, may sometimes help in their production. Another theory is that advanced by Volhard, who declares that they are brought about by the deposition of emboli in the vessels of the kidney, resulting in an anaemia and consequent degenerative changes - these emboli being produced by the action of a ferment in the blood which causes coagulation.

As we have before noted, eclampsia has occurred and post-mortem examination showed no signs of changes in the kidneys; while, again, in marked disease, and in recent cases, removal of the kidney by operation has not led to eclampsia on the superintention of pregnancy. We are forced to conclude, then, that in the vast majority of cases the renal changes which are certainly present are not sufficiently marked or constant in appearance as to warrant one in regarding them as the characteristic primary causes of eclampsia.

THE URINE IN ECLAMPSIA.

The urine is generally considerably diminished in QUANTITY: in some cases there may be complete anuria. Cases are met with, however, where there is no diminution at all in the urine secreted. Herman tells of a case where, instead of diminution of urine, there was polyuria: in this case the amount of albumen present increased during the fits, and the percentage of urea, notwithstanding the increased amount passed, was much below the average of health.

The urine may at any time show such great and rapid variations in its constitution that it is necessary to examine it very frequently, if one wishes to arrive at anything like an accurate conclusion. In eclampsia it is very often a matter of no inconsiderable difficulty to collect the urine: the proper method to adopt is frequent collection by the catheter, every portion of the urine being separately examined.

In pregnancy it has been found that great variations in the rate of excretion, both of water and salts occur - especially in primiparae. During labour there is diminished diuresis. After normal labour, during the first day or two, more urine is passed than

subsequently. In cases where there has been marked intra-abdominal pressure, this diuresis may be even more marked: probably in these cases the abnormal pressure has interfered with the function of the kidneys; and when this is relieved, the kidneys - having increased work to perform - act more vigorously until a normal condition is attained.

ALBUMEN frequently appears in normal labour, but it disappears during the first 24 hours. When recovery takes place in eclampsia, we find a rapid increase in the amount of urine passed, and an increase in the amount of urea, while the amount of albumen rapidly decreases.

As regards the kinds of albumen present in the urine, it is necessary to note that there are several - the two most important being serum albumen, - which is not precipitated by magnesium sulphate, and which is more diffusible than the other variety, its presence consequently denoting more serious injury to the renal cells, - and serum globulin or paraglobulin, which is, however, precipitated by magnesium sulphate. The relative proportions of these varies; and the preponderance of the one over the other has been regarded by some as constituting a valuable prognostic sign. Herman found that, in cases where there is much serum albumen and little paraglobulin, the outlook was graver than where there was much paraglobulin. He says, moreover, that in two cases in which albumen was precipitated, this substance was mostly composed of paraglobulin, and both recovered. In three in which the amount of paraglobulin was less than in the majority, two died, and in one the renal disease persisted after the confinement.

In most cases of eclampsia albumen will be found present in varying amount. Very often the urine becomes solid on boiling. But many careful observers have placed on record cases of eclampsia where albumen was altogether absent from the urine, or present only in minute quantity. Nearly all these cases, however, showed a decrease in the amount of urea eliminated during the fits. Even in the same case, the amount of albumen present has been found by different observers to fluctuate considerably, even within a few hours; and this substance is often found increased in amount after a convulsion.

In eclampsia **TUBE CASTS** may be present in the urine in great variety. There are three kinds of tube casts:

1st. Those composed of cellular elements - red blood-corpuscles, leucocytes, or epithelial cells.

2nd. Casts of degenerated cellular elements and unorganised protoplasm - granular casts.

3rd. Casts composed of a homogeneous hyaline material - hyaline casts.

Each of these varieties carries with it its own peculiar significance. In normal urine hyaline casts may be found: in fact, as we have already seen, it is now regarded as almost a truism that the presence of a few hyaline tube casts, like that of a trace of albumen, unless found in repeated examinations, does not of itself mean anything out of the ordinary. But it is quite different with the other tube casts, which all point to renal mischief. Furthermore, we may find in eclampsia that they are absent altogether; or, again, they may be present only in one variety and few in number; while, moreover, all the varieties may be found in abundance.

SUGAR may sometimes be found in the urine in eclampsia. This, in the form of lactose, is not infrequently found in the urine of pregnant women towards the end of gestation, or during lactation. Its source there, and also in eclampsia when it occurs, is generally regarded as the breasts, lactose being absorbed from the milk which they contain: this may happen when the supply of milk exceeds the demand, or when any obstruction exists to its free outflow through the nipple.

In a case of mine, of post-partum eclampsia, I found sugar in the urine towards the end of pregnancy, and there was practically no milk in the breasts. In this case there was a trace of albumen in the urine, which I examined frequently, and which was also not much diminished in quantity, even during the eclamptic seizure.

According to Stumpf, - whose theory that the disease is caused by acetone we shall discuss presently, - sugar may be frequently found in the urine of eclamptics if searched for.

The sugar may be in the form of glucose. This we now know is not merely confined to diabetes, for it appears during convalescence

in many acute febrile diseases, and also in various injuries and affections of the nervous system. It has not any special significance in eclampsia, unless perhaps to show some interference with the glycogenic function of the liver.

An increase of CHLORIDES occurs in diabetes, during the convalescence from acute fevers, and during the post-convulsive stage of epilepsy. Diminution or absence of chlorides is found in cases of inanition or starvation, however produced; in all fevers; and in all cases of albuminuria, etc.

Zangemeister, after many minute and careful examinations, has found that the chlorides of the urine behave differently to the other salts. In pregnancy all the salts are considerably diminished, except the chlorides, which only undergo slight diminution. The chlorides, therefore, are present in greater proportion than the other salts at that time. He also found that in eclampsia during the attacks we have marked diminution of chlorides, which same commenced at the start of the convulsions; on the cessation of the attacks, we have an increased secretion of these substances. This diminution in the excretion of the chlorides, with the lessened quantity of the urine, he regarded as the most constant and most marked change which the urine undergoes in the course of eclampsia. He quotes Naumeyer and Schreiber's experiments on brain pressure in support of the view that the cerebral phenomena of Eclampsia can be explained by increased intracranial pressure, and especially by variations in the latter; and he says that these in turn depend upon high general blood pressure brought about by arterial contractions. In fact, all the phenomena of eclampsia may be due to arterial spasms giving rise to periodic anaemia of the kidneys, liver, pancreas, and brain. Since eclampsia occurs in pregnancy, labour, and the puerperium in certain instances, - that is, when rhythmical uterine contractions are, or may be, present, - it is probable that arterial spasms are brought on with the uterine pains, or by them. Whether the irritation of the vasomotor centres depends upon a toxin, or on a reflex stimulus, is uncertain. The poison of eclampsia certainly does not arise from imperfect action of the kidneys, nor is it eliminated by them; the organic changes can be explained without the necessity of supposing that they are due to a toxin. Against the toxæmic theory it may be further urged that the affection is generally more severe the earlier in pregnancy it makes its appearance, and that convulsions often come on with the labour pains. In conclusion, Zangemeister admits that the arguments are far from proving that eclampsia cannot be due to toxæmia, but urges that later researches should reckon more with the possibility that the affection may be, after all, a purely reflex neurosis.

The diminution in the chlorides does not depend upon the constitution of the blood, since the quantity present in that liquid in eclampsia is not changed. The sudden variations in the secretion of the chlorides is brought about by sudden changes in the renal circulation, owing to the contraction of the muscles, such as always occurs to a certain extent in labour.

SULPHATES. Sulphur is present in normal urine, existing therein in two combinations: First, as the mineral sulphates (oxidised sulphur) in combination with sodium and potassium; second, as organic or ethereal sulphates (unoxidised sulphur) in combination with indol, skatol, and phenol. The proportion of the two is 84% sulphates to 16% unoxidised sulphur. The bulk of the sulphates is derived from the breaking up of albuminous substances in the body - only a small percentage of the mineral sulphates being derived from the diet of the patient. As one would expect from this, we find that in febrile diseases an increase in sulphates; and this also happens in wasting diseases like diabetes.

Zweifel found that the proportion of the sulphates to non-oxidised sulphur depended upon the severity of the attack. The more severe the attack, the greater the quantity of non-oxidised sulphur found. In puerperal albuminuria, the non-oxidised sulphur was present in greater quantity than normal, and he says that this increased till birth. Again, in uraemia the non-oxidised sulphur was present in more than double the usual amount. During convalescence from eclampsia, the non-oxidised sulphur, increases in amount. The only special significance, however, that this can have in eclampsia is that it points to deficient oxygenation in the tissues.

One of the earliest theories of eclampsia was that it was due to the defective elimination of UREA, which collected in the blood and gave rise to convulsions. Although this theory has been abandoned, urea has received fresh attention recently, but from a different standpoint. A great deal of work has been done as regards the estimation of the amount of urea present, and as to whether its presence in decreased amount in the urine may serve as an index of the eliminating power of the kidneys, and as a reliable indication of the disposition to eclampsia. By some it is regarded as the most valuable premonitory sign of eclampsia we have; and when, on examination of the urine of a pregnant woman, especially a primipara, the amount of urea excreted falls below a certain percentage, stimulation of the excretory organs should be proceeded with at once. But, more recent observations have shown that while diminution in the amount of urea excreted is a significant sign, still, it has not the overwhelming importance attached to it by some observers, at least in some cases.

The liver and kidneys are intimately connected with one another - the kidneys being, in a manner, the servants of the liver, as they excrete the waste products of the latter, and thus enable the liver to perform its function efficiently, and at the same time to keep the blood free from toxic products. Both may be thrown out of gear by the one, the liver, through disease, throwing an extra burden on the kidneys; while, again, the other, the kidney, through disease, by not eliminating the waste products, in a manner clogs the liver.

In the non-pregnant condition the amount of urine varies, as it depends closely upon the amount of proteid material consumed, and also partly on the body weight. The liver is now regarded as the organ which manufactures urea. Urea, then, is derived from the unused proteids of the diet, and also from the physiological waste of the body, or tissue proteids.

As some of the intermediate bodies in the formation of urea have been supposed by certain observers to be the principal poisons in eclampsia, it will be necessary to describe as briefly as possible the formation of urea. This, I may add, has not yet been settled beyond dispute; but the chief steps I am about to describe are allowed by most physiologists. Convulsions seldom occur in acute diseases of the kidneys, except in the nephritis of infectious maladies where, of course, there is, as in eclampsia, a toxin in the blood.

Proteid material taken into the system with the food, after being acted on by the various juices in the alimentary canal, is finally absorbed into the portal system, and carried to the liver where it is formed into material which goes to build up the tissues, while there is elimination of the waste products.

By the action of the juices, then, proteid matter is split into: (1) Alkali albuminates; (2) albumoses; (3) peptones; (4) leucin and tyrosin (to a small extent); (5) skatol, indol, and phenol, which are formed by putrefaction - i.e., by bacteria - and excreted as ethereal sulphates. These are increased in quantity if there be any delay in absorption; and, as constipation is often a feature of pregnancy, they are often found in great abundance then.

The peptones in the liver are changed into a material which goes to nourish the body, and also waste products which are eliminated by excretion. These waste products are, firstly, leucin and tyrosin. The other waste products, which are ultimately built up into urea and uric acid which pass into the blood to be excreted by the kidneys, - these intermediate products are creatin, creatinin, amino-acids, and xanthin. These have ammonia for a base, like the vegetable alkaloids, and have been noted by some observers in the urine in eclampsia, and supposed by them to be the chief poison in that condition.

If the liver is in any way handicapped in its work of building up this urea, the final product, we find imperfect oxidation of waste products. If slight, excess of uric acid; if more serious, we would have retention of the intermediate ammoniacal products; whilst a more serious condition would lead to the appearance of leucin and tyrosin in the urine, as seen in acute yellow atrophy of the liver and in certain cases of eclampsia. Whitney and Clapp believe that these are the conditions in pregnancy and in eclampsia, namely, that there is less urea excreted, with an increase of ammonia and the

antecedents of urea. Zweifel has pointed out that, in eclampsia, the severer the case, as shown by the abundance of albumen in the urine, the less the relative amount of urea is related to the total nitrogen of the urine. In the normal condition there is about 86% of nitrogen in the form of urea in the urine. About 86% of the urinary nitrogen in the normal condition is found in the form of urea; about 6% as creatinin; 2% as uric acid and the xanthin bases; and the remaining 6% in varying proportion composed of hippuric acid, indol, and skatol, pigments, etc.

The excretion of urea may be regarded, again, as an index of the activity of tissue destruction: consequently, we would find it increased in acute febrile diseases. It is found increased in poisoning by certain drugs - e.g., phosphorus. The largest amount of all is seen in diabetes. Urea is decreased in various renal diseases; and also, as we might expect, in certain hepatic affections - e.g., cancer and cirrhosis. The amount of urea in the urine, then, furnishes an excellent gauge of the eliminating ability of the liver - a diminution of its amount indicates a retention of toxins.

In puerperal albuminuria and eclampsia the amount of urea excreted is very low. There is a great excess in the amount of urea the first few days after delivery. This is partly accounted for by the process of involution; but we must conclude, from the fact that there is no retention of urea or ammonia in the blood before labour, that it must be due to unoxidised material - these half-way products we have described above being oxidised after delivery. Herman has pointed out that this is most marked where abdominal distension was greatest. In eclampsia, where the case is going to end fatally, it is sometimes found that the urea progressively diminishes till death.

AMMONIA. The normal urine invariably contains small quantities of ammonium salts. When there is excessive production of acid, as in certain forms of diabetes or where mineral acids have been given, it has been found that the ammonia increases at the expense of the urea. And, conversely, where alkalies are given, the ammonia gradually disappears - its place being taken by urea. Seemingly, this is the method the system adopts to prevent an excess of acid - acid-poisoning resulting in excess of ammonia from its nitrogenous metabolism at the expense of urea.

Zangemeister holds that in eclampsia there is an excess of ammonia in the urine, resulting from an increase in the acidity of the blood. In acid-poisoning in animals, he pointed out that he found low temperature, dyspnoea, somnolence, and collapse. Zangemeister has received the support of Zweifel, who also considers that eclampsia may be produced by acid-poisoning, as in certain of his cases he found the urine alkaline; and a great many observers have demonstrated that, with the decrease of urea in the urine in eclampsia, there is an increase in ammonia and other antecedents of urea. Furthermore, many observers have found the urine alkaline during the convulsions, and from that urge the possibility of acid-poisoning being the cause of the disease.

The appearance of **LEUCIN** and **TYROSIN** in the urine, as they are seldom found in normal urine, points to acute lesions in the liver where there is considerable destruction of hepatic parenchyma, as in acute yellow atrophy and, to a less extent, in phosphorus-poisoning. Williams reported two cases where they were found in the urine in eclampsia, both of which patients showed symptoms pointing to hepatic disturbances (icterus and enlargement), and in one of which marked lesions were found in the liver, at the autopsy, similar in type to those found in acute yellow atrophy of that organ.

It is probable that these two bodies would be found more often in the urine, were they searched for, in that type of eclampsia where the various phenomena point to hepatic rather than to renal disturbances.

LACTIC ACID has been detected in the urine in eclampsia by one or two observers. Its appearance, however, does not seem to have any special significance, for it has been found after an epileptic fit, and also in albuminuria and pregnancy; while, again, physiologists have shown that it occurs in the urine after great muscular exertion, and also that in animals where the arterial supply has been cut off or diminished from an organ, where they are deprived of sufficient oxygen, that it appears in the urine. It is a product of proteid

metabolism, and the result of diminished oxidation in the body.

The normal urinary pigment, UROBOLIN, has been found in increased quantity in eclampsia by a few observers: such an increase is found in large haemorrhages, in some cases of cirrhosis of the liver, in other hepatic disturbances, and in cases of pernicious anaemia.

"Edsall found that as an index of danger urobilin was of no value. In half of the cases he examined it was in excess: it was present in one case of eclampsia and absent in two cases of severe albuminuria. It did not cause the death of the foetus.

"Merletti finds that during the last two months of pregnancy the quantity is three times as great as in the non-pregnant, and with the death of the foetus there is a marked increase" (Berkeley).

In those cases where icterus is found in eclampsia, or where there is any hepatic disorder, BILE may be found in the urine.

Massen reports the finding of LEUCOMANNs in the urine in eclampsia, but this experience has not been substantiated. Some hold that eclampsia is produced by the presence of these in the blood: I shall deal with them when I come to the condition of the intestine in eclampsia.

As regards those OTHER NITROGENOUS DERIVATIVES which are ordinarily present in the urine in small and varying proportions, - such as xanthin, creatin, creatinin, etc., - very little is known regarding their importance in the disease under consideration.

Lately, careful chemical investigation has proved that what were formerly regarded as PEPTONES are really propeptones or albumoses. They are found in the urine in many of the specific infectious disorders, as well as in the majority of septic inflammations; and several writers have told of their occurrence in the urine of eclampsia. They are often found during the puerperium, when they are common during the first eight or nine days. Any degenerative changes in the wall of the intestine would permit these diffusible proteids getting into the circulation.

THE LIVER IN ECLAMPSIA.

Next to the kidney, the liver is the organ in which changes are most frequently seen: in fact, they are of such common occurrence that one observer - Bouffede de St. Blaise - though that the disease was brought about by impaired hepatic function - what he termed "hepatotoxaemia"; and that the liver changes represent the primary lesion of the disease - the kidneys being secondarily affected.

In discussing the toxaemia of pregnancy, I showed how frequently are found manifestations of hepatic mischief, and also the relationship of both pathological and clinical phenomena of eclampsia to acute yellow atrophy of the liver. I have given a short account of the development of the modern pathology of that organ in eclampsia from the work of the older observers. Some of these - e.g., Frerichs, Vinay, and Braun - pointed out that in certain cases the kidneys were practically unaffected, and that the principal lesion was in the liver. In course of time, observers pointed out that the lesions found therein varied from fatty degeneration, through the different stages, up to extensive necrosis as seen in acute yellow atrophy.

Jurgens, as the result of careful examination of the liver in eclampsia, declared that the typical lesion in eclampsia was haemorrhagic hepatitis; and about the same time his observation was confirmed by no less an authority than Klebs.

Then came the brilliant work of the French school, which has, indeed, done so much to put the pathology of the liver in eclampsia on a sound basis, and likewise contributed largely to our knowledge of the etiology of this disease. Pilliet, in 1886, pointed out that the most prominent and characteristic changes in the liver were small haemorrhagic lesions, with occasional large ones. His work soon received support from Létienne, Schmorl, Bar, Bouffe de St. Blaise, and many others; and, from a perusal of the works of these observers, the following may be regarded as the most characteristic changes encountered in this important organ:

The liver is often enlarged, and sometimes to the naked eye appears to be in a state of passive congestion: much more frequently

it is of fatty appearance. Petechiae are frequent on the surface of the organ, and large haemorrhages under the capsule have been often encountered. Prutz describes one fatal case where the bleeding was so great that it burst through the capsule, and found its way into the peritoneal cavity.

On section, it has a marked mottled or marbled appearance produced by irregular areas of dark red (the haemorrhages), in the neighbourhood of which are yellowish or yellowish-white blotches, which are patches of haemorrhage from which the colouring matter has been absorbed, or areas of fatty degeneration or of necrosis. These areas vary from the size of a pin's head to large patches; and small haemorrhages, more particularly around the branches of the portal vessels or in minute necrotic areas, may be made out under the microscope: in the latter we find a mass of dead liver cells, blood-corpuscles, fibrinous tissue, and blood-vessels. The larger areas are the result of the coalescence of the smaller ones. Round about these areas, as is usually found in necrosis of any part, we have dilatation of capillaries, as well as an exudation of round cells. Some observers hold that masses of the dead liver cells may find their way into the circulation and cause characteristic lesions - embolism and thrombosis - in other organs.

These haemorrhages are regarded as minute infarctions of the smaller branches of the portal vein, but opinion is acutely divided as to how they arise. Some few observers hold that they are the direct result of the convulsions which cause rupture of the smaller blood-vessels. By far more numerous, however, are those who believe that the liver changes are the result of some toxic substance in the blood, which acts directly on the hepatic cells, causing fatty degenerative changes up to necrosis, more or less extensive in area according to the virulence of the poison.

As we shall see in discussing the various theories of the causation of eclampsia, a good deal has lately been done as regards the relation of the placenta to eclampsia. Some of the supporters of this view hold that the changes in the liver are the result of the deportation of cells (Syncytium and Langham's layer) from the chorionic villi into the maternal circulation, where they give rise to emboli and necrosis; while others believe that the cells secrete a ferment, which causes coagulation and thrombosis; while, again, Dienst holds that the placenta secretes a toxin, which in normal pregnancy is not permitted to enter the maternal circulation, but under certain circumstances finds its way there, and so leads to coagulation of the blood and destruction of the corpuscles. Liepmann (A Contribution to the Question of Treatment by Rapid Delivery, - Abstract from the Jour. of Obstet. and Gynaecol., March, 1904) sums up the question to the following effect: (1) In the placenta a toxin is formed which is absent from the normal placenta. (2) It is identical with the toxin of eclampsia; for the more the toxin that is absorbed from the organism, the less there is found in the placenta; and, conversely, the less the toxin that passes into the maternal organism, the richer the placenta is in toxin. (3) The toxin exercises a marked affinity for the brain cells, which neutralise it and are paralysed by it. (4) The toxin acts deleteriously on the renal parenchyma, as well as on the liver. (5) The injury to the kidney is secondary to the toxæmia: an already existing albuminuria may be markedly increased by the toxin. (6) The chronic epithelium appears to play an important part in the genesis of the toxin; the placenta is thus the place of elaboration and point of departure of the toxin.

The changes, however, in the liver in eclampsia are not absolutely characteristic; for they may be found, as we have already seen, in acute infectious disorders, phosphorus-poisoning, and in toxæmia from whatever cause arising. Changes in the liver in eclampsia also have been found almost identical with those observed in acute yellow atrophy of that organ. Williams, and some American observers, declare that the post-mortem changes furnish us with distinctive lesions for eclampsia and acute yellow atrophy. According to them, in eclampsia the lesions begin in the portal system and invade the lobule from the periphery towards the centre; whilst, in the other, the necrosis begins in the centre and spreads peripherally, never invading the portal system. But most observers in acute yellow atrophy find, in less degenerative parts of the liver, that it is the periphery of the

organ which is most affected. Bell, in a careful post-mortem examination of a case of eclampsia, found the degeneration most marked at the centre of the lobule. He also found that the amount of fat present is far more than is usually seen in acute yellow atrophy, where the granular debris is in excess of the fat. He also pointed out that there was none of the infiltration round the portal vessels, and formation of fibrous tissue seen usually in severe types of acute yellow atrophy. As I pointed out before, there are well-marked types of eclampsia where there are no lesions found in the kidneys, but considerable ones observed in the liver and spleen. Again, in other cases which ~~have~~ ~~discovered~~, no symptoms or signs of renal insufficiency may have been present, as in a case reported by Williamson. In this case, there were forty eclamptic seizures within twenty-four hours of delivery. The urine was not reduced in amount, the percentage of urea never fell below 1.6, only a slight trace of albumin was present, and there were no tube-casts. After the cessation of the fits, the patient became deeply jaundiced, and bile was found in the urine. From this and other cases, one is forced to the conclusion that we have two distinct types, at least, of eclampsia - one in which the most marked changes are in the kidneys, and this is the commonest type; and another in which the most marked changes are seen in the liver. Furthermore, it is not only pathologically that this distinction may be drawn, but clinically as well, as we have already noted. As we found in the case of the kidneys, in the majority of instances in which recovery takes place, the liver shows no signs of permanent damage.

In eclampsia, most writers are agreed that the changes found in the liver are more characteristic of acute toxæmia than of acute yellow atrophy; nevertheless, we must admit that the lesions found in eclampsia, acute yellow atrophy, phosphorus-poisoning, etc., are very similar - no marked differentiating characters being yet pointed out. They all show lesions, in fact, of an acute toxæmia; and the mere fact of these changes in the liver occurring in eclampsia is very strong evidence in favour of the theory of toxæmia which I shall shortly describe presently.

I may add here that Glockner has lately reported a case of carbon bisulphide-poisoning, where the lesions found post-mortem were almost identical with those found in eclampsia; and that Meyer-Wirz has seen a case of carbolic acid-poisoning, and another or corrosive sublimate poisoning after delivery, in which the appearances were very like those of eclampsia.

An indirect method of proving the toxæmic origin of eclampsia advocated was to show, by the injection of inert colouring matter into the tissues, the time which would elapse before the pigment would appear in the urine, and also the rate and regularity with which it was eliminated by the latter. The object of this was to prove that elimination by the kidneys was diminished during pregnancy, and especially during albuminuria and eclampsia. If this could be demonstrated, it would be evident that a toxæmic condition of the blood must result.

Oliver noted that in pregnancy, labour, and sixteen cases of eclampsia the elimination was deficient; but during the puerperium it was increased. In seventeen cases of eclampsia it was not diminished. In the albuminuria of pregnancy the results were found to be very contradictory.

It seems to me at this juncture apposite to discuss briefly:

Stumpf's Theory of Eclampsia. - According to him, the convulsions are the result of some poisonous nitrogenous body produced, by abnormal decomposition, within the organism of the mother or foetus. The fact of this theory being based upon the post-mortem changes found in the liver lends it additional importance. It is a theory, I may add, which has recently received considerable support - more especially from Fehling. Stumpf, in the course of two post-mortem examinations, was struck by the resemblance the lesions in the liver bore to those produced in that organ by acute yellow atrophy. We have already noticed that a considerable number of observers have been struck by this. He also believes that, under abnormal processes of decomposition, a substance, free from nitrogen, toxic in its action, and perhaps resembling acetone or actually that body, both reacting to the same

tests, may be formed. That this body produces, by its excretion, an irritation of the kidneys; it has a tendency in this way to give rise to nephritis; it leads to destruction of the colouring-matter of the blood; greatly alters the activity of the hepatic cells; causes sugar to appear in the urine; and produces destruction of the parenchyma of the liver, leading to acute yellow atrophy of the organ, with the formation of leucin and tyrosin; and it also induces coma and convulsions by irritation of the brain. We have already noticed that when, in eclampsia, the changes in the liver are most marked, there seems to be no distinct demarcation of these, in this disease, from acute yellow atrophy. Stumpf detected acetone, or aceto-acetic acid, - which he regarded as the actual poison, - in the expired air and urine of eclamptics; and Schaefer has pointed out that, if acetone is given in sufficiently large doses to animals, convulsions and coma are produced. Stumpf also regarded the foetus as the source of the poison, as well as the mother. Many observers have noted the fact that, when the foetus dies, the convulsions may disappear; but we also see eclampsia in the puerperium when this influence has disappeared; while, again, cases are frequently noticed of patients being cured of eclampsia, going to full term, and giving birth to a living child. Removal of the child, in a certain number of cases, may cause a cessation of the fits, but this is by no means the general rule. Stumpf also pointed to the fact that the foetus is sometimes in a state of rigor mortis in eclampsia; but it has been shown that this happens very exceptionally, and may also be found in other cases as well as eclampsia. Dührssen holds that acetone, far from being the poison which produces eclampsia, is the result of it owing to the dissolution of the blood. Acetone, in small quantity, may be found in normal urine; but it is in diabetes, some cases of which terminate by convulsions and coma, that it is found in greatest amount. Some observers, more particularly Costa, have found acetone in greater amount in the pregnant than in the non-pregnant condition, and affirm that it is especially seen near full term. They have observed that it is increased during labour, especially if it be prolonged; while it is decreased during the puerperium, still remaining greater than seen in gestation for six days. Costa and others have proved that acetonuria cannot be regarded as an indication of the death of the foetus, as some have held. Audibert and Barraja declared that acetonuria is due to several causes - e. g., labour, eclampsia, and the death of the foetus.

One of the principal supporters of Stumpf's theory is Fehling, who affirms that the metabolism of the foetus, with the elimination of its products into the maternal circulation, can be regarded as of prime importance in the causation of eclampsia. He considers the nephritis as the first sign of intoxication, and not the cause of eclampsia, which, he says, is the next sign of the intoxication in question.

The work of Baron and Castaigne also throws light on the relation of the foetus and mother to each other; for they found that certain substances, when directly injected into the amnion, rapidly passed into the maternal organism, - but that the passage was more rapid from the foetus to the mother. But, when the foetus is dead, this elimination of substance into the maternal circulation does not take place. This has been taken as an argument in favour of the foetal causation of eclampsia.

CHANGES IN THE SPLEEN.

The changes that have been found in the spleen are much the same as those we have described under the changes in the liver. There is an enlargement of the organ, with petechiae on its surface; and haemorrhages under the capsule are often met with. Minute haemorrhages are also found throughout its substance, with larger or smaller areas of necrosis; and, in severe cases, where the poison at work has been very virulent, severe infarction, with more or less extensive haemorrhages, may be seen in the substance of the gland.

CHANGES IN THE LUNGS.

The lungs are frequently found congested or oedematous. Lobar, lobular, or broncho-pneumonia are conditions frequently seen in patients dying from eclampsia. Infarctions of the lungs are sometimes found; also subpleural and submucous petechiae. Blood is often found in the bronchial tubes; and thrombi and fat emboli have also been described in the arterioles; and certain little areas of thrombosis and necrosis (similar to those seen in the liver, and in which hepatic and placental cells have been found by different observers) have also been seen.

THE BRAIN IN ECLAMPSIA.

The changes found in the brain in fatal cases of eclampsia, by different observers, are very variable and contradictory in character. In some cases, no changes at all could be detected. Oldhausen, who very carefully examined 30 fatal cases, found oedema present in 16, apoplexy in 5, hyperaemia in 5, and large clots of blood in the pia mater in 2. These haemorrhages, which are frequently described by authors, are probably the result of convulsions. Goldberg reports 4 with haemorrhages, 9 with oedema, 4 with anaemia, and 2 with hyperaemia.

In looking through the reports of other observers, we find that the post-mortem findings in the brain are by no means constant - hyperaemia, anaemia, and oedema being found in varying proportion.

Certain observers have described punctiform haemorrhages into the cortex and basal ganglia, with an occasional large haemorrhage; while others, again, have seen considerable areas of softening.

THE THYROID IN ECLAMPSIA.

Since Lange called attention to the condition of the thyroid in pregnancy and eclampsia, considerable attention has been directed to it, - especially by Nicholson, who had adduced evidence, mostly clinical in character, to prove that eclampsia is brought about by a condition of thyroid and parathyroid inadequacy.

Lange made a very careful examination of more than 100 women who were in the last months of their pregnancy; he found that there was thyroid enlargement in no less than 81.2%. (This enlargement, I may add, generally begins about the sixth month - just about the time that the toxæmia of pregnancy is apt to appear. Furthermore, very few authors have given an account of the pathology of this organ based on actual post-mortem findings.) If the women were supplied with iodothyron, which is the active principle of the thyroid gland, he found that the organ grew smaller, - to enlarge, however, when the drug was discontinued. It is apparent, then, that more iodothyron is required in the pregnant condition: consequently we have hypertrophy of the gland. In 18.8% of his cases, there was no hypertrophy of the thyroid; and, out of these 22 cases, 16 developed albuminuria with tube-casts, and 6 had eclampsia.

Lange's results have been largely substantiated by recent researches. From his investigations, Nicholson has built up a theory which has already received a good deal of clinical support. He points out that the thyroid gland is of very great importance to the organism in perfecting metabolism; and in pregnancy, of course, more cells would be produced by the energy of this organ. If the latter failed to secrete sufficient iodothyron, the result would be that the metabolism of nitrogenous bodies would not be completed up to the stage of urea-formation, with the result that toxæmia would develop. Auto-intoxication is the result; as we saw before, this is the outcome of imperfect function of the various metabolic organs of the body, - the so-called "defence organs"; and Nicholson believes that iodothyron is necessary for the proper working of these organs. He pointed out, moreover, that the symptoms in advanced myxoedema and eclampsia corresponded in many important respects. There is a decrease in urea-secretion in both; the solid oedema of myxoedema may be present in certain cases of

eclampsia; albumin is very often present in both; while, again, cases of myxoedema may end in convulsions. In cases of removal of the whole thyroid system in animals, the symptoms produced are very like those of eclampsia. The latter condition, indeed, may be regarded as a temporary athyroidia. The result in both cases is that proteid derivatives are not built up into the final product urea, with the result that we have toxæmia produced. Under the administration of iodothyryn metabolism is stimulated, and the amount of urea in the urine is increased. In cases where the thyroid is small, or absent or removed by operation, there is faulty metabolism and impaired secretion.

Nicholson, dealing with the condition of the renal vessels in eclampsia, says: "There was a marked contraction of the smaller blood-vessels, and as this became more pronounced the secretion of urine ceased. In these cases, at an early stage, the quantity of urine might be largely increased; but later on, when the constriction affected the renal arteries, and thus virtually shut off the blood-supply to the glomeruli of the kidney, urine could not be secreted." Whether a deficient thyroid secretion was responsible for this or not he could not say, but he has no doubt whatever about the activity of very large doses of thyroid extract in bringing about a complete vascular dilatation. Thus, the full force of the circulation became again turned on to the glomeruli, and the secretion of urine commenced.

There is a considerable amount of evidence - both clinical and experimental - to support Nicholson's theory. When a woman with the premonitory signs and symptoms of eclampsia (or where there are albuminuric manifestations) is placed absolutely at rest, and her nitrogenous diet restricted (only milk being allowed), she very often improves greatly; and the reason for this is that the diet in question does not entail so much work for the thyroids, with the result that we have the balance of nitrogenous metabolism re-established. Many observers have found that an attack of eclampsia has very often occurred after the ingestion of a meal rich in nitrogenous substances; while, again, it frequently supervenes on hard work or unusual exercise.

Herrgott has reported a most unusual case of pregnancy, occurring in a cretin fifteen years old. In this disease, I may add, there is usually non-development of the pelvic organs, so that pregnancy is rare. Pregnancy was well advanced before she was sent into hospital for induction of labour, on account of the contracted pelvis. There was no history of oedema, nor of headache, nor of ocular disturbances; and there was never more than a trace of albumin in the urine, even after the fits had developed. Before delivery, she had twelve typical epileptic fits, in the intervals of which coma was not complete. The convulsions, eight in number, continued for twenty-four hours after delivery, gradually becoming less severe. She made a good recovery. Herrgott discusses the experimental work of Schiff Hoffmeister, and other authors, all of whom declare that the thyroid gland secretes a substance which modifies toxic substances in blood in some way - making them less harmful, or even destroying them. Herrgott does not agree with Nicholson that thyroid inadequacy is the cause of eclampsia. He regards the thyroid, then, as belonging to the "defence organs"; and also affirms that it, along with the liver and kidneys, are the most important members of this group. We have already seen that two distinct types of eclampsia may be differentiated - one in which the liver is at fault, the other (more numerous) where the kidneys are at fault. To these Herrgott would add a third - viz., where the thyroid gland is at fault, and where there is absence, non-development, or greatly altered condition of the parathyroids.

Verstracter and Vanderlinden reported a case where the thyroid gland was removed in a cat. Three years afterwards, the animal became pregnant. With the onset of labour, which was long and tedious, convulsions set in and the cat became comatose. Extract of sheep's thyroid was injected into the muscles, with the result that the coma and convulsions disappeared, a dead kitten was born, and the cat herself made a good recovery.

Richon and Jeandelize reported, last year, a case where the whole thyroid and two of the parathyroids were removed from a pregnant rabbit. Labour came on prematurely, and was remarkably slow; it lasted three days, and four dead fetuses were born. The rabbit was in a semi-comatose condition during labour, and it actually died in a state of coma. The urine contained traces of albumen, but the temperature was subnormal. They were struck by the likeness of the signs

and symptoms in this case to those of uraemia without albuminuria and eclamptic coma.

Iodothyron is a good diuretic and diaphoretic. Again, urea - the final product of nitrogenous metabolism, for which completion an adequate supply of iodothyron is necessary - is another excellent diuretic. Iodothyron is a powerful vaso-dilator; and, by virtue of this, it tends to promote and maintain renal activity. Again, the internal secretion of the suprarenals contracts the vessels; this contraction is also seen in cases of hypothyroidism. If the suprarenal secretion were unopposed, we would have contraction of renal vessels, which might be a factor in leading to the arrest of renal secretion. Thyroid extract may have some specific action in rendering certain toxic substances harmless. It is stated that the parathyroids normally render entero-toxins harmless.

It is evident that the real significance of the pre-eclamptic state points to a break-down of some part of the defensive mechanism. Furthermore, this break-down is the result of some inadequacy of the thyroid and parathyroid glands, whereby the process of nitrogenous metabolism, instead of resulting in the formation of urea, ceases with the production of intermediate substances, which, when absorbed, excite toxæmic symptoms. In this way the degree of toxæmia of pregnancy comes to be dependent - directly or indirectly - upon the quantity and activity of the thyroid secretion; the thyroid glands may therefore be given a primary rôle in the causation of eclampsia.

THE ALIMENTARY CANAL IN ECLAMPSIA.

The changes found in the alimentary canal of patients who have died of eclampsia are by no means constant. Lesions, from congestion of the mucous membrane of the stomach and intestines (small) to erosions and ulcerations, have been described. These are often particularly marked in the duodenum, where they may be caused by the liver excreting such poisonous substances as inflame and erode the mucous membrane. These ulcerative processes may extend through a blood-vessel, and so give rise to very profuse or even fatal hæmorrhage, as occurred in a very interesting case observed by my friend Dr. Logan, of Wishaw.

It appears that this patient had been in ill-health for some time before her confinement, complaining of obstinate constipation, and developed a well-marked and extensive ~~perianth~~ ^{perianth} eruption, fourteen days before labour, which was ushered in by eclamptic convulsions. These she recovered from; but, on the ~~fourth~~ ^{fourth} day, she began to vomit - at first coffee-grounds, later on blood unacted on by the gastric juice. She finally succumbed to this hæmorrhage. There were no signs of peritonitis, nor of pulmonary complication, while her temperature was normal.

It is not uncommon to find small hæmorrhages into the intestine, as well as those fatty and necrotic changes in the epithelium that I have already described in connection with the liver and kidneys.

Constipation very frequently accompanies pregnancy: in fact, the majority of women at all times suffer from this. It is often exaggerated by indiscretions in diet and insufficient exercise. In pregnancy it is partly accounted for by the pressure exerted by the gravid uterus, and partly by the deficient innervation of the muscular coat of the intestines leading to torpidity. Again, owing to distension, the abdominal wall cannot be brought into play during the act of defæcation.

Does the Condition of Constipation, and its Consequent Copraemia, Predispose the Patient to Eclampsia?

Although some observers believe that eclampsia is the result of leucomaine and ptomaine poisoning, the evidence is altogether against this.

The PTOMAINES are chemical compounds, basic in character, which are the result of the action of bacteria on organic matter; they are also termed putrefactive or vegetable alkalis, in order to

distinguish them from leucomaines, or animal alkaloids; which latter are the result of tissue metabolism going on within the body - the intermediate products of metabolism.

In the intestine there are numerous bacteria and putrefactive and fermentative processes continually going on, these being held in check to a certain extent by the liver secretion, bile, and partly by the fact that the bodies manufactured by certain bacteria have often a destructive action on other bacteria. Whenever the hepatic function is interfered with, so that the excretion of bile (which acts as an intestinal antiseptic, curbing the action of bacteria) is lessened, then fermentation and putrefaction go on unchecked, with the result that we have absorption of toxins. In this way the intestines may add to the already toxic condition; while, again, when constipated, they would allow the toxins to be absorbed into the blood which ought to have been expelled by them from the body; and in this way they play a prominent and important part in predisposing the patient to an attack of eclampsia.

The nitrogenous products of putrefaction, which ought to have been rendered inert by the liver, have even been regarded as the primary causes of eclampsia, but the symptoms of ptomaine-poisoning do not much resemble those of that condition. Those who support this theory point to convulsions in babies, - which are often caused by intestinal disturbances, - and to the fact that it also occasions fits in epileptics. Again, in albuminuria of pregnancy, the albumin may be diminished by smart purging; while Savory has reported a case of eclampsia where the convulsions disappeared on the removal of foul-smelling faeces from the bowel, and the thorough irrigation of the part.

As regards the LEUCOMAINES, several writers hold that they constitute the poison in eclampsia. These, as we have already seen, are the result of tissue metabolism going on in the body, and also the intermediate products of metabolism; and those who regard eclampsia as the result of poisoning by these say that the liver, owing to functional derangements or disease, is unable to synthesise the lower nitrogenous products of metabolism to urea; which then collect in the blood, owing to the kidneys breaking down and failing to excrete them.

We have already considered a few of these intermediate products products of urea under the chemistry of the urine. Carbonic acid is the only one known to have very poisonous properties; and Ludwig and Savor held that this was the poison in eclampsia.

Massin, of St. Petersburg, was one of the first to assign to the liver, on account of its being a great oxidising organ, a special rôle in the production of eclampsia. He was one of the assistants of Pawlow, who carried out the classical experimental work on the so-called "eck" fistulae of the portal vein and of the inferior vena cava. This research was also used by Ludwig and Savor, and other authors, in support of their theories that eclampsia was the result of the accumulation of unsynthesised antecedents of urea. Recent investigations have shown that during pregnancy the process of oxidation is carried on very imperfectly - with the result that many intermediate bodies, containing nitrogen, are produced.

The above-mentioned observers, as well as many others, are agreed that eclampsia is produced by some disturbance in the system during pregnancy, which results in a poisoning from deficient oxidation; but they disagree as to the actual substance.

As regards carbamic acid, it is very difficult to estimate the quantity of this substance, as it is so variable and easily decomposed. It does produce, when injected into the blood, poisoning with symptoms not unlike those of eclampsia. The experiments of Pawlow on dogs, who had fistulae of the portal vein and inferior vena cava, showed that the amount of carbamic acid in the blood of these animals was considerably in excess of the normal. At certain times, however, the dogs seemed to be in a normal condition; and their condition could ~~even~~ be altered by diet, etc. The same conditions, these observers hold, may take place in pregnancy. All may go on well until something ensues to bring about an outbreak: such conditions we have already discussed under the predisposing causes of eclampsia.

We have already seen that in eclampsia the oxidation of substances containing nitrogen, as measured by the total amount of

nitrogen in the urine, is considerably lower than the proportion of nitrogen in the urea. Many declare that metabolism during pregnancy takes a different course to that in the normal condition.

Massin found that the quantity of leucomaines contained in the urine, just before the attack, is ~~increased~~ very considerably above the normal. This increase disappears very rapidly during convalescence. He holds, therefore, that eclampsia is the result of a poisoning by the leucomaines. During pregnancy the leucomaines are present in appreciably greater numbers than normal. Should the liver by any reason be unable to perform its work and of turning them into inert bodies, we would have poisoning of the system by them, and also the development of eclampsia therefrom. He was, however, unable to separate the particular leucomaine which was at fault.

TOXAEMIC THEORY.

The toxæmic theory of eclampsia is the one which has received the greatest amount of support, its existence being supported by numerous clinical and pathological and pathological facts. Many of these we have already discussed when bringing forward the toxæmic theory of pregnancy; and, in describing the pathological changes seen in the different organs, we have noted their similarity to those produced by other poisons.

Most authors are agreed that in eclampsia we have a condition of toxic poisoning; but, when we come to consider their views regarding the poison (or poisons) which produce this disease, and the source of same, we find very great divergence of opinion. We have already seen that eclampsia in itself is not a specific disease, but the expression of many pathological changes in the organs; also that we may have many different types of the affection. Some observers have gone the length of saying that each type is the result of a separate poison, while others declare that the disease is ~~caused by~~ the combined action of complex poisons. But, even if there be only one poison, we might expect to meet with very different signs and symptoms in different patients, in a way depending upon the idiosyncrasy of the patient; or, again, one organ may be more vulnerable than another to the poison, and consequently break down earlier, - its insufficiency being expressed by certain definite signs or symptoms; furthermore, the toxin may have a special selective action for a particular organ.

We know that even in the healthy body the toxic substances are very numerous, and also that their origin is very various. Many are taken into the body with the food and drink; whilst others are ~~ferred~~ in the alimentary canal; and, instead of being injurious in some cases to the organism, some of these, we are told by physiologists, play by no means an unimportant rôle in the complicated processes of digestion and assimilation. But, should there be any interference with the normal processes, - e.g., interference with the functions of the liver, - we find an enormous increase in the poisons in the bowel, the bulk of these resulting from the action of micro-organisms, although some have other sources.

Bouchard's Theory. - The modern conception of the etiology of eclampsia and the occurrence of a toxæmic condition is founded more especially on the work of Bouchard and his disciples. It was in 1887 that his great classic ("Leçons sur les Auto-intoxications") first saw the light. He believed that the health of an individual was closely related to the production and elimination of toxic material: if there was an increase in their production or defective elimination, according to him, we would have a condition of auto-intoxication. The cells of the tissues are in a state of ceaseless activity, each cell continuously absorbing new material and throwing off waste products - all this involving chemical changes of a very intricate character. The result is that a constant elimination of toxic substances would pass from the tissues into the fluids of the body, so that, as Bouchard says, "man is constantly menaced by poisoning; labouring each minute for his own destruction; and making continual attempts at suicide by auto-intoxication."

The organism has two principal lines of defence, however, to ~~prevent~~ its destruction. To the one line of defence belong these ~~organs~~ which have to do with the removal of toxic substances from

the system - the skin, the lungs, but more especially the intestines and the kidneys. Bouchard regarded the excretions as an index of the blood and tissues of the body generally. If these toxic bodies were being properly excreted, we would find the toxicity of the excretions high, and the blood again low; while the opposite condition would lead to the production of toxæmia. Failure in the eliminating power of an organ would be shown by decreased toxicity of excretion, while the blood would be proportionately increased in toxicity. In pregnancy one would have a great increase in the toxins produced, and consequently both blood and excrementitious matter would be more toxic; if the excretory organs responded to the extra demand made upon them, there would be no toxæmia. Our author likewise affirms that the pregnant woman is in a state of potential auto-intoxication. He contends that the kidneys are able, when in a healthy condition, to eliminate a much greater amount than they are ordinarily called upon to do. As a proof of this statement, we may point to cases where a kidney has been removed by operation; and, the patient becoming pregnant, neither albuminuria nor eclampsia results.

Our second line of defence, whose chief member is the liver, has to attack these toxic substances; and, by transforming them into other bodies, render them harmless. Some substances (e.g., albumoses), were they allowed to circulate in the blood, would soon give rise to poisoning; these are attacked by the liver, and after being transformed into harmless, but at the same time valuable bodies, again pass into the blood stream to take part in building up the tissues. The liver at the same time is an excretory organ, for it excretes certain poisons (e.g., bile), which, again, have an antagonistic action upon toxin-producing bacteria. We now know that there are other organs which are also concerned in the protection of the organism, - the principal amongst these being the spleen, thyroid gland, suprarenal capsules, and other glands. A great deal of work, however, still requires to be done regarding the function of these glands.

Bouchard declared that menstrual blood was hypertoxic; and also that in pregnancy the blood was more toxic, but the urine less so; while after delivery the urine becomes exceedingly toxic, and the blood is less toxic. He held that eclampsia and many other grave conditions were intoxications very similar to uræmia (and by uræmia he used the term in its broadest sense, not the uræmia of chronic nephritis only), and that this term covered a complexity of phenomena. He demonstrated, by means of experiments upon animals, that the normal urine contains several toxic substances - two of which were able to cause convulsions, and one coma. Of the first two, one was a mineral substance, - potash, - while the other, which was unknown, was organic and not unlike an alkaloid, though insoluble in alcohol. The narcotic poison was an organic alkaloid. Our author does not believe that any one of these substances is alone able to produce uræmic poisoning, etc., but that rather all the poisons in unequal proportions (normally introduced into the organism or physiologically produced therein) contribute - i.e., when the quantity of poisons formed or introduced in the twenty-four hours can no longer be eliminated in the same way by the kidneys which have become scarcely sufficiently permeable.

Maurice Riviére was the first to study eclampsia in the light of Bouchard's investigations, his treatise (*Pathogénie et Traitement de L'Auto-intoxication éclamptique*) appearing the year after Bouchard's well-known publication. He declared that eclampsia was caused by an auto-intoxication, the result of the accumulation in the blood, - from faulty elimination by the emunctories (especially the kidneys), or insufficiency of the liver and the other protective organs, - of complex substances. Anvard's work followed on the same lines as Riviére's.

It was not long before experimental researches were commenced, on the lines suggested by Riviére, to prove experimentally that eclampsia was the result of a toxic condition, and also to discover the exact nature of the poison in question. A theory such as that of Bouchard would be, one would think, very easily proved by experimental evidence; but the methods adopted by Bouchard and his followers have been largely discredited by the more careful and scientific experiments of recent observers.

Bouchard and his pupils tried to find out first the degree of toxicity of the secretions in the normal condition; and this, once

found, would be of infinite help in enabling one to come to a conclusion regarding abnormal conditions such as are found in pregnancy, albuminuria, and eclampsia. The excretion which Bouchard used was the urine; and he injected this by a syringe into the circulation, or allowed it to run into the auricular vein of a rabbit by hydrostatic pressure. The amount required to cause the death of the rabbit was called the urotoxic dose, and was worked out in c.c. per kilogram. Bouchard's work has been considerably discredited, and his deductions set at naught, by the fact that he took no trouble to collect the urine aseptically, paid no attention to its temperature, the length of time it was kept, nor took any precautions in its preparation. The result is that he arrived at quite different conclusions than those of investigators who were more thorough in their methods. In fact, the only precaution he took was to filter the urine and render it neutral in reaction by the addition of bicarbonate of soda.

Tarnier was the first investigator to apply Bouchard's theories and methods to a study of the urine in pregnancy. He found the toxicity of the urine to be diminished in pregnancy, and that in eclampsia it was very low, owing, he thought, to the hypertoxic state of the blood.

Chamberlent and other students of Tarnier continued the work of the latter; and they came to the conclusion that there was an accumulation of toxic material in the blood in eclampsia, which should have been eliminated by the kidneys, and that consequently the blood-serum became hypertoxic - with the result that hepatic and renal insufficiency developed, still more increasing the toxic condition of the serum.

Blanc, in 1893, made a minute investigation of the urine in pregnant and puerperal women in the first week after delivery. The urine he employed was warm, filtered, and free from albumin, being also rendered neutral by the addition of a standardised solution of soda. He came to the conclusion that the toxicity of the urine was not diminished in pregnancy, but increased after the conclusion of labour. I may add, however, that he did not use sterile urine for his experiment.

Labadie-Lagrave carried out an experiment on the toxicity of the urine in 23 women at different periods of pregnancy and after labour. He stated that the toxicity of the urine diminished during the first three months of pregnancy, remaining stationary afterwards, while the toxicity slowly increased after the termination of labour till it reached a maximum about the second month.

Then came the work of Ludwig and Savor. They experimented upon the urines of pregnant and eclamptic women, which they collected aseptically, but did not sterilise, though they placed it on ice until about to be used. They carried out their investigations on the urine during the convulsions, and again within forty-eight hours after their cessation; and found that in eclampsia there were great variations; but, as a rule, the average urotoxic dose was lessened during the convulsive stage, but raised afterwards. They believed that the poison at work in eclampsia was carbonic acid, the result of imperfect metabolism going on in the liver. Other observers, however, found such a variety of results in testing the toxicity of the urine that no deductions could be made at all.

Voldhard, in 1897, contributed some very painstaking work on this subject, although he, like most of the preceding writers, did not exercise great care to prevent contamination. He proved that a very important factor in the results produced is the technique adopted in carrying out the experiments. He injected the urine, under low and even pressure and very slowly, into the jugular vein - taking great care to guard against the entrance of air, and to see that the temperature of the fluid injected corresponded to the body heat of the animal experimented upon. He noted that the urine was much less toxic on boiling, and affirmed that this was probably the result of sterilisation. He found considerable difference in the toxicity of the urine of pregnant women, even the rate of injection into the animal's blood having considerable influence on the results obtained. In his experiments upon the urine in eclampsia, the results obtained were so contradictory that no conclusions could be attempted.

The most efficient work on this subject was carried out by

Schumacher and Stewart. The former, adopting the precautions insisted upon by Volkmann, showed, to begin with, that - by injecting saline solution alone into the circulation of animals - when the strength of the solution was sufficiently raised, it would act as a poison. He believes that the so-called toxicity of the urine in healthy persons, apart from pregnancy, depends upon its concentration and specific gravity. In one case, where normal urine was injected, the attacks of epileptiform convulsions developed. This is interesting, because it was formerly thought that attacks of this nature could only be produced by pathological urine. Tarnier and Chamberlent, as we have already seen, held that the urine excreted by women during pregnancy was less toxic than that passed after labour - owing to accumulation and retention of toxic substances in the system, which were afterwards eliminated during the puerperium. But Schumacher demonstrated that the urine in pregnancy and labour possessed an equal density and an identical toxicity. Again, in the albuminuria of pregnancy the amount of albumin in the urine has no influence on the toxicity, which differs very little from that of normal pregnancy. As regards the toxicity of the urine in eclampsia and albuminuria of pregnancy, he found that it depended altogether upon its density, that it acted, in fact, as a very concentrated saline solution would do, and that, when the density was reduced by the addition of distilled water, its toxicity was correspondingly reduced. He did not find any difference in the urine from normal pregnancy, puerperal albuminuria, or eclampsia. As regards the diminution of toxicity of the urine in eclampsia during the convulsive stage and the increase of same after the cessation of the fits, which Ludwig and Savor held, Schumacher affirmed that their dependence was upon the conditions we have already mentioned. Although these are the conclusions arrived at by Schumacher, he still accepts Bouchard's theory that eclampsia is the result of a toxic condition produced by certain products of metabolism; but he considers that the method of investigating, by experiments on the toxicity of the urine and blood in eclampsia, will not help in the elucidation of the poison in this disease.

In his experiments, Stewart took every precaution against the contamination of the urine, collecting it under strict aseptic conditions and afterwards placing it in a sterilised flask. This he would keep for twelve, twenty-four, or forty-eight hours before he injected it intraperitoneally into animals. In fresh unboiled urine, newly used, death occurred in 10% of the cases; but fresh boiled urine he found innocuous. Stale urine caused 100% deaths. He also found that stale urine became more toxic the longer it was kept; and that fresh urine, when care was taken to collect it under strict aseptic conditions, was practically harmless. Again, after other experiments, he called in the aid of the microscope - testing each specimen of urine for micro-organisms. Urine which proved toxic also gave proof of the presence of micro-organisms, and showed that death in these cases was due to septicaemia, and that boiled urine, again, whether stale or fresh, was non-toxic.

Some observers declare that Stewart's method of procedure (boiling, etc.) would destroy the toxic bodies which the urine may have contained; but, again, Stewart proved that fresh unboiled urine, where care had not been exercised in collecting it, would cause death in 100%, pointing therefore to contamination as a source of toxicity. Schumacher's and Stewart's conclusions would seem to prove, then, that the toxicity of the urine in pregnancy depends upon decomposition; or, again, to defective methods in collecting it, whereby it became contaminated; while, moreover, the toxicity of the urine in puerperal albuminuria and eclampsia depends upon its concentration and specific gravity.

The first method, therefore, of proving toxæmia from experiments carried out on the urine has so far ended in failure. Let us now consider the investigations carried out on the blood.

The Toxicity of the Blood in Eclampsia.

While the work we have just described was being carried out, attempts were at the same time being made to demonstrate the toxicity of the blood in pregnancy and eclampsia. Many observers have been in the field, and much experimental work has been carried out, the same reflecting the utmost credit on their painstaking care and ingenuity; but their deductions as to the toxicity of the

blood (by experimental evidence) has not so far been accepted as conclusive. It is now known that experimental proof of the toxicity of the blood or serum, by injections into the circulation, must be altogether deceptive, as it is known that the serum of one animal is poisonous to another if injected into the blood-stream. The animals were often destroyed by the production of blood-clot in the heart and blood-vessels. When it was injected into the peritoneum, or into the tissues themselves, greater quantities were required to cause death. It has been found, too, that removal of the coagulable elements of the blood leads to a greater reduction in the toxicity.

Tarnier and Chambrelent injected the blood-serum of eclamptic patients into the auricular vein of rabbits. They collected the blood obtained by venesection, which was allowed to stand until the serum separated, when they drained it off ready for use. They did not in any way adopt precautionary methods against septic contamination of the serum; yet, everyone is aware that one of the best culture media for bacilli is blood-serum. They proved, to their own satisfaction, that the serum of eclamptic patients was more toxic than in normal pregnancy.

Chambrelent, in 1892, as the result of further investigation, published the result of a series of experiments carried out on the blood of eclamptic women. The following are the conclusions he arrived at: (1) In pregnancy the blood is more toxic than normal, the urine being less so. (2) In eclampsia, on account of the faulty elimination of physiological poisons, the urine is less poisonous than usual, and also less poisonous than the urine in normal pregnancy. (3) Lastly, that in eclampsia the serum was considerably more poisonous than normal, its toxicity being in inverse proportion to that of the urine.

It has never yet been proved, however, that the blood-serum of eclamptics is more toxic than normal. Careful investigation has shown that when the blood-serum of eclamptics is injected into animals, all necessary precautions having been taken, the same results are produced as by normal serum. Both sera cause disintegration of blood-corpuscles, as well as the appearance of haemoglobinuria.

Bouffle de St. Blaise pointed out that the blood of an eclamptic was more toxic than the blood of a normal one. Again, Chambrelent found in one case that the blood of the foetus was more toxic than that of the mother; and Tarnier also has found this to be the case on more than one occasion. Later, Volhard and Schumacher found that the serum of the foetus is not more poisonous than the serum of the mother, whether healthy or albuminuric.

Schumacher declared that the results obtained by Tarnier and the French school were due to imperfect methods of observation, and he also held that the details of the experimentation with serum are of even greater importance than with urine. He showed that there was no difference between the serum in normal pregnancy, puerperal albuminuria, and eclampsia, and that the gravity of the eclamptic symptoms had no effect on the degree of the toxicity of the urine. He also demonstrated that it was impossible to draw inferences as to the condition of the serum from the condition of the urine.

We have already seen that the urine acts practically in the same way as a saline solution, that is to say, the greater the density the greater the toxic effect; but this is not the case with blood-serum, for, if distilled water is added, it will be found that its toxicity is only reduced to the degree of dilution. In other words, if the serum is diluted with double its own bulk of distilled water, its toxicity would be the same as the same quantity of undiluted serum; whereas, if a specimen of urine were diluted in a similar way, there would be no toxic action at all.

Bell, in his case of eclampsia, found the blood toxic, and an examination of the blood in two cases of uraemia at the same time gave negative results.

In 1894, Mauret and Bosc endeavoured to estimate the toxicity of the normal serum. They found that the method of investigation, by injecting blood-serum into the veins of animals, was bad (for it caused clots to form in the heart and pulmonary artery, which would lead to death by asphyxiation), and that, when the coagulable elements were removed, toxicity was greatly reduced. In other experiments, they separated from healthy dog's blood, by a special process, an albuminoid body, which, when injected into the circulation, caused death

without coagulation of the blood. They called this non-coagulative poison a toxin.

Again, Doleris and Buttle demonstrated that in eclampsia a crystalline inorganic body could be separated from the blood, which, when injected into the tissues, caused death with convulsions in animals. They came to the conclusion, therefore, that in eclampsia there is present in the blood a poison which causes convulsions. The disease they regarded as of the nature of an auto-intoxication, and not unlike that produced by bodies analogous to ptomaines.

With regard, then, to the experimental work on serum.- although no direct proof has been forthcoming from the enormous amount of work performed, still, the results are not so negative as those obtained from the experimental work on the toxicity of the urine. We have seen, for example, that a substance has been extracted from a healthy animal's blood which is highly toxic; it remains to be proved whether such a toxin can be extracted from human blood.

Again, a crystalline inorganic body, - also highly toxic, - has been separated from the blood in eclampsia; but here, also, we have no confirmation of this by any recent observers. Our knowledge of the properties of normal blood-serum, notwithstanding the great amount of excellent work recently done regarding it, is very far from complete; but when it is, then perhaps thoroughly scientific examination and laborious and careful experimentation with the blood (in puerperal albuminuria, the pre-eclamptic stage, and during the eclamptic seizure) may discover the real cause of this disease.

Eden, in his valuable and comprehensive essay ("An Inquiry into the Toxaemic Theory"), concludes by saying: "Methods much more precise in their aims and careful in their execution are called for if this difficult subject is to be elucidated by the fallacious method of experiment upon animals."

"The observations of Doleris and Buttle and Majret and Bois are full of promise; but, unless physiological chemistry can separate definite toxic bodies from the blood, it seems impossible to devise experiments which will not be vitiated by serious sources of error."

Finally, Schumacher, although he does not believe that the theory of Bouchard has been invalidated by the fact that negative results have been obtained from experimental investigations of the urine and blood carried out in animals, holds that it is needless to pursue these methods further, as it is not through these methods that the poison in eclampsia will be discovered.

THE MICROBIC THEORY.

Special Reference.- For a summary of the work on the microbic theory of eclampsia, see Schmidt's Jahrbuch, Bd. 2, 37.

Arguments in Favour of the Microbic Theory.

(1) Eclamptic seizures are often coincident with contraction of the uterus, which leads to deposition of poisonous substances from the region of the placenta; and eclampsia often comes on during labour. (2) Pleural births predispose to eclampsia. (3) Eclampsia seldom recurs. (4) Evacuation of the uterus leads to amelioration of symptoms. (5) Tendency to nephritis during pregnancy. (6) The frequency of nervous complications after eclampsia, as so frequently happens after infectious diseases - e. g., diphtheria, etc. (7) In many of the infectious processes, the fever is ushered in by vomiting. (8) The appearance of albumin in the urine.

The confident manner in which Gerdes wrote of his bacillus gained him the support of many obstetricians, prominent amongst them being Kaltenbach. He asserted, with absolute assurance, that "the eclamptic bacillus is the sole origin of eclampsia", and that "eclampsia is a highly characteristic disease with strictly anatomical limitations". He found a bacillus in the organs (liver, kidneys, etc.) of eclamptic women, and in several cases in the blood. In one case so many were present in a blood-vessel as to suggest microbic embolism.

Hofmeister, who several times criticised Gerdes' findings, held that the Gerdes bacillus was nothing more than the bacillus proteus

vulgaris, and that, as Gerdes began his investigations from 14-23½ hours after death, this bacillus had had time to penetrate into the various organs. His searching criticism of the microbic origin of this disease has received the support of most of the recent investigators; and Haegler, working along the lines adopted by Gerdes, came to quite opposite conclusions to him, and upheld the work of Hofmeister. Indeed, what happened to the Gerdes bacillus was the fate of the bulk of the work of those who upheld the microbic origin of this disease.

The writings of Favre, regarding the discovery of the fungus-like growths on the white infarct of the placenta, have been entirely set aside, as also the work of Blanc, Combemale, and Buë, and, more recently, Herrgott, Sourel, and Levinowitch. The last-mentioned observer found, from an examination of the fresh blood of 44 eclamptic women, large diplococci distinguishable by their great mobility. In 25 of these cases, he was able to obtain spontaneous culture from these. Gerdes, Favre, and the bulk of those who support the microbic theory, have to assume that there had been endometritis before pregnancy, on which the infection of the decidua is based. But, we know that primiparae are much more frequently attacked by eclampsia than multiparae, and that endometritis is infrequently met with in them.

Döderlein carefully investigated 8 cases of eclampsia, and decided against any possibility of a bacillary origin of eclampsia. He subjected the work of Blanc, Favre, etc., to a most searching and damaging criticism.

The careful work of Bar and Guyeisse, Schmorl, and Lubarsch also showed that the conclusions of these writers could not possibly be sustained, and that no satisfactory proofs and arguments in favour of the microbic influence in eclampsia had been forthcoming.

As one might expect, then, from an age which has discovered in micro-organisms the cause of so many diseases, we have had different observers coming forward with a particular bacterium or bacillus which they regarded as the cause of this affection. We know how easy it is to demonstrate the presence of microbes almost anywhere, but it is a different matter when it comes to the proof of this germ having distinctive characters and being special to eclampsia.

Délore and Rodet were the first in the field to suggest invasion by bacteria as the cause of eclampsia. Many observers, as we have already seen, - chief amongst them being Levinowitch, Combermarle, and Buë, Herrgott, and Blanc, - have since then cultivated bacteria from the blood, urine, and placenta of eclamptics; but the evidence they produce, so contradictory in character, renders their deductions impossible.

Lately, however, Müller has come forward with a theory, which has certainly something to be said in its favour. He believes that eclampsia is a general intoxication due to the action of bacteria within the uterine cavity. As the disease only appears in pregnancy, labour, and the puerperium, the poison must be uterine in origin - the fact of eclampsia occurring during the puerperium excluding, in Müller's opinion, the foetus and placenta as ~~sources~~ of the poison. He supposes that the bacteria gain an entrance into the uterus before pregnancy begins, and lie latent in the endometrium during the early months of pregnancy; while, during the later months, they again assume such activity as to produce poisons of high virulence. The convulsions are caused by the sudden absorption of a large quantity of the poison.

Albert supports the views of Müller. He regards eclampsia in the light of a variety of latent microbic endometritis. The uterine cavity is well drained, except when it is closed during pregnancy, which the result that the poisons escape into the circulation and give rise to the disease. In these cases, according to our author, it is often possible to obtain a history of the existence of septic endometritis before pregnancy. In two cases of eclampsia where the patients died undelivered, the post-mortem examination showed bacterial infection of the decidua. He also showed that any disorder which was brought about by bacterial influences predisposed to toxæmia of pregnancy.

Neither of these observers, however, have discovered any particular organism. As we have already seen, eclampsia is much more frequently seen in primiparae than in multiparae; if the theories of Müller and Albert were true, the opposite would be the case, - as endometritis is comparatively seldom found in primiparae, while

bacterial invasion would be much more easy in multiparae.

Strogannoff declares that eclampsia has many of the characteristics of an infectious disease. It is a widespread affection; it involves many organs; it comes on suddenly with well-marked premonitory symptoms; its development is rapid; fever is present, and is similar to the pyrexia observed in infectious diseases (especially the hyperpyrexia often found immediately preceding death); and one attack usually appears to confer immunity. In certain years, our author affirms that many cases may be encountered, while in others only a very few; and he adds that the affection is common in the overcrowded wards of lying-in hospitals in populous cities. Physicians carry infective poison with them, for often one case will develop in hospital after the physician or nurse has visited a case outside. I have already pointed out and discussed the fact that cases of eclampsia are much more frequently prevalent in some districts than in others, and also during certain periods of the year than in another; but there is nothing to prove that this prevalence is the direct result of infection.

Notwithstanding the support that it has received from the above-mentioned observers, the bacterial theory of eclampsia has not made much progress. In the first place, no distinct bacterium with definite characteristics has yet been isolated; while it is still to be proved that eclampsia is an epidemic affection. Again, the results which so often quickly follow medical treatment show conclusively, in my opinion at least, that we are not dealing with an infectious disorder.

THE ROLE OF THE FOETUS AND PLACENTA IN THE PRODUCTION OF THE POISON OF ECLAMPSIA.

THE PLACENTA IN ECLAMPSIA.

As regards the pathological changes found in the placenta, nothing distinctive has yet been described. Infarctions (either old or recent, and either large or small) are frequently met with; but they are not common to eclampsia, being found in syphilis, albuminuria (where they are frequently of large size), and in other diseases. Again, they may be found in healthy women.

Klebs, Lubarsch, and Schmorl have, in their pathological investigations, drawn attention to a condition of coagulation in the vessels in various organs giving rise to areas of necrosis. Klebs believed that this coagulation was the result of coagulation-producing ferments, the same resulting from the destruction of liver cells which had gained an entrance into the blood. Lubarsch also came to the conclusion that, as a result of liver cells gaining access to the blood, thrombosis is brought about by these cells producing a coagulation-ferment. From his investigations, he found that placental cells also passed into the circulation, and that they also might give rise to thrombosis as well as the liver cells; although he regarded the latter as the more important, and affirmed that liver cell embolism is a very important factor in the production of eclampsia.

Several other observers besides Lubarsch, principally Schmorl and Winkler, have described giant cells in the pulmonary capillaries and other parts of the circulation. They are distinguished by their dark protoplasm, in the centre of which there is a heap of rounded nuclei. These observers stated that these cells are detached from the placenta in some manner, and so get into the circulation where they give rise to embolism and necrosis, as Schmorl, for example, found these cells free among the placental villi and in the uterine veins.

Poten and Pels Leusden have even described chorionic villi, which they found in the maternal circulation. The large cells we have just described are now regarded as coming from the foetal chorionic epiblast (syncytium) and Langhan's layer, both of which are now regarded as being of foetal origin. The former, until recently, was usually regarded as being of maternal origin. It is now known that these pass into the circulation, even in normal pregnancy. The fact that they can do so is easily demonstrated by what occurs in deciduoma maligna, where we have a metastasis of protoplasmic masses, composed of the syncytial layer of the chorionic epithelium, and

individual cells from Langan's layer. In pregnancy it has never been shown that metastasis of these cells, which does occur, can cause any bad effects; and they have been found frequently in pregnant women who have died from some other disease.

Schmorl, by means of his experiments, found that the lesions in eclampsia were much the same as those in animals in whom coagulation-producing ferments had been introduced into the blood; that the placental cells regularly pass into the maternal circulation; and that experiments show that these can produce coagulation, if present in sufficiently large numbers. As a result of this, he held that eclampsia is an auto-intoxication caused by the coagulation-producing ferments originating in the placenta. In a later communication, however, he modifies considerably these opinions. But he still holds that in eclampsia coagulation of the blood occurs, owing to parenchymatous embolism, and that dying cells have been ~~proved~~ by experiment to coagulate the blood; and adds that we do not know sufficient about the metabolic processes going on in the placenta to prove that the coagulation-ferments, which he holds give rise to the lesions in eclampsia, originate in the placenta.

Dienst, in 160 cases, injected a solution of methylene-blue into the umbilical artery or vein just before separation of the placenta, and directly the child was born. In 20% of these cases, the urine became blue, and after a few hours the colour disappeared. As he injected the solution under so slight pressure that the chorionic vessels could not possibly be ruptured, he held that these experiments proved ~~that~~ the power of spontaneous permeability of fluids from the foetal to the maternal circulation in the placental tissue.

Bulius and Falk found a marked proliferation of the syncytium in the placenta; and Colomi also found that the syncytial buds were much more abundant, and more typically developed, in eclampsia than in ordinary pregnancy; and he regarded eclampsia as caused by an excess of internal secretion from these buds. He also held that the latter may become detached, and be carried as emboli into the maternal organism, - as described by Schmorl, etc., - where they continue their secretive function.

Politi made extracts from the placentas of healthy women, albuminurics, and eclamptics, the bulk of which in the two latter conditions showed old or recent infarcts. The extract he mixed with a 1% solution of sodium bicarbonate, filtered, and sterilised, and then injected into the veins of rabbits. The rabbits in a short time had short spasms, followed by great prostration, and died with great dyspnoea. The toxicity of the placental extract varied with the maternal condition, being least in the placenta of a healthy woman, increased in the placenta with old infarcts, still greater in the placenta with recent infarcts from the albuminuric patient, and most toxic in the placenta of the eclamptic. Although he regards auto-intoxication of the mother as a very important factor in the reproduction of eclampsia, he considers the alteration in the placenta (which allows a placental toxin to gain entrance into the maternal blood) as the immediate cause of ~~eclampsia~~ eclampsia. Many observers have demonstrated that extracts of parenchymatous organs, when injected into the circulation of animals, cause lesions very similar to those found in eclampsia.

The fact that the villi of the chorion are in intimate connection with the maternal blood which bathes their peripheral cells, leads one to believe that toxins, - if they are produced by these cells, as some observers hold, or even cells detached from the periphery of the villi, - may easily gain an entrance to the maternal circulation.

Schultze and Veit declare that when certain cells are introduced into the living blood, a substance is formed there which fixes these cells to the red blood-corpuscles. Part of this substance, according to Ehrlich, enters the serum and forms an antitoxin which tends to destroy the foreign cells. When villi (Langan's layer and syncytium) from the periphery of the chorion gain an entrance to the maternal circulation, the result will be the formation of a substance injurious to these cells, which may be called a cytotoxin or syncytiolysis. By a series of experiments, where they introduced pieces of placenta into the peritoneal cavity of rabbits, they tried to prove this.

Perhaps the most interesting of all researches conducted in

this department of pathology are the experiments recently carried out by Dienst, culminating in a new theory as to the causation of this disease. He tested the blood, - which had been carefully prepared, being defibrinated and rendered sterile, - of 118 mothers and their offspring. In 24 cases, when the maternal blood was added to that of the foetus, it caused agglutination and disintegration of the foetal red blood-corpuscles. In these 24 cases, he tried to find out the permeability of the placenta, as explained in a preceding chapter; the placenta was impermeable in 15, all of which women remained healthy. But, in the remaining 9, - in all of whom the placenta was permeable, - 7 were eclamptic, and 2 had severe albuminuria. His theory, then, is to the effect that we have a reaction between the maternal and foetal blood, such as we have when we mix the blood of one species with another (and we have already seen that one animal's blood has a toxic action when injected into the circulation of another), should the placenta allow in these cases of a free interchange of blood from the foetus to mother, we have either eclampsia or albuminuria.

I have, by a description of a few of the principal researches carried out in this department, given an idea of the trend of a certain kind of experimental work which is being conducted by many workers today; and that is to prove that eclampsia is produced either by the placental cells themselves, or that they produce a certain toxin or ferment which sets up certain changes in the maternal blood.

THE FOETUS IN ECLAMPSIA.

Since Winckel first called attention to the fact that when the foetus dies during pregnancy the threatening signs and symptoms of albuminuria and the pre-eclamptic stage disappear, and that where eclampsia has occurred and resulted in the death of the foetus, - very often the seizures cease, - many others have verified it. And this, and other facts, have led some observers to declare that the foetus plays a very important rôle in the causation of eclampsia. Some of these, principal among whom is Fehling, hold that the main source of the poison in eclampsia originates in the foetus - that here certain products of metabolism are elaborated, to be excreted into the maternal circulation where they give rise to intoxication. (Against this theory we have the cure of eclampsia in pregnancy with the foetus alive, and delivery at full term without recurrence of the fits.) This view, in their opinion, is confirmed by the fact that there is a great predisposition to eclampsia in multiple pregnancy. ~~(Sturmer has recently reported a case where eclampsia appeared during different pregnancies: in this case the kidneys were only very slightly affected during the attacks).~~ They also point to the frequency of the death of the foetus during eclampsia; to the work of a certain school, who hold that they have demonstrated that the salvation of an eclamptic patient lies in the rapid evacuation of the uterus; and to statistics which prove that eclampsia is most fatal when it occurs during pregnancy, and less during labour, while the post-mortem variety is the least fatal. As regards the last

	No. of Cases.	Fits Ceased After Delivery.	Fits Continued.
Schreiber	105	63	42
Zweifel	129	55%	45%
Bidder (forceps cases)	...	66	40
Oldhausen	143	92	51

Observer.	Mortality.
Veit.....	2 in 60 cases - 3.3%
Bidder	400 cases, 17% deaths.
Knapp	22 " 4.5% "
Fehling	17% "

statement, however, several prominent obstetricians deny it, declaring that in their experience it proved as fatal, if not more fatal, during this period than during pregnancy or labour. Again, eclampsia is very seldom found before the 5 $\frac{1}{2}$ and 6th months, just when the waste products are beginning to be produced, so to speak, in sufficiently large quantities for the maternal organism to take notice of. A very important argument against this theory is that many cases

are on record where eclampsia broke out during pregnancy to disappear on the exhibition of appropriate treatment, so that the patient goes on to full term without the reappearance of eclampsia, although the foetus was still increasing in size, and consequently more waste foetal material was being poured into the maternal system.

Baron and Castaigne have lately carried out experiments on the transmission of substances from mother to child and from the child to the mother; they found that the transmission of substances from the foetus to the mother stops immediately on the death of the foetus, and that they could poison the mother by injecting poisons into the foetal circulation.

Wilke, Woyer, Dohren, and Stumpf have all reported cases, where the children borne of eclamptic mothers were attacked by a condition closely simulating that of the mother. Schmid also reported a very interesting case, and passed in review all the cases so far reported by other observers. All these observers were struck by the similarity between the attacks of the child and that of the mother. Not only did Wilke and Woyer find the clinical condition identical in their cases, but that the urines of the child and mother were of the same chemical composition, and, even on microscopical examination, showed the same constitution. These are the only observers who have been able to report such similarity. One is forced to conclude, therefore, when one finds a child born of an eclamptic develop convulsions very shortly after its birth, that it was contaminated by the same poison which produced the eclampsia in the mother. There are very few cases in our literature, however, where it has been absolutely proved that the death of the child was caused by the same poison as caused the eclampsia in the mother.

A great deal of work has lately been done on the pathological changes found in the organs of the child. The bulk of these changes are very similar to those I have already described under the pathology of the mother, and can only point to the circulation, in the blood of the mother and of the child, of a toxin, or toxins, the same being capable of producing such lesions. The experimental work of Krönig and Füth, upon the osmotic pressure of the foetal and maternal blood at each side of the placenta, shows that substances may pass from the mother to the foetus, and vice versa, and also that the above conditions are quite consistent with what we found.

The work done in this department, however, has not done much to elucidate the exact nature of the actual poison in eclampsia.

CONCLUSIONS.

The most important conclusions regarding recent researches into the etiology of eclampsia are to the following effect:

There has been a total failure to find out the causation of eclampsia by any end-product or intermediary product of metabolism.

The most significant experimental work, in my opinion, is that which points to the placenta forming toxic products. These may not only cause albuminuria and eclampsia, but many of the vague signs and symptoms complained of in pregnancy.

Most authors are agreed that in eclampsia we have a condition of toxic poisoning; but, when we come to consider their views regarding the poison, or poisons, which may be etiologic of this disease, and their source, we find great divergence of opinion.

We have already seen that eclampsia is itself but the expression of many pathological changes in the organs, and that we have many different types of the disease.

Some observers have gone the length of saying that each type is the result of a separate poison, while others affirm that the disease is caused by the combined action of complex poisons. But, even if there be only one poison, we might expect to meet with very different symptoms in different patients, depending on the idiosyncrasy of the patient; for one organ may be more vulnerable to the poison than another; or, again, the toxin may have a special defective action for a special organ, and consequently break down earlier - its insufficiency being expressed by certain signs or symptoms.

DIFFERENT TYPES OF ECLAMPSIA.

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We have already seen that the post-mortem results show differences in the pathological changes in the organs, so that we must not look for a uniform clinical history; and this is what happens, for we find great differences in the clinical history of different cases.

For example, we see cases where the patient has two or three fits which come on during labour, and which in some cases are the result of some reflex stimulation. Such cases are invariably mild. We know that many cases of convulsions occur in adults (either from cold or gastric disturbances) who have never had a fit before, nor have they recurred again. One can quite easily understand that in a pregnant woman where her nervous system is in an irritable condition, severe uterine contractions may occasion a fit. Cases of eclampsia have been noted to occur after a shock, fright, or great mental excitement. In these cases one would not expect to find albumin in the urine.

Again, a patient may have very few convulsions (two, three, or four); but, after each, there is very profound coma, and the patient dies without becoming conscious. These are the cases which have been termed "fulminant," or what Vinay has designated "malignant". Another patient, however, may have a very large number of fits where the coma is only slight after each convulsion, consciousness rapidly returning and the patient recovering.

Then, we may have different renal types. In some cases we have anuria or passage of very small quantity of highly coloured urine, or urine dark from the presence of blood, with a large quantity of albumin. Very careful observation has shown that there are exceptional cases of eclampsia in which, especially at the beginning of the attack, albuminuria is present in exceedingly small quantity, or even absent altogether, and in which no lesions of the kidneys can be made out post-mortem. While, again, there may be polyuria, or the urine is passed in normal quantity, or but slightly diminished.

Lastly, there are variations of the hepatic type, where all the signs and symptoms point to the liver as being principally affected.

Ostroil has lately divided eclampsia cases into three groups, viewing the cases from a practical standpoint:

1st.- Those in which one or two fits occurring during labour, and in answer to some reflex stimulation - such as obstetric manipulations. In these, treatment is unnecessary.

2nd.- Those which end fatally or show at the autopsy old lesions of the heart and kidney, or other organs. No treatment will save such.

3rd.- Those in which, if fatal, the ordinary pathological changes are seen (degenerations, thrombosis, haemorrhages) in the liver, kidneys, brain, etc. In these, treatment may alter the course of the disease.

TREATMENT OF ECLAMPSIA.

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PROPHYLACTIC TREATMENT.

The theory of eclampsia which has the most support today is, as we have seen, that of toxæmia; and one of the earliest expressions of that condition is the appearance of renal incompetency, as evidenced by the appearance in the urine of albumin and tube-casts, as well as by other signs and symptoms. It is the treatment of this condition which constitutes the prophylaxis of eclampsia, and which is now regarded as so important. In fact, Edgar and Davis, of America, and some continental observers, consider that it is in many ways of far more importance than the curative treatment. Many writers account for their decreasing mortality from this disease by the early recognition of the pre-eclamptic state and the adoption of prophylactic precautions.

(A) GENERAL PROPHYLAXIS.

American writers hold that the prophylactic treatment of eclampsia means the early recognition of the toxæmia of pregnancy, whose signs and symptoms I have already described. If these are efficiently treated, no attack of eclampsia will occur. If signs and symptoms point to the organs of elimination being at fault, or the organs of defence being weak, we must correct this condition by appropriate treatment; and, if this fails, premature labour must be brought about, - if these rules were adopted, they hold that we should see fewer cases of eclampsia, and also that many lives would be saved.

In the very first place, then, will stand the rational treatment of pregnancy. If pregnant women paid more attention to hygiene, perhaps the physician would hear less of those minor symptoms of pregnancy, and certainly many of the dangers we have described above would be averted. The most important of all measures to be taken to ensure good health during pregnancy are proper attention to the bowels and the skin. The functions of the skin, bowels, and kidneys are intimately connected with each other. The old experiment, of producing albuminuria in a healthy dog by varnishing its skin, shows the intimate connection of the skin and kidneys; while one can very easily demonstrate in cases of albuminuria that increased function of the skin or bowels leads to improved condition of the kidneys, and diminution in the amount of albumin in the urine.

I am afraid that the bulk of women in our country are averse to cleansing of their skins thoroughly during pregnancy. More attention ought to be paid to the condition of the skin at this period than at any other. Tepid baths ought to be the daily routine, with energetic rubbing with rough towels, to promote the healthy action of the integument. Light, warm, and suitable clothing ought to be worn, and worn loosely, - tight corsets being tabooed. Exercise in the open air with regular meals, which ought to be light and nutritious, will go a long way to keep the patient in fit condition; and, lastly, the bowels must be regulated. Here care and habit will go a long way to promote their regular function. Strong purgatives ought to be avoided, reliance being placed on such aperients as cascara, liquorice powder, ~~Hayden's~~ ~~Sanos~~, or other waters; while the drinking of lots of fluids - e.g., Imperial drink - will not only help in flushing out the kidneys, but be in other ways beneficial.

If one could carry it out, the regular examination of the urine of a multiparous woman every ~~an~~ days or once a fortnight, and of that of a primipara every week during the last three months of pregnancy, has a great deal to recommend it. At any rate, every primipara ought to be cautioned to seek medical advice whenever she complains of headache, with disturbance of vision, dizziness, etc.

I may also say, at the same time, that I have found, among working people especially, the belief that the ailments of pregnant women, be they severe or light, are perfectly natural phenomena; and also that they seldom seek advice unless marked oedema of the face sets in (they ignore oedema of the legs altogether, as they regard it as being practically a normal condition), or should severe vomiting

occur. (As I pointed out before it is now recognised that extensive oedema, instead of adding to the gravity of the prognosis, is in a way a safety-valve. So we find that in treatment the cases which have extensive oedema are much more amenable to remedial measures, and also under appropriate treatment are less liable to develop eclampsia than those in whom it is absent.) So it is that in many cases the eclamptic fit is near at hand before they think that anything out of the ordinary is happening; or, indeed, a fit itself may be the primary indication of the serious condition they are in. On enquiry, in practically all the cases, it will be found that many signs and symptoms of approaching eclampsia presented themselves to these women - only to be ignored, as nothing in the appearance of these suggested to their minds anything extraordinary.

When the premonitory signs and symptoms of eclampsia, - such as a rapid and hard pulse of high arterial tension, headache, oedema, lassitude, anorexia, deficiency of urine, and constipation, - are found, an examination of the urine ought to be made at once. The percentage of urea eliminated ought to be ascertained and the presence of albumin looked for, although the absence of the latter does not in all cases mean that the patient will not develop eclampsia. When these are present, ^{the patient must be} ordered to bed at once, as absolute rest is one of the best means we have of combating the onset of eclampsia. Indeed, we have already seen that an eclamptic seizure sometimes even follows on a hard day's work or excessive exercise. As the symptoms show signs of ameliorating, she may be allowed up a little, and afterwards, as improvement goes on, slight exercise may be indulged in out in the fresh air; but great care must be taken against fatigue. Where exercise is contraindicated, a course of massage is of great value in stimulating the skin, and also by helping the muscles to get rid of waste products. Patients ought to be very particular in avoiding cold and damp, which might, by placing an extra amount of work suddenly upon the kidneys, throw the latter out of gear, and thereby determine an attack. It is also very necessary to protect the patient against excitement, worry, and anxiety; and she should avoid also the going into crowded rooms where the atmosphere is loaded with organic impurities. Alcoholic stimulants should on no account be allowed.

The amount of nitrogenous food ought to be reduced to a minimum.

The ideal method of treating the pre-eclamptic condition is by means of a rigid milk diet, as advised by Tarnier and many others. The former observer is so sanguine in his belief in the efficiency of this treatment that he says: "When a patient suffering from albuminuria has been on a milk diet for a week, she almost certainly escapes eclampsia"; and Dührssen is as dogmatic in his advocacy of this treatment, for he says that he has never seen yet a case of eclampsia occur among numerous cases of the kidney of pregnancy where this method had been adopted during the pregnancy. Bouchard says: "From whatever side we look at it, milk is opposed to all the sources of intoxication." Pinard found that out of 5000 pregnant women, many of whom had albuminuria to a slight degree, 61 had albuminuria in a very marked degree. All were placed on a milk diet, and none of them developed eclampsia. All, then, are agreed as to the great value of a milk diet where the premonitory signs and symptoms of eclampsia appear. It was Jaccoud who first insisted on the great value of a rigid milk diet in albuminuria; but Tarnier was the first to apply the method to the treatment of eclampsia. The plan of the latter observer was to give:

1st day	1 litre of milk	-	2 portions of food.
2nd "	2 litres "	-	1 portion " "
3rd "	3 " "	-	$\frac{1}{2}$ " "
4th & following days	3 " "	-	No other food or drink.

But, in severe cases the preceding graduation was not observed, the patient being placed at once on 3 litres of milk per diem.

As the adoption of an exclusively milk diet is apt to be irksome to the patient, the following may with advantage be followed: For the first few days the diet ought to be exclusively milk: it will be found that it is better to begin with this, as the patients do not object so much when it is rigidly adopted to begin with, and only for a few days. The milk may be given either hot or cold,

or mixed with boiling water, soda, or other table water; - or a pinch of bicarbonate of soda may be added to it, which often makes it more digestible. At least two quarts of milk should be taken during the twenty-four hours. She should at the same time be allowed an abundance of fluids to drink - water, Imperial drink, lemonade, etc.

To keep the bowels open at the same time, the method I follow is to give a 6-gr. dose of calomel at night to begin with, followed by a dose of salts in the morning. After this, I give a 2-gr. tabloid of calomel every evening, following up with some of the lighter salines (apenta, Hunyadi Janos, etc.) in the morning. I have found the combination of calomel and saline act very well. To relieve the monotony of the diet, in four or five days she may be allowed a little bread and butter. The treatment usually results in increased action of the kidneys, with decrease in the amount of albumin. The patient may now be allowed fish or chicken with a little tea, and, where it agrees with the patient, fruit as well: the citrates, etc., which the latter contain, may act as diuretics. The more energetic the action of the kidneys, the more extended may be the diet; but red flesh-meat must be forbidden at all times.

It is very necessary to look carefully to the action of the skin.

In the milder cases, daily tepid baths should be indulged in, with energetic friction with rough towels, while the clothing must be warm. Massage is of considerable value in promoting healthy action of the skin. When the case is more severe, we should have recourse to hot baths or hot packs, - the latter, from the ease with which they may be got ready, are especially valuable in promoting diaphoresis.

(B) PROPHYLAXIS BY MEDICAMENTS.

As regards the stimulation of the skin and kidneys, certain of the simpler diaphoretic and diuretic mixtures may be tried - such as potassium bicarbonate, potassium acetate, digitalis, solution of the acetate of ammonium, etc; diuretin is also a valuable diuretic, and one much used. If the patient has made no progress under the above treatment, we will have recourse to other remedies. It is now recognised that the skin does not excrete much waste material, and that its chief function is the elimination of water; and by doing this it regulates at the same time the temperature; but by promoting its healthy action we not only relieve the kidneys of a certain amount of work, which in their weakened condition is a great consideration, but at the same time the vascular tension is lessened.

Pilocarpine. - Although the majority of physicians are opposed to the use of pilocarpine during eclampsia, many advocate its use during the pre-eclamptic stage; but I think that, as there is always the risk of oedema of the lungs and glottis and profuse bronchial secretion causing embarrassment of the breathing and great depression of the heart's action from its use, complications which we dread should eclampsia develop, and as we get quite as good results from less heroic treatment, - its use ought to be discarded altogether. Furthermore, the drug is especially dangerous where cardiac troubles exist.

Nitroglycerine has many advocates, and may be employed with advantage.

If the premonitory signs and symptoms are marked, there is nothing which acts so well in stimulating diaphoresis and diuresis as the giving of large injections into the bowel of saline solution, or the employment of saline subcutaneously. We shall discuss this mode of treatment more fully afterwards under curative treatment.

Chloral and Bromide. - If the patient is very restless, sleeps badly, and is irritable or excited, chloral and bromide are of the utmost service.

As regards the administration of morphia in the pre-eclamptic stage, - although most observers use chloral and bromide to relieve nervous excitability and restlessness, not a few place great reliance on the administration of morphia; which, they declare, instead of interfering with the function of the kidneys, is actually of

value,relaxing the contracted vessels,and so producing diuresis, while it allays the restlessness quite as efficiently as chloral. Nicholson greatly advocates its use in combination with thyroid extract,as he holds that it ~~has~~ has an inhibiting action on metabolism, with the result that less toxic material is produced,and that it at the same time quickly re-establishes the diuresis.

Thyroid Extract.- The administration of thyroid extract in the pre-eclamptic state was a form of treatment introduced by Nicholson, who has written several papers advocating its use in the albuminuria of pregnancy,the pre-eclamptic state,and in eclampsia.The views which lead to his adoption of this drug we have already discussed when dissertating upon the theory of thyroid inadequacy as the cause of eclampsia.Where there is marked diminution in the quantity of urine,oedema,headache,and vomiting,the patient should be placed on 5-gr. doses of the extract twice a day.In administering this agent,Nicholson pushes it till even symptoms of thyroidism appear. In this condition the vessels are in a state of absolute relaxation, with the result that diuresis and diaphoresis are established.It also aids the metabolism of nitrogenous substances,and the formation of urea;and he also considers that it has some antagonistic action to toxins.

A considerable number of observers have used it since its introduction,and the bulk of them agree as to its value as a prophylactic.One is struck,in looking through Nicholson's papers on this subject,by the fact that in several instances in different patients who were improving under thyroid treatment,as soon as the drug was suspended,the symptoms returned - to disappear,however,on the resumption of the remedy.I have had no experience with it in eclampsia;but I used it once,with beneficial results,in a patient suffering from albuminuria of pregnancy,where there was considerable oedema present,and the urine - scanty in amount - was loaded with albumin.From milk diet,administration of calomel and salines,and thyroid extract she derived great benefit,and was delivered of a strong and healthy child at full term.

Venesection.- Where the symptoms become very threatening and urgent,with the manifestations of renal insufficiency,with the pulse rapid,full,and of high tension,and after these and other remedies have failed,- bleeding is a very valuable means of averting convulsions.About twelve ounces may be withdrawn;and,after its removal,saline solution ought to be administered subcutaneously or by the bowel.

Induction of Labour.- If the premonitory signs and symptoms of eclampsia persist,- notwithstanding the carrying out of these measures I have already indicated,- and become also graver and more threatening,the question of interfering with the course of pregnancy will present itself for earnest consideration.From my reading,I find that the majority of British observers are against the induction of premature^{labour},holding that any interference with the course of pregnancy will precipitate the convulsions.But other writers,American and German,strongly maintain that the correct method of treatment is the rapid evacuation of the uterine contents.We have no means of gauging,from the severity of the pre-eclamptic condition,whether or no the impending attack of eclampsia will be severe. Again,the onset of convulsions usually brings on labour,with further serious danger to the child.I shall discuss this question more fully under obstetrical treatment.

Before leaving the subject of prophylaxis,we must again repeat that certain cases have been reported where the patient was under close observation,and where there was no albumin present in the urine up to the appearance of the fits,and no signs or symptoms present to point to the near approach of eclampsia;also that cases of albuminuria and oedema are met with where by proper treatment the oedema disappeared,the albumin became less,and the output of urine was increased;and,notwithstanding this,eclampsia appeared under these improved conditions.

CURATIVE TREATMENT.

Under this heading we shall separately discuss the principal methods and remedies which are used to combat this disease, and conclude by describing a remedial procedure which may be followed in the bulk of cases. It is necessary to state here that there is no specific in this affection, and that consequently each case must be judged and treated on its own merits. At the same time, also, we must remember that eclampsia is not a specific disease, but the expression of many different pathological conditions; and this may in a way explain the reason why treatment varies so much in its efficiency.

GENERAL TREATMENT.

To begin with, we must see that the patient does not injure herself during the convulsions by knocking against the bedstead, or by actually falling out of bed. Care must be taken to protect the tongue against injury by being bitten, as frequently happens during convulsions - sometimes the tongue has been found terribly lacerated. The handle of a tablespoon may be wrapped in a handkerchief, and inserted between the teeth for this purpose. Care must also be taken, should the tongue fall back during the convulsion to pull it well forward - as this condition frequently adds to the asphyxia present by interfering with the respiration. This would be prevented were the patient to be placed in the lateral position.

Although admitting that oedema of the lungs is fairly common in eclampsia, the Dublin writers hold that many cases, which are supposed to be oedema, are conditions caused by the presence of fluids from the mouth, - either natural secretions or medicines or *nutrient* fluids, - finding their way into the bronchial tubes. Consequently, they insist on keeping the patient lying in the lateral position instead of the recumbent, as in the former position fluids formed in the mouth are less liable to enter the tubes. So that we see that this position helps respiration both directly and indirectly.

The patients must on no account be dosed, as is frequently done when they are in a semi-unconscious condition. If drugs or nourishment are to be administered by the mouth, I think the best method is to insert them directly into the stomach, by means of a soft tube passed through the nose into that organ. This is the method adopted in the Rotunda, where, in addition, the stomach is thoroughly washed out before anything is given. I think that this is also a very much better and more scientific method of clearing out the stomach than by the administration of emetics, which latter used to be the routine practice and is still employed in certain quarters. The stomach, at any rate, is usually emptied by the vomiting which is so frequent before the eclamptic seizure. Where an emetic is administered there is always danger of the vomited material finding its way into the air-passages, during a fit or coma, and causing septic pneumonia.

The bladder should always be emptied by a catheter. In the bulk of cases it will be found that there is only a very small amount of urine present, but there have been cases recorded where large quantities of ammoniacal urine could be drawn off by the catheter, - on the accomplishment of which the convulsions ceased forthwith.

Those who believe in the mechanical causation of eclampsia advocate placing the patient in such a posture as will relieve the pressure - e.g., the genupectoral position, or the semiprone with the legs well drawn up.

MEDICINAL TREATMENT.

The convulsions may be arrested by:

- I. The administration of sedatives.
- II. The elimination of toxic substances from the blood and tissues.
- III. Obstetric treatment.

Regarding the medicinal treatment of this disease, - it is difficult to come to a definite conclusion as to the value of many of the drugs employed, for in some cases more than one is used. A combined method of treatment is adopted by many, and also because

cases of eclampsia vary so much in type and in severity. Many cases, fulminant in type, or what Vinay called malignant, are hopeless from the beginning; in others, the patient may be in a moribund condition before the arrival of medical aid; while, again, others would recover without any treatment, - or one might say even in certain cases in spite of the treatment adopted. The more serious the case of eclampsia, the more does the medical man fly from one remedy to another, the result being that none of the remedies adopted have a chance of succeeding. In the old days, and perhaps even now, I strongly believe that many patients died from overmedication. I consider that we are too prone to think that, because certain eclamptic patients can stand enormous doses of poisonous drugs (e.g., chloral, veratrum viride, and morphia), they are proof against anything. The simpler our method of treatment in any disease, the nearer do we approach a rational treatment. For a great number of years the treatment of eclampsia suffered from the multitudinous array of theories regarding its etiology, the natural outcome of which was that we had an equally imposing array of drugs and variety of methods adopted for its relief. The bulk of these drugs have now disappeared from the modern list; and methods of treatment formerly held in high repute have been discarded. The result is that, although there is no routine method of treatment which has become generally adopted, nevertheless, the number of drugs employed has been narrowed down to three or four.

As regards the bearing of the etiology of this disease on treatment, - I do not believe that the mere fact of our knowing what the actual poison is which causes the toxæmia will help us much in treatment, provided we develop our treatment on the lines that we are dealing with a peculiar toxæmic condition. Most modern writers are agreed in accepting this theory as a good working hypothesis of a rational method of managing any given case. for the development

I. The Administration of Sedatives

Chloroform.

With regard to the administration of chloroform, modern opinion has changed very much from the days when, replacing venesection, - which from abuse had become discredited and practically abandoned, - it was regarded as the routine method of treatment for eclampsia. Formerly, it was a common thing to keep the patient under its influence for hours at a time; and Vinay, and many others, reported cases where patients were kept ten or twelve, or even as long as twenty-four hours under this anaesthetic.

The members of the French school, and other advocates of chloroform narcosis, say that it tends to lessen the number, and modify the severity of the fits; that it not only prevents the temperature becoming high, but actually lowers it; that it relieves the vascular tension; that it has a beneficial action on the comatose condition, in which it may be administered with safety; and that it relieves the venous congestion, a condition so frequently found in eclampsia.

If chloroform is to affect the convulsions, there can be no doubt that the proper method to do this is to keep the patient deeply under its action; and Schmorl, and many others, have pointed out the danger of causing fatty degeneration of the heart and other organs by deep chloroform narcosis. Lately, excellent experimental work has been done by Thomson on the relation of chloroform anaesthesia to urinary secretion; and his conclusions have considerable bearing on the value of prolonged anaesthesia in a disease where the kidneys are so often profoundly affected - for he found that while in the early stages of chloroform narcosis the quantity of urine is increased, during full anaesthesia, or after prolonged anaesthesia, the urinary secretion is always diminished, and may even be suppressed.

In the Rotunda Hospital, it is claimed that the results have been much better since chloroform treatment was abolished and morphia used instead, as they found that deep narcosis can be more easily produced by the latter, whose action, too, is more easily regulated and controlled than that of chloroform.

The majority of obstetricians now seem to limit the use of chloroform to the control of the more severe convulsions, and also to generally facilitate the manipulation contingent to delivery. Where the convulsions are severe at the very outset, it is advisable

to use chloroform till the other drugs employed have had time to act; but its use should not be kept up for any length of time, as there can be no doubt that protracted chloroform narcosis leads not only to enfeeblement of the action of the heart, - when our whole treatment ought to be directed to the saving of an organ whose capacity is tested to the utmost in this disease, - but also impairs the functional activity of the liver and kidneys, - two organs which play a great part in eliminating, and protecting the body against, the poison of eclampsia.

In looking over the literature on treatment, I find ~~that~~ many observers who only administer the chloroform during the fits. But, surely, the anaesthetising of a patient takes much longer than this, and the fit would be over long before the patient was sufficiently under its influence for it to have any effect. We also know that during the height of a fit the chest wall is fixed, and chloroform given at that time could not find its way into the circulation as the patient cannot breathe. Again, some affirm that the administration of chloroform should be begun just before a fit comes on; but I think that often it will be very difficult to say definitely when a convulsion is at hand. I have seen a patient in a most restless condition, and expected every minute to see another convulsion which did not appear for long after the danger-signals were displayed; while at other times I have seen convulsions develop without the slightest warning.

Chloroform is also used where the patient is very restless; but I think that in these cases as good, if not better, results are obtained from chloral, to which may be added bromides and morphia. Chloroform should not be administered to any great extent in cases of post-partum eclampsia, where there has been much haemorrhage - on account of its great tendency to lower the blood-pressure.

I would administer chloroform, then, only at the commencement of an attack, where I desired to control the convulsions till morphia or chloral had had time to operate; and also in obstetrical manipulations and operations.

Chloral.

This drug has long been a favourite in France and in our own country in the treatment of eclampsia, although recent reports show that it is not so generally employed as it once was. When the administration of chloroform was the routine practice adopted to combat the convulsions, chloral was also employed along with it - as it gave the same action as chloroform, but more prolonged and continuous, and it did not require such constant supervision on the part of the obstetrician. The popularity of this drug owes a great deal to the writings of Winckel and Charpentier, who enthusiastically supported it. The former has had wonderful results with it - only 7 deaths out of 92 cases. The advocates of the morphia treatment point to the large foetal mortality of Winckel as a definite contraindication to the exhibition of chloral, - he had a foetal mortality of 77%, - but this high death-rate was not due to the action of the drug, but to the fact that our author did not believe in accouchement forcé: in fact, he postponed operative interference till the very last. Chloral has many supporters in France; but few in Germany favour this line of treatment, the bulk there adopting morphia. As Veit - the great advocate of the morphia treatment - believed in what we regard as heroic doses, so Winckel and Charpentier - the great exponent of the chloral line of treatment - used very large doses. Few observers can boast the wonderful results which followed their daring methods; and this, Veit and Winckel observe, is because these drugs are not sufficiently pushed to warrant success.

Ordinary doses of chloral do not have much influence on sensibility and reflex excitability, but these disappear on the exhibition of large doses. It depresses the functions of the spinal cord, and also of the centres at the base of the brain. It lessens arterial pressure, and at the same time distinctly lowers the temperature, while it also helps to dilate and render more pliable a rigid os, - all of which actions point to its being a useful agent in eclampsia. One good feature about its administration is that it is usually freely eliminated from the body, having no tendency to accumulate. Some hold that, as it decomposes in the blood into chloroform, its dose is difficult to regulate; and they condemn the method of administering this drug which is usually adopted (that is, by the bowel),

as the medical man is quite in the dark regarding what is going on there, and as to how much of the remedy is being absorbed. They also say that when chloral is given in full doses, it aggravates to a marked degree the serious after-effects of eclampsia.

Chloral has many supporters in France; and Charpentier and Tarnier regard it as the remedy par excellence in eclampsia. The former found that, in 239 cases collected from different sources, the mortality when chloral alone was used was 4%, and when it was used in combination with other drugs - 8.5%. Very few would care to give the enormous doses advocated by Charpentier and Winckel. The former starts with 3i-doses per rectum: if not retained, he repeats the dose until it is. In the course of six hours, he repeats the dose, and, if necessary, continues its administration every five or six hours till he has administered as much as one-half ounce in the twenty-four hours. Should the convulsions show no signs of abating, he shortens this interval. Charpentier also holds that in no case should the administration of chloral be abruptly stopped; and he always gives one or two smaller doses after the convulsions have entirely disappeared.

When chloral is given by the rectum, it should never be employed mixed with water, as in this solution it is very apt to cause tenesmus. It ought to be mixed with milk, to which may be added a well-switched egg. The bowel should be thoroughly washed out before this emulsion is injected. The method of administering the drug by a tube passed into the stomach has much to recommend it; it is thoroughly scientific, and the one adopted by numerous writers of repute. Doses of 20 grains may be given to begin with, and repeated in an hour. The stomach should be washed out before commencing its exhibition in this way. When given by the mouth with the stomach-tube, large quantities of milk should be administered along with it.

In post-partum eclampsia, chloral is generally regarded as being very useful; but I did not secure good results with it in my case.

Morphia.

The bulk of modern writers are now agreed as to the great value of morphia in eclampsia, and most of them use it as a routine medicament. British practitioners were amongst the last to adopt it. The knowledge that it had a most injurious effect in nephritis, and that the chief symptoms in the bulk of the cases of eclampsia pointed to renal insufficiency, accounted for their temerity in prescribing it. It was left to the Dublin school to popularise this drug in our country; and the splendid results obtained there went a long way to overcome our insular conservatism and prejudice. The reputed bad effects that this drug has in renal disease is certainly not seen in the majority of cases of eclampsia, most patients bearing large doses without showing signs of morphia-poisoning, and nowadays morphia is administered by numerous practitioners, in acute and recent forms of nephritis, whenever the signs and symptoms call for its exhibition - although most still withhold it in chronic cases where there is much degeneration of the epithelium. Morphia regulates the convulsions by allaying the irritability and sensibility of the cerebro-spinal system. It appears to have quite as good a sedative action as chloroform, and it certainly has not the great depressing action of the latter. The opponents of the morphia treatment, however, say that if chloral and chloroform have a depressing action on the heart, morphia has the same on respiration. To obviate this, the Rotunda physicians keep the patient lying on the side; while others recommend the addition of atropine to counteract this effect. Byers, again, declares that morphia tends to lessen the acute catarrhal process in the lungs, which often brings about a fatal termination in eclampsia. Morphia has one special weakness, and that is its inhibiting action on the intestinal secretion; but this influence is readily counteracted by the routine exhibition of purgatives. As morphia acts by inhibiting metabolism, it prevents excess of waste products being thrown into the blood, and by so doing to a considerable extent relieves the liver and kidneys. The supporters of this drug hold that not only has morphia no detrimental action on the kidneys, but it actually promotes and encourages diuresis - as in large doses it acts as a powerful vaso-dilator, removing the state of spasm in the renal vessels so commonly met with in eclampsia.

The use of this drug received a great impetus from the work of Veit, in Germany, who has had wonderful results with it. He has employed it in 60 cases, with only 2 deaths. I do not think that there are many, or any, observers who can publish such results as that; and Veit declares that the reason of failure with this drug is because it is only used in a half-hearted fashion: certainly few can be found who would care to push the drug in the daring way he does. He has given as much as 3 grains in four hours.

The method adopted by the majority of practitioners is to give an initial dose of half a grain, and follow this up with a quarter of a grain, every two hours, till the convulsions cease - 2 grains per diem being the maximum amount exhibited. The dose Oldhausen starts with is $1/3$ grain; but he increases this rapidly to $9/10$ th gr., while he has given as much as 11-12 grs. in four days. Some observers have reported cases of poisoning by this drug, even when given in $1/2$ -gr. doses; and Fehling declares that large doses of morphia are unnecessary, as the same effect may be obtained by moderate doses. Fehling has come across several cases of morphia-poisoning in the mother; and there can be no doubt that death of the foetus may sometimes be traced to a fatal narcosis by this drug; and often the child is born in an asphyxiated condition, or breathes with difficulty, and takes a long time to bring round.

When morphia is used, it is frequently in combination with some other line of treatment. In our own country, many combine it with chloral; while in America, when it is used, it is generally with veratrum viride - because, besides its own effect, it makes the administration of that drug safer and surer in its action.

We shall treat these other combination of treatment more fully later on.

Veratrum Viride.

Of this drug I cannot write from personal experience, as I have never employed it; and, so far as I am aware, it has been very little used in this country. Cases where it has been employed have certainly been reported in our journals from time to time; but where it was used it was generally in conjunction with some other drug; and I have failed to find anywhere a series of cases in which it was given ~~as the extent~~ ^{to the extent} so common in America. ~~There~~ this drug has had a wonderful vogue in eclampsia since it was first introduced by Baker, in 1859.

This drug is a powerful spinal and arterial depressant - its diaphoretic action being the direct result of this profound arterial impression. Edgar, and other American physicians, hold that veratrum viride stands second only to chloroform in controlling convulsions. Its chief action in eclampsia is in depressing the motor centres of the spinal cord. By its use, also, the pulse is remarkably diminished; the temperature is reduced, and the rigidity of the cervix relaxed; while diaphoresis and diuresis are promptly effected. Jewett holds that it has been proved by experience that no convulsions occur when the pulse is brought in rate down to 60 per minute; but several writers have lately shown that this statement is too sweeping, - as in some cases convulsions still appear, with little diminution in their severity, with a pulse-rate of under 60; that the patient's condition does not always improve with a diminished pulse-rate; and also that ^{in extra cases} the pulse-rate was unaffected by full doses of this drug.

The method usually adopted is to use the fluid extract subcutaneously, giving an initial dose of 15-20 minims, and continuing with 10 minims ^{every 15 min.} till the pulse-rate is below 60. Care should be taken during its administration to have the patient in the recumbent position, as the erect position (or where too much has been given) may lead to a marked cardiac action, with vomiting and collapse. Where this occurs, recourse should be had to stimulants, and morphia administered.

This drug was used with extraordinary success by Parvin, who was able to save 92 out of 100 patients with it. The average mortality in America from this disease is between 20 and 25%. Hirst is another American who has had strikingly successful results with this remedy - in nine years of his hospital service he had only two deaths. But he, and many other Americans, combine other remedies with it. Hirst, for example, used chloral and saline injections, and administered chloroform during the fits; whilst others used it in combination with morphia - so that in many cases it is difficult to

come to a correct conclusion with regard to its efficacy. Mangiagalli has found that the drug is very valuable in eclampsia, in pregnancy, in labour, and during the puerperium. In 18 cases, only 1 died; and death in that case occurred nine days after the cessation of the fits. In one case, during pregnancy, eclampsia appeared at three different times - always to disappear, however, on the exhibition of veratrum viride.

In examining the writings of those who have employed it, one is forced to conclude that this drug is certainly of value in strong robust women in the early stage of the disease where the pulse is of large volume, bounding, and rapid - as it certainly does good in relieving the high tension and reducing the pulse-rate. But when the pulse is weak and irregular, or after coma has been present for any length of time, this drug - one would imagine from its physiological action - could only do harm.

Pilocarpine.

I only mention this drug here as it was, at least until quite recently, very largely used, and to point out the dangers and disadvantages contingent to its exhibition. The reason why its employment was advocated in eclampsia was that it is the most powerful diaphoretic we possess; and the supporters of this treatment naturally thought that this would be a good method of ridding the system of the eclamptic poison. At the same time, however, pilocarpine is a powerful depressant of arterial pressure. Since Fordyce, Barker, and Braun pointed out the great dangers of its use, many others have written strongly against its employment. Pilocarpine often causes great increase in the bronchial secretion, which embarrasses the action of the lungs; and it appears to have a great tendency to increase those pulmonary complications which are so common in eclampsia, and which we are so anxious to avoid. It also causes oedema of the lungs and glottis, and depresses the heart very much. As for its action in ridding the system of toxic material and reducing the blood-pressure, we have other methods of treatment which are much more efficient in this respect, and also without the dangerous action of pilocarpine. Notwithstanding all that has been written against the employment of this drug, I still note in the journals cases where it has been used, too, after all other remedies had failed; and at the very time, I should think, that this ~~drug~~ ^{deadly drug} is most apt to act injuriously.

Combined Medicinal Treatment.

We have already seen that morphia is very often combined with some other drug. For example, the Americans largely use it in combination with veratrum viride. I shall here describe shortly a few of the more important combinations:

Chloroform and Chloral.

This combination was for a long time the favourite treatment in this country, and it is still one that is largely used. Formerly, the chloroform was given the more prominent position of the two; but, later on, the bulk of practitioners made it subsidiary to the chloral, using it only in the very severe convulsions, or in order to give the chloral time to operate, or during obstetrical treatment, and relying more on the chloral for its continuous action. The greatest advocate for this combination is Tarnier, who also advises venesection in suitable cases, relying on the blood which he has extracted being replaced by absorption from the large quantities of milk which he orders the patient to take, - & if the patient is unconscious he administers the milk through a stomach-tube. The great objection to the chloroform treatment in conjunction with the administration of chloral is its depressing effect, and it is not in my opinion so good as the next combination.

Morphine and Chloral.

I consider this by far the best combination of medicinal agents we can apply in eclampsia; and it is the one I should adopt as a routine treatment in practice. I would begin immediately with a $\frac{1}{2}$ -gr. dose of morphia, given subcutaneously on account of its more rapid action. If the patient be ~~un~~conscious, I would then administer 20 grains of chloral by the mouth, or, if unconscious, 40 grains in an

emulsion by the rectum, as previously described. The chloral is exceedingly useful in allaying peripheral reflex action and restlessness, and it helps at the same time to lessen the severity of the convulsions and prevent the development of fresh ones. I think that when one combines the morphia with the chloral the depression of the heart, which is found when chloral is administered alone, is avoided. This combination also has a most beneficial action on the rigid os. Stroganoff, who considers eclampsia an acute infectious disorder, has had wonderful results with morphia and chloral. He considers that the number of fits diminished notably under the influence of this treatment, while it also had a favourable influence on the course of labour and on the mortality of the children. He had 5 deaths out of 92 cases. Out of these 5 deaths, one died of pneumonia, which he holds ought to be regarded as something quite distinct from the eclamptic seizures; but I think that this ought to be considered a legitimate complication of the disease. One died after being received into the hospital in a moribund condition. Another died, on the twenty-seventh day after eclampsia developed, of septicaemia. A fourth had severe pulmonary oedema on arrival at the hospital; and a fifth died four days after eclampsia disappeared - she had severe post-partum haemorrhage. So that, in 1 of these cases death was exclusively the result of accidental illness, and in 3 it was the result of the very grave condition in which the patient was found on admission to the hospital.

Other Combinations.

Other combinations are of morphia and chloroform, the same being largely used by the Germans; and morphia and veratrum viride constitute a favourite combination with the Americans.

Whatever narcotic, or combination of narcotics, we administer, we must remember that in exhibiting them it is to tide our patient over a certain danger by allowing other methods of treatment to clear the poison from the system, or to permit the organism to reassert itself and combat the toxic substances. We must bear in mind also that they may postpone or prevent the fits, while all the time the toxic matter may be accumulating in the system.

II. The Elimination of Toxic Substances from the Blood and Tissues.

Purgatives.

Whatever line of treatment is adopted in eclampsia, although we do not regard purgatives as possessing all the wonderful virtues which old observers credited them with, I think that all are agreed as to the value of clearing out the bowel - most administering drugs of this class as a routine procedure. Some observers hold that the condition of the intestine plays an important rôle in the causation of the disease, and consequently for them the purgative is the all-important drug to be exhibited. A case has been reported where, after the evacuation of the foetid motions and the thorough lavage of the large bowel, the convulsions ceased. As regards the routine use of purgatives, - we must remember that we have not the tendency to diarrhoea which is so often found in uraemia, where, indeed, it may be very profuse (Osler) - the general condition in eclampsia being one of obstinate constipation.

Purgatives not only remove from the intestines irritant substances, which might otherwise be absorbed into the blood and which might predispose the patient to eclamptic seizures, but at the same time they remove a quantity of serum (in which will probably be found a larger or smaller percentage of the toxins which are causing the disease), and by so doing they lower the high vascular tension so common in this affection. In this way it acts like venesection; but is superior to it, in that no corpuscles are removed. Purgation has a beneficial action on the kidneys, getting rid of waste products, and so allow the kidneys ^{time} to recover by performing part of their work. As we saw in discussing the pathology and etiology of this disease, the liver is frequently affected; and its function may be stimulated by a purgative whose principal action is on the duodenum.

Such a drug we possess in claelmel; and it is ^{it} ~~one~~ that I principally rely, administering it in 6-7-gr. doses, sometimes following it up with sulphate of magnesia, - but in every case relying as well on thorough lavage of the large bowel with soap and water, to ~~which is~~ added 3iii of turpentine, as this often relieves the flatulent distension of the large intestine so frequently present in those cases where constipation has been a marked feature of the pregnancy.

Croton oil has, I find from a survey of the recent literature, fallen from the high place it once occupied.

Jalap may be given along with the calomel, whose action it accentuates. In fact, for many years this combination - so enthusiastically advocated by Paul Dubois - was the routine practice adopted. What is aimed at is to get a copious bilious evacuation.

Some observers employ concentrated solutions of salts introduced well up into the bowel. By this means we have the same result, the evacuation of large quantities of serous fluid, as the giving of large doses of salts in concentrated solution (Hey's method), without running the danger of introducing large quantities of fluid into the stomach of a semi-unconscious patient.

Venesection.

As regards bleeding in eclampsia, - we are still suffering from the reaction ~~which~~ set in against the indiscriminate bleeding - a feature of the treatment of eclampsia thirty or forty years ago - the natural outcome of which was to throw discredit upon it. From their experience, the old observers considered that one of the chief ends to gain in a disease where the arterial tension was markedly high was the lowering of the blood-pressure, and that one of the most powerful and rapid methods of lowering vascular tension is by bleeding. I may say here that Herman has drawn attention to the question whether it is always good policy or scientific treatment to advocate reducing high arterial tension, because this may be a protective measure as is found in renal disease, the arteries contracting to prevent the further inroads of the poison, and an attempt as well by the organism to eliminate these poisons by the increased blood-pressure through the kidneys. The extensive bleeding carried out by the older observers (e.g., De Paul, Ramsbotham, etc.) is truly aweinspiring; and we find one observer, Meigs, - in a work on midwifery which was very popular in ~~his~~ day, - writing that: "If there be a case of disease in which bold and daring employment of the lancet is demanded, it is the case of the puerperal convulsion. It is scarcely worth while to open a vessel to draw off 8 or 10 oz. of blood. The patient ought to lose from 30 to 60 oz. at one venesection if possible, and ~~if~~ signs of faintness appear, they should be hailed as the harbingers of success." While another wrote: "The only real resource in the puerperal convulsion is the use of the lance."

In course of time, however, other drugs - e.g., purgatives, and other methods of treatment - were found to produce the same effects as bleeding, which consequently gradually fell into disrepute - more especially after chloroform became more extensively adopted in the treatment of eclampsia. Lately, however, there has been a movement towards a moderate and rational use of venesection in certain cases, and I consider this to be a step in the right direction. But, in this revival of venesection, as usually happens in such cases, we have some coming forward and advocating bleeding - and copious bleeding - not only in those cases in which its use is we think justifiable, but even in cases where the pulse is thin and weak; while other observers fall back on venesection as a desperate remedy to be used only in hopeless cases after all other drugs have failed; and others hold that this method is called for in all cases where delivery does not lead to amelioration of the eclamptic condition, even when the pulse is weak. But the bulk of modern writers are agreed that, if venesection is to be employed at all, it must be during the early stage when the pulse is full and bounding. It will be found of great service in stout and plethoric women when cyanosis is marked. By employing it here, we have not only a rapid decrease in blood-pressure, but also a means of removing a quantity of blood charged with toxic substances from the blood. It is held by some that the removal of a certain amount of toxic blood, by causing contraction of the small vessels, still further prevents the toxic blood from reaching the convulsive centres. Venesection is often combined with saline injections - presently to be considered.

Lumbar Puncture.

Helme advises the withdrawal of a quantity of cerebro-spinal fluid, by means of lumbar puncture, in order to relieve the intra-cranial pressure caused by the increase in the cerebro-spinal tension which brings about the convulsions (Med. Press, April 27, 1904).

Thyroid Extract.

We have already discussed the use of the thyroid gland in the prophylactic treatment of eclampsia, where it has been proved to be of considerable value. As regards its value in the eclamptic seizure, - its use has not been sufficiently extended to allow one to come to a definite conclusion. Nicholson, who ~~has~~ originated the use of this drug in eclampsia, has already reported a considerable number of cases where he has seen considerable benefit follow the exhibition of this substance; and Sturmer, who has had a considerable experience with it, writes that he found, ~~with~~ 30-40 grs. of thyroid given in the twenty-four hours, the urine after the first two or three doses has shown a considerable increase, and by the end of twenty-four hours a very large increase has been noted. However, as Sturmer always used saline injections and also administered morphia (as also does Nicholson), it is difficult to decide how much of the benefit derived was from the administration of the thyroid. Another observer has recently reported a case of eclampsia where he used thyroid subcutaneously (the extract being dissolved in saline), with beneficial results.

Saline Injections.

The treatment of eclampsia by means of saline injections has almost become universal as an adjunct to internal medication. Porak, who has had wonderful results, was one of the first to employ the method: he first bled, and then injected the saline solution subcutaneously. This method he soon discarded for the injection of saline into the bowel after thorough rectal lavage, believing, as he did, that many cases of eclampsia were caused by poisoning from the intestine.

Some authors recommend the infusion into the veins of saline solution; but this method, besides being more difficult, has at the same time an element of danger - for there is always the possibility of the entrance of air into the vessels, and also the risk of sepsis. In the case of an ordinary country practitioner (who has perhaps to battle with a case of eclampsia himself or with the aid of a midwife) this operation, easy enough under ordinary circumstances, might require considerable dexterity with a restless patient or one in convulsions; the risks, too, of air entering the vessel or of the occurrence of sepsis would also be increased. This method is now practically abandoned for two others - either of which gives as good results, and both of which are altogether free from any risk:

In fusion of Saline into the Subcutaneous Cellular Tissue. - This is the method generally adopted; and its popularity owes a good deal to the writings and results of Jardine. Of his latest series of cases, he has been able to report 15 without a death. He first used 30 grs. of bicarbonate of potash with each pint of saline solution, and would inject as much as three pints at a time. The salts of potash, when directly injected into the blood, have a terribly depressing and toxic action on the heart; but Jardine has never found any signs of poisoning when he injected them into the cellular tissue. Lately, however, he has used acetate of soda - 3i to the pint. The apparatus required is very simple - a long rubber tube with a funnel and needle, which should be thoroughly sterilised before use. Another apparatus (Bacon's) has lately been put on the market - it consists of a glass funnel with a long rubber tube connected, by means of a Y-shaped glass tube and two short tubes, with two hollow needles, so that the saline may be injected into both breasts at the same time. The fluid is quickly absorbed, and there are seldom any septic complications; but the patient often complains a good deal of pain in the part for a day or two afterwards. The injection may be repeated in a few hours if necessary.

The injection of saline subcutaneously acts in several ways. In the first place, it dilutes the poison; while, by causing diuresis and diaphoresis, it tends to eliminate the toxin; and it also acts as a stimulant to the flagging circulatory powers. It improves the patient's

general condition, and seems to act like a sedative in allaying muscular twitching and restlessness, while the cyanosis and coma become less pronounced. The infusion should not be used where there is much oedema of the lungs. Some observers hold that, where there is no great deficiency of urine or little or no albumin present, it should not be used; but in such a case I have found saline subcutaneous injections of the greatest value.

The method I adopted in my last case of eclampsia is the one I shall follow as a routine procedure in future: I refer to the irrigation of the large bowel with saline solution, to which may be added diuretics (salts of potash). It is the method advocated by Porak, and carried out by many other observers in France and America. The bowels should be thoroughly emptied by means of calomel and magnesium sulphate given by the mouth, or, where the patient cannot swallow, through a stomach-tube; then the large bowel should be washed out with hot water and soap, to which turpentine may be added. This is effected by means of a soft rubber tube, four feet long, to which a funnel is attached. Large quantities of hot normal saline solution, to which is added bicarbonate of potash and acetate of potash (30 grs. to the pint), should be made. The patient's hips should now be raised and the fluid allowed to run into the bowel slowly, by means of the soft rubber tube inserted well up the rectum. The procedure is very simple, and has all the valuable properties of the subcutaneous saline injections.

Experiments have lately been carried out to ~~ascertain~~ if enemata of saline solution have any action on the kidney; and it has been demonstrated that they have a marked diuretic action, and that they are of the utmost service in flushing out the kidneys in conditions which have to do with the eliminating power of these organs.

Another explanation regarding the action of infusions in eclampsia has been put forward by Hey Groves, who is a great advocate of this line of treatment. He holds that they dilute the blood, and consequently prevent the appearance of the multiple capillary thrombi, while they may even dissolve thrombi already formed. If we accept this theory, it would help to explain the rapid beneficial results obtained when they are employed - the other theory, that the good results are caused by the diuretic action of the saline, not being proved, as this action appears very much later than the improved condition of the patient.

Hydrotherapy.

Hot Baths and Packs. - The hot water bath is used by numerous observers; but one gets the same results, and more easily, by hot wet packs. The latter can be used at any time, even when the patient is in labour and very restless - two conditions which almost prohibit the employment of the hot bath. The hot pack is carried out by wringing sheets or blankets out of very hot water, rolling the patient in them, and placing a macintosh over them, and then covering her well with blankets.

Cold Baths. - In these exceptional cases where the temperature is progressively rising to a very high degree, Herman advocates placing the patient, to begin with, in a tepid bath, and gradually cooling it. She may be kept in from twenty to thirty minutes, when the temperature falls below 102° F. On being taken out, she should be rolled in blankets, and sweating encouraged.

III. Obstetrical Treatment of Eclampsia.

As regards the obstetrical treatment of this disease, we are at the outset of our discussion faced with considerable diversity of opinion. The older school of obstetricians believed that in every case of eclampsia which occurred in pregnancy the proper course to adopt was to induce labour at once, and where it occurred during labour to hasten it as speedily as possible, as in their opinion the rapid evacuation of the uterus added greatly to the woman's chances of recovery.

Owing largely to the work of Veit, Winckel, and, more recently, Charpentier, - another school came into existence which held that the proper course to adopt was to leave the case to nature, aiding

her in the least obtrusive manner possible. When eclampsia appears post-partum the patient is said to always recover. From the figures collected by Charpentier, 12.5% deaths, however, occurred at this time. Early intervention saves the mother, as it is only after several fits that her life becomes compromised; and, moreover, the child is saved, as it is not often killed by the first fit. Buttner found his death-rate drop from 36.6% to 24.6% when he adopted the active treatment in his practice; and Bumm has recently published statistics to show that his death-rate has considerably fallen (from 30% to 12%) since he adopted active treatment. Veit, Winckel, and Charpentier would not interfere if the convulsions occurred during pregnancy or early part of labour, as they hold that rapid artificial dilatation of the cervix led to the development of fresh convulsions, and caused great shock to the patient. They pointed to the fact of the disease being the result of a toxæmia, - which had been going on for some time, till a crisis with the development of convulsions had been reached, - and that one could not expect that the mere rapid evacuation of the uterus would result in the speedy disappearance of the poison. They affirmed that the induction of labour did far more harm than good, and that many have died from accouchement forcé who might have survived had less heroic methods been adopted. They pointed to the deceptive nature of many cases of eclampsia ("the disease of surprises", as Tarnier calls it), as many grave cases which were looked upon as hopeless ended favourably, while benign cases often suddenly changed for the worse and ended fatally; and they dwell also upon the fact of patients recovering after accouchement forcé does not mean that the same patients might not have recovered where less active obstetrical measures had been adopted. The supporters of the expectant treatment say also that in eclampsia, as we have a condition of great nervous disturbance, the condition of the kidneys - that of degeneration - is easily influenced through the nervous system; and that one would expect this condition of the kidneys to be greatly changed for the worse where the nervous system was subjected to increased shock by forcible manipulations. But, where the patient is deeply under chloroform, this latter objection could not possibly arise.

Dührssen, whose work on the treatment of eclampsia has done a great deal to revive the old teaching that the salvation of the patient lay in the rapid evacuation of the uterus, affirms that as soon as the convulsions develop no time should be lost in commencing this operation; for, the earlier it is begun, the better chance the patient has of ultimate recovery. But many find that the proper course is to steer between these extreme views, holding, as they do, that each case should be judged on its own merits, and that no routine obstetric treatment ought to be followed.

The great point of difference between the rival schools is - should labour be induced when eclampsia occurs in pregnancy? Here the first point of importance in this discussion is the effect of delivery on the convulsions: whether they ceased altogether immediately on delivery, or became much milder and less frequent. Also, whether an active line of treatment gives a lower death-rate than the expectant treatment - not only as regards the mother, but also the child.

Herman, in an interesting inquiry into a collection of 2142 cases of eclampsia, found that in 905 cases the fits disappeared after delivery, and that in 816 they continued - i.e., they stopped in 52%. Again, as regards whether active interference has a beneficial effect upon mortality or not, he found, from the cases collected by him, and recent cases where strict antiseptic methods were adopted, - that the difference of 2% to 3% in favour of active interference was too little to justify its use; and also that, if hurried delivery were indiscriminately practised by all who attend labour under all circumstances, the mortality arising from operative interference would soon overbalance the trifling and doubtful benefit of emptying the uterus. From a study of his statistics, Herman holds that there is no call for active interference, and that the woman has a far better chance of recovery where the expectant method is adopted. Zweifel, again, used the expectant treatment, up to 1892, and had a death-rate of 28.5%; while, from that date to 1896, after adopting more active treatment, it fell to 11.25%; but after 1900, when more heroic measures were adopted, the mortality was 23.5%. In 84 cases,

however, where surgical interference was begun immediately after the appearance of the first fit, only two died - giving a death-rate of 6.6%. He concludes that the mortality is 32% with the expectant, and only 15% with the active form of treatment.

Dührssen, in advocating early interference, says that it is not intervention which is serious in eclampsia, but the eclampsia itself; and that, by rapidly evacuating the uterus, one saves the mother from pulmonary and other complications. When labour has been induced and proved unsuccessful, he declares that it is always when the surgeon has interfered too late.

Dührssen's most persistent and formidable opponent, Charpentier, has a considerable following in this country, and in France; but, from a survey of the literature of the past ten years and from the reports of the International Congress at Geneva, in 1896, one is forced to conclude that the majority of writers, regarding the presence of pregnancy as a vital factor in the production of eclampsia, hold that the termination of pregnancy in all cases is desirable, and that in serious ones the sooner this is accomplished the better. Is the gravity of eclampsia in direct relation to the number of the fits? It is not the number of fits, as we saw before; but a few fits with profound coma without intervals of consciousness between, which add to the gravity of the outlook. Fits may be more frequent, and even more violent in appearance, but consciousness may be completely regained during the intervals. Dührssen holds that the fits stop in 89% of the cases, and Charpentier that they do so in 28.3%. Charpentier contributed a brilliant article on the treatment of eclampsia, in 1892, in which he subjected the statistics of Dührssen and the German school to a most searching examination. After a minute and able analysis of 454 cases, he utterly condemned accouchement forcé in every form; and came to the following conclusions regarding obstetrical treatment of the disease:

1st. It is advisable to wait until labour begins spontaneously, and allow it to terminate naturally whenever possible.

2nd. Induced labour should be reserved for those exceptional cases in which medicinal treatment has completely failed.

3rd. Interference should be delayed until the cervix is dilated, or dilatable, so as to avoid danger to the mother.

4th. In eclampsia Caesarian section, manual dilatation of the cervix, and especially deep incisions of the cervix, are absolutely unjustifiable.

I am quite in accord with those more moderate observers who are of the opinion that it is only when the other means which we have at our disposal have failed, that labour should be induced. It is certainly obnoxious to every practitioner to stand by and carry out the expectant plan of treatment to the point advocated by Winckel, Herman, and others; for by so doing he may run the risk, a terrible one in his eyes, of seeing his patient die undelivered. If eclampsia comes on during pregnancy, then I think that it is better at first to leave the uterus to take care of itself, all attention being directed to the elimination of the poison and the control of the convulsions. Should these means fail, then we should have recourse to emptying the uterus in as rapid a manner as can be done with safety - care being taken to accomplish our intention as gently as possible, under complete anaesthesia, and so as to protect the patient from shock and the onset of other complications.

We may further note that Charpentier, Winckel, and the other advocates of the expectant treatment have been blamed for acting in the interests of the mother only, and of ignoring the child. But, in a large number of cases, it is only the interests of the mother which are at stake; and Charpentier, taking the German statistics, showed that nearly 21% of the children died before delivery, and 15.2% during delivery - in all 36.12%. To these numbers fall to be added the considerable percentage of the survivors who may die a few hours, or days, after birth.

INDUCTION OF LABOUR.

If we have resolved to induce labour, what is the best method to adopt to accomplish it? Here we have a variety of methods, each of which has its special advocates and followers. I shall now describe these different methods, their respective values, and the cases which demand their application.

Abdominal Caesarian Section.

In 1878, Halbertsma first performed Caesarian section in eclampsia. Since that time, it has been carried out in a considerable number of cases. Hilmann, in 1900, collected 40 cases, with a mortality of 52% - of 41 children 18 died, and 23 survived. As this operation was generally carried out in desperate cases after other methods had failed, as, for example, where a hard and rigid os had resisted all attempts at dilatation: this may explain the terribly high mortality. Those who favour this operation in place of other violent methods at delivery, say that with it there is less shock; that it is much quicker and more scientific; and that it gives the child a better chance of living. Herzfield holds that Caesarian section is the proper treatment for eclampsia with anuria occurring early in labour in a primipara, where the ureters are presumably dilated. Oldhausen, out of his last 250 cases, has done Caesarian section three times - two of the mothers and all the children survived.

The bulk of observers, however, regard this operation as quite unjustifiable - unless in very severe cases, as when eclampsia occurs in a woman with a markedly contracted pelvis, or where there is some obstruction; while many hold that the only time this operation ought to be performed is in these exceedingly rare cases where the patient has just died during an attack, and the foetus is still alive, or where the woman is in articulo mortis.

Vaginal Caesarian Section.

Although this operation was introduced by Accouche, it was Dührssen who first employed it in eclampsia; and it is principally owing to his work that the operation has now become popular amongst certain obstetricians. The operation consists in the delivery of the child through the vagina, artificial dilatation of the cervix being brought about by deep incisions into the intra-vaginal portion of the cervix, and reaching as far as the insertion of the vagina into the cervix. When necessary, deep incisions into the vagina, vulva, and perineum should be made. It is interesting to note that De Paul, many years ago, held that where the child was living and where fresh attacks being imminent, its life was in danger, and where the os was rigid, incisions into its edges would be justifiable.

The advocates of Dührssen's operation hold that, even in the most difficult cases, the uterus can be emptied within ten minutes, and most cases in five minutes, while from twenty to thirty minutes would be sufficient to close the wound by suture. There is no shock; and Dührssen strongly insists on the patient being deeply under the anaesthetic. Haemorrhage is easily controlled, the pulling on the uterus being usually sufficient; while, as there is little traumatism, the clean-cut wounds are not long in uniting. Dührssen advocates version instead of forceps. He declares that where his operation is practised early, with rigorous antisepsis and regular supervision of the perineum, the prognosis is the same as with spontaneous delivery. In the advocacy of his method, Dührssen came into sharp conflict with those who favour the use of steel dilators, which he condemned in no hesitating manner. The result has been the formation of two camps - the one with a great German following, the other getting considerable support from English surgeons and those who do not believe in the heroic obstetric treatment of this disease. The supporters of the latter school hold that Dührssen's method exposes the patient to severe haemorrhage, on account of the foetal parts tearing by extending the incisions, and that there is danger of irregular cicatrization, with its attendant risks, in the following confinements. As regards the rapidity with which the uterus can be emptied, which is one of the chief points which Dührssen lays stress on, the operation can be performed in from four to ten minutes at the outside. Dilators take at least thirty minutes; but, in the bulk of cases (unless we follow the teaching of Dührssen, who holds that the operation must be begun immediately after the first fit), there is no necessity for such hasty and urgent delivery. As regards the absence of shock, in the great majority of cases, - where the cervix is forcibly dilated, provided the patient is deeply anaesthetised, - there should be little shock, although some observers have reported profound shock in certain cases where the patient was apparently

deeply under the influence of the anaesthetic.

I believe that there are cases, few and far between, where the os is so rigid, and so resistant to all conservative methods to dilate it, and where mechanical dilators, such as Bossi's, however carefully employed, could only result in extensive lacerations, where the proper method to adopt, should expert assistance be at hand, would be vaginal Caesarian section. In the hands of skilled operators - and I think that most will admit that the operation requires considerable surgical skill and experience - it is certainly a more scientific method than forcible dilatation by special instruments.

In the hands, then, of the general practitioner where the os is rigid, and where skilled assistance is not available, expectant treatment will certainly have better results than forcible dilatation; and as for Dührssen's method, however good the results may have been in the skilled hands of its brilliant originator and his disciples, were it to be adopted (even in a modified form) by every practitioner when called in to a case of eclampsia, I am much afraid that the mortality in this disease, high as it is already, would be considerably greater.

Dilatation of the Cervix.

There are various ways of dilating the cervix. One way, for example, is to begin the dilatation with Hegar's dilators, and with the fingers, till the os is large enough to admit Barnes's or De Ribes's bag - the latter by preference. This is introduced into the lower uterine segment, and pumped full with sterile water or saline solution. In a few hours, its presence usually stimulates uterine contractions, which cause dilatation of the cervix with expulsion of the bag. Forceps may be applied, or version performed, or the case left to nature.

Manual Dilatation. - This method may be begun when the os is large enough to insert the index finger. Where rapid delivery is required and the os is firmly closed, it is generally better to use steel dilators to begin with, - as they dilate more easily and more quickly, - and then complete dilatation by means of the fingers. The patient must be fully anaesthetised; where the os is soft and patulous, it is astonishing how quickly dilatation can be effected in this way. It should not be used where the os is very rigid, as it is apt to cause lacerations.

Bossi's Dilator. - Of the metal dilators, the favourites are Bossi's four-bladed instrument (which dilates with a screw), Trommer's (which has eight blades), or some modification of these. Since Bossi introduced his instrument, in 1890, many writers have used it, or some modification, with varying results. Leopold, who is a great advocate of this instrument, holds that it should be in the bag of every practitioner, and that its use, if care and caution are exercised, is perfectly safe. The shanks of this instrument are ~~closed~~ ^{constructed} in such a manner that they do not overdistend the vagina, even when the cervix is fully dilated. The points of the instrument should be so thin as to be able to enter the undilated cervix. Bossi advises covering the points with rubber drainage tubes; and, when the os is slightly open, the points may be thickened with metal plates, which will cause less chance of the instruments cutting. In dilating, which invariably sets up uterine contractions, care should be taken to do it as carefully and slowly as possible, allowing one or more pains to intervene between each turn of the screw. Bossi advises the use of the forceps rather than the performance of version; and this should be done immediately dilatation is completed, as the os tends to contract again. Where version is performed, there is often difficulty with the after-coming head - owing to the contraction of the os.

Many well-known observers, prominent among whom is Dührssen, condemn its use. They hold that the very points which constitute its value in the eyes of those who advocate its use (e.g., the ease with which it can dilate the cervix, with practically no fatigue to the operator) is where its great danger lies, as it is very apt to cause extensive lacerations with their contingent risk of shock, haemorrhage, and sepsis. Bossi holds, however, that this can be prevented altogether if care is taken; and he advises the precaution of keeping the middle and index fingers in the cervix while dilatation is going on. It can be used in the vast majority of cases; but it is

necessary to again add that in cases of very rigid os, the general practitioner would be better to rely on expectant treatment rather than run the risk of the extensive tearing which may result from brutal and forcible dilatation.

Many surgeons also have lately warned us against forcible dilatation of the os in the early months of pregnancy, that is, about the seventh month, as they hold that lacerations are more likely to occur at that time. Some advise dilatation to a certain degree with Bossi's dilator, and then complete dilatation by means of multiple incisions. Many authors have drawn attention to the great danger in primiparae from careless dilatation of the os by hand or instrument. In eclampsia great care should be taken to effect complete dilatation, especially before version, as after convulsions have taken place, constriction frequently occurs.

When one has made up one's mind in grave cases of eclampsia in pregnancy to bring on labour, the following is the method I recommend: I would begin with injection of morphia ($\frac{1}{2}$ gr.) - this not only acts as a sedative, but helps dilatation of the os, and prevents shock. Under deep chloroform anaesthesia, the os should then be dilated to the size of a florin with Bossi's dilator, care being taken not to rupture the membranes. Complete dilatation with the fingers may now be effected; when the case may be left for a little time to see whether uterine contractions are going on. The labour can then be completed as quickly as possible by version, or by means of the forceps.

PERSONAL OBSERVATIONS.

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Case 1.

Mrs. C., aged 28, 11-para; robust and inclined to obesity.

On November 20th, 1900, I was called in to see the patient, as she had been ailing for a few days; and her husband had noticed that his wife from being a bright good-natured woman had altered strangely in manner, becoming morose, forgetful, very fretful, and subject to strange outbursts of temper. She had been complaining for the past day or two of severe headache - this had been pretty constant and confined to the occipital region, occasionally shooting down the ~~neck~~ **back**. She was feeling very ill, had been restless for the past night or two, sleeping badly, and much troubled with nightmare. She "saw double". She had also a feeling of giddiness, which brought on attacks of nausea, with the vomiting of bilious material. In fact, she assigned her whole trouble to a very "bad attack of the bile".

When I saw her, the bowels had not moved for three days, and during that time she had passed very little urine. Her face was puffy and oedematous; the oedema was very conspicuous in the lower limbs, which pitted deeply on pressure, the hands and ~~forearms~~ **arms** being also very much swollen. Her pulse was rapid, full, and bounding, but the temperature was normal. Just before my arrival, she had had a severe attack of vomiting - prior to which latter she had experienced a "terrible pain at the pit of the stomach". This pain she described as agonising, and informed me that it made her double up and cry aloud; it was, however, considerably relieved by the vomiting.

I gave her a large dose of jalap and calomel, and also prescribed a rectal injection of hot water and soap. I ordered her to bed, and ~~heaped~~ **placed** the bedclothes over her, placing hot flasks round about her to encourage sweating. I called back in half an hour, and gave her potassium bromide (40 grs.) and phenacetin (10 grs.) to allay her restlessness and the intolerable headache. I also obtained a small quantity of her urine, which on examination I found solid with albumin and to contain blood. I then left her.

I had not been away for more than one and a half hours, when I received an urgent message to return, as the patient had taken a convulsion. On arrival, I found that she had taken two fits within half an hour - during the first of which her bowels had moved pretty freely. Her temperature was 101.8° F. She was very dazed and stupid-looking, and appeared to have no conception of what had happened. I gave her, by the mouth, 20 grains of chloral and ~~1/2~~ **1/4** of potassium bromide, and washed out the large bowel with hot water. Shortly afterwards, she had another fit, and in half an hour another, to be followed five minutes later by yet another - the fifth from the onset of the disease. I roused her sufficiently after the fit to administer 15 grains of chloral and 30 grains of potassium bromide. I again took her temperature, and found that it was 102.5 F.

She was expecting her confinement at any time, so I resolved to bring on labour, and sent off for my chief, Dr. Morrison, to bring a dilator. I put her under chloroform, and examined her. As the tissues were soft and dilatable, I resolved to dilate the cervix with the fingers. I began with one finger, and was soon able to insert the other two. At this stage she had two severe fits in rapid succession, followed by profound coma. There was a good deal of cyanosis present, and slight oedema of the lungs. The temperature was now 102.8 F.; and the pulse 132, slightly irregular, and of small volume.

On examining her after these two fits, I found to my surprise that the os had become quite rigid, and that it resisted all my efforts to dilate it. Dr. Morrison arrived at this time, and we put the patient more deeply under chloroform, dilated with a two-bladed dilator of the ancient pattern, and soon had the os dilated to the size of a crown. The membranes were then ruptured, and an enormous quantity of liquor amni escaped. We found the head to engage by pressure over the fundus, applied the forceps to the head, and quickly delivered - the patient being under the anaesthetic the whole time. While we were dilating with the dilator and while delivery was being effected with the forceps, she had only one slight convulsion - the

dilatation here, then, instead of inducing convulsions as is often the case, actually had the reverse effect. From the time I commenced digital dilatation, when she had two fits in rapid succession, till the completion of labour, one and a half hours had elapsed. We had considerable difficulty in bringing ~~found~~ the child, which was a very large male, but we ultimately managed it, notwithstanding that the mother had had eight fits. The child got on very well, thriving immensely on cow's milk; and it is now five years of age, and in perfect health. The placenta came away very easily, but there was a good deal of haemorrhage after it; still, we did not feel uneasy on that account, and took no steps to control it for a little, as the pulse had considerably improved during labour - becoming ~~farmer~~, more regular, and of much better volume.

Half an hour after delivery, she had another fit - the most severe and prolonged that she had as yet taken (delivery took place at 3 p.m.). During the afternoon and evening, she took eight fits in all. I saw her at 5 p.m., just after she had taken a fit - she was comatose, and had a very weak pulse. I gave her a large saline injection, with a tablespoonful of brandy, into the bowel. She had been getting 40 grains of chloral and 30 grains of potassium bromide bt the bowel, every ~~two~~ and a half hours. Later on, we administered nutrient enemata of beef-tea and milk. I took her temperature before leaving; and, notwithstanding the four fits ~~she~~ had taken since her delivery, her temperature was down to 100.8°F. I saw her again at 10 p.m.: she had taken three fits since my last visit, but they were less severe than heretofore. Her pulse was better, and her temperature was the same as at the last time of registration - viz., 100.8°F. At 11.30 that night, she had another fit, and yet another at 1.30 in the morning - both being very mild. There were no more convulsions after this.

The patient in all had 17 fits - 8 before delivery, and 9 after; 7 of these fits were comparatively mild. The ones during labour were mostly mild, the severest occurring immediately after labour had been terminated. The day after she was delivered, there was still an abundance of albumin in the urine - the urine being, however, considerably increased in quantity. Albumin was very much less next day, and it progressively decreased till a fortnight after, when it disappeared altogether.

Interesting points in this case were - the fact that the fits became less severe and less frequent during labour, while she had as many fits after the uterus had been evacuated as before labour commenced. *Also the fact of child surviving after mother had eight convulsions.*

Case II.

Chorea in a pregnant woman, with the occurrence of eclampsia during labour.

Mrs. P., aged 20, the wife of a miner, and a primipara.

The history in this case is one of very great interest. When I saw ~~hats~~ patient first, she was 2½ months pregnant. She was tall, thin, and slightly anaemic, and also of the above-mentioned age. She had been married about two months. She complained then of severe morning sickness. This gradually became worse, in spite of my exhibition of the usual remedies for its relief. About the fourth month it disappeared suddenly. On two or three occasions during the ~~third~~ and fourth months, on examining her urine I found albumin present, although not in great quantity. From the fourth to the 5½ months, she then made excellent progress - rapidly regaining the ground she had lost during the earlier months. In this period I examined her urine twice, but there was no albumin present.

When she was 5½ months pregnant, her mother asked me to see her - as she had noticed her daughter's manner changing, also that she was continually "making faces" and moving her arms in a peculiar manner. When I saw her, I noticed at once an alteration in her temperament: she was more emotional, was stupid, did not seem to comprehend a question put to her, and choreic movements in the arms and facial muscles were well-marked. Hitherto the patient had been bright, smart, and good-tempered; but she now became dull, querulous, and depressed. The movements, slight in character to begin with, gradually became worse - involving the muscles of the legs and shoulders.

About the seventh month, convulsive movements and jerking of the body developed; and it was with great difficulty that she could be kept in bed and prevented from doing herself some serious injury. She became very restless, and slept very little; while, later on, she became almost maniacal. All this time I had been treating her with arsenic, iron, chloral, bromide, and all of the drugs advocated for the control of this disease. As none of them did any good, I stopped all medication. We had great difficulty in feeding her; and from this, and the loss of sleep, and excessive movements she was greatly debilitated. I now thought of bringing on labour; but, as the foetal movements were good and I considered the mother's condition such as to be able to stand the inroads of disease for a little while, I put off for a day or two. I may say that during this attack of chorea I had examined the urine several times, and on all occasions found albumin present.

At the 7½ month, a week after I had stopped all medication, the chorea suddenly disappeared - the patient having a good sleep, and able next day to take her food much better. Had this sudden cessation of the disease taken place during the administration of one of the many remedies I had tried, I am afraid I would have lauded it to the skies as a specific for chorea. The latter was entirely absent until labour came on at the full term, when slight movements returned with the first pain.

The labour commenced at 6 a.m., and I was sent for about 10 o'clock. On arrival I was informed that she had had two convulsions, quite different from the movements she had had before; and I now noticed that her appearance was strongly suggestive of a previous fit. She was semi-comatose; and, on shaking her up, I found her quite dazed and bewildered-looking. On examining her, I found the breech presenting at the perineum. Her pain were strong; and in ten minutes a living child was born: it was not a particularly strong-looking infant, but thrived well, and in a year's time appeared to be in perfect health. There was a good deal of haemorrhage, but it was easily checked.

The patient made a good recovery. I took her temperature when she was in a semi-comatose condition and half an hour after delivery, when it was 100.6° and 100.2° F. respectively at these times. Slight choreic movements were witnessed for two days after delivery, but after that they disappeared entirely. There was no return of the chorea in her next pregnancy, although after her confinement she developed acute melancholia and had to be removed to the asylum.

The interesting points in this case - and there are several - were the severe sickness in the earlier months, the occurrence of albuminuria, and the disappearance of the former and the latter simultaneously. As regards this and the causation of chorea in pregnancy, we know that a certain number of these cases may be caused by rheumatism - in this case there was no history of rheumatism, of scarlet fever, or of any other febrile disease. The patient had been quite healthy up to this illness, and she had never had any previous attack of chorea. Might not the theory of toxæmia of pregnancy - which would account for the appearance of albuminuria and severe sickness - be also the case of the chorea, and, lastly, the two eclamptic attacks? In toxic poisoning we find very often that it is the nervous system which is the chief sufferer - polyneuritis of pregnancy, etc., etc. The sudden cessation of chorea would point to the establishment of compensation - either the defensive organs, which would have to do with the destruction of poisons in the blood, or their elimination from the body, working more efficiently. If the toxins are not the direct cause of the chorea, they have a considerable influence in predisposing the patient to attack.

Again, the patient went to full term, and was delivered of a living child. Chorea in pregnancy (see Hirschl, - Monats. f. Geburt. und Gyn., Jan., 1903) brings on abortion in about 16%-20%; while the mortality for the foetus is usually put at 40%, and for the mother at various figures - e.g., 30% (Barnes), 27% (Spiegelberg), and 17.5% (Buist).

The reappearance of albumin in the urine, with the development of chorea, I regard as likewise of considerable interest.

It is a common thing in cases of multiple neuritis in pregnancy to observe an association with hyperemesis. It is particularly

interesting in this case of chorea to find it preceded by hyperemesis, and followed by eclampsia during labour. The latter two conditions are regarded as typical toxæmic manifestations. May not chorea, then, also be a nervous manifestation of auto-intoxication produced by the circulation of poisonous substances which ought to have been eliminated from the maternal organism? As I have already pointed out, certain poisons circulating in the blood seem to have a marked affinity for the nervous system - as seen in poisoning from alcohol, infectious diseases, etc. I do not believe, however, that every case of chorea in pregnancy is produced by toxic poisoning; for another case of chorea gravidarum I had come on after the patient had received a great fright, and it recurred in a later pregnancy - owing to great anxiety and worry, caused by the serious illness of one of her children. This patient had neither rheumatism nor chorea before marriage.

Case III.

Post-partum Eclampsia. Mrs. D., farmer's wife, resident in Douglas:

On February 8th, 1903, I was called to attend this patient in her confinement. She was 43 years of age; but, on account of the hard work she had all her life been subjected to, looked ten years older. Her husband had a small farm. She had previously had four children, - three of whom were alive, - and two miscarriages. There was a history of syphilis. She was 29 years of age when she married; and she gave birth to her first child two years later. She had never had any difficulty at her confinements, - "getting off" very easily, as she put it, - but in the earlier months of pregnancy she was "very bad with sickness" during her first and third pregnancies; while all her life she had been a martyr to attack of "bile".

The patient consulted me, two months before her confinement, for obstinate constipation, which had greatly troubled her for two or three months, and was blamed for her "bilious turns" and severe headaches. I was struck with her appearance: she was looking considerably older than her years, she was thin, while the rest of her face was deeply pigmented in patches, the remainder having a dirty yellowish-gray appearance - the latter being, she told me, her usual complexion during the last ten or twelve years. She had, however, never had the pigmentation so so marked a degree before. At her confinement I was particularly struck by the pigmentation on her body - she looked as if she had Addison's disease. Indeed, I never saw a case where it was so marked.

I examined her urine at this time, and again within a fortnight of her confinement - on both occasions I found a slight trace of albumin, no tube-casts, but the urine reduced Fehling's solution. I told her to keep to a milk diet, prescribed tabloids of calomel (2 grs.) to be taken at night, and a simple saline in the morning.

When summoned (on the above-mentioned date) to her confinement, I found on my arrival that the child had just been born. Indeed, she appears to have had a very easy labour. Fifteen minutes later, I expressed the placenta, and with it numerous clots. As there was a good deal of hæmorrhage and her pulse was rapid, I gave her a dose of ergot and kneaded the uterus well. I stayed with her for nearly two hours, as I did not wish to risk being called back six miles from my residence to hers. When I left her, about 10 p.m., the pulse was slower and better; and, except for headache which had troubled her more or less all day and slight after-pains, she felt quite comfortable.

I was sent for in a great hurry about 6 o'clock next morning - just ten hours after her confinement. When I got there, I found that the patient had taken three fits in rapid succession; and the nurse, on seeing them and on account of the curious state of mind her patient was in after being roused from the first fit, had sent for me. When the nurse put the baby into the patient's arms, the latter pitched it away saying that it was not hers. She had been very restless since I left the night before, continually complaining to the nurse of severe headache coming on in spasms, and which, to use her own words, "left her blind"; also of dizziness, ringing in the ears, and - an hour or so before the first fit came on - severe pain at the "pit of the stomach", accompanied shortly after by the

vomiting of "yellow stuff like the yolk of an egg." Her temper all the time had been most irritable, but the nurse paid little heed to this as her patient was naturally of a peculiar disposition.

When I examined her, I found that she was just coming out of a comatose condition. She was lying on her back, with the face of dusky hue, the pupils widely dilated, and the expression of the countenance very dazed and bewildered. The pulse was 118, and of fairly good volume; the temperature did not exceed 100°F. The face was not swollen, and there was no swelling or oedema anywhere. I drew off the urine, and examined it when I got home; it amounted to about twelve ounces, and gave with cold nitric acid only a faint trace of albumin, but reduced Fehling's solution. The sugar could not have originated from absorption from the breasts, as they contain practically no milk during pregnancy or at any time during the puerperium. This reaction disappeared four days after, but I could still detect a trace of albumin in the urine for eight days. The amount of urine secreted during the attacks - so far as I could judge, and I drew it off frequently - was not greatly diminished. A good deal of it was lost when her bowels moved with the enemata, but it was certainly not much less than normal.

I always carried in my bag a bottle of bromide of potassium and chloral, as I find it very useful, combined with opium, in the early stages of labour in primiparae. I gave her $\frac{1}{2}$ drachm of potassium bromide and 25 grains of chloral, as well as two 2-gr. tablets of calomel. At the same time I gave her a large enema of hot water and soap, and a hypodermic injection of morphia. Ten minutes later, she had another convulsion for three minutes, and during which her bowels moved. After forty-five minutes, I gave her 15 grains of chloral and 30 grains of potassium bromide, as well as another large enema of hot saline solution. Her temperature was 101.6°F. I waited with her for two and a half hours, during which time she had six convulsions - four of them being in rapid succession. After these, I again took her temperature, and found it to be 103.2°F. I had now to leave her, but before doing so gave her $\frac{1}{2}$ grain of morphia hypodermically, and 40 grains of chloral by the rectum.

I returned to the patient in two hours, when I found that she was very much worse, and that for one hour immediately after my departure she had only two fits; but after that time she had gone from one fit into another - the nurse estimating that she had five or six, at the least, in a very short time. Her condition was now very serious indeed - she was absolutely comatose, her pulse was small and 156, and her temperature was 103.4°F. I gave her a hypodermic injection of strychnine and morphia, and a saline infusion into the abdominal tissues. I followed this up with copious enemata of hot water, to which was added the acetate and the bicarbonate of potash, allowing the fluid to flow slowly into the bowel - the hips being well raised all the time. The salts of potash were added for their diuretic action. Half an hour afterwards, as she had no fit, as the coma was not so profound, and as her pulse had improved, - I made up my mind to try the hot pack; so I had a blanket wrung out of hot water, rolled the patient well in it, and arranged the bed-clothes around her. She gradually improved, but had another fit at 12 o'clock, which was of a much milder type than those she had previously experienced. I took her temperature ten minutes before this fit came on, when it was 103° - after the convulsion 102.8°F. During the next twelve hours, she had in all four fits - each one becoming less severe. I had stopped the administration of chloral and morphia, preferring to rely upon the subcutaneous saline injections and enemata of a solution of the two above-mentioned salts of potassium. I gave two of each during the next twelve hours, and gave the patient hot beef-tea and brandy. During the rest of the afternoon and night, she had only two fits; while one appeared, the last, about 2 o'clock next morning. All that day the district nurse had been in attendance; and I gave her instructions to take the temperature every two hours, and also after each fit. The temperature gradually fell, and was practically uninfluenced by the fits - rising only .2 and .6 degrees after two of them; and when I saw her in the morning it was 100.2°, while at midday it was 100°, and at night 99.6°F.

The patient's condition was now nearly normal, although she had still the dull and dazed expression of countenance. She had also a peculiar hesitancy in her speech, which was not present before her illness, and she was also slightly deaf. Loss of memory was a prominent

feature of this case. Sixteen days after her confinement, I found on questioning her that she had forgotten everything that had happened six or eight weeks previous to her confinement - she had no remembrance of consulting me at that time, nor of a visit from friends who stayed with her ^{for a} fortnight before her confinement, and no recollection of her labour and subsequent serious illness. All that time had been completely blotted out from her memory, although her recollection of everything up to the period in question appeared to be perfect.

For a few days after the subsidence of the fits, I kept her on a milk diet and barley-water; while I carefully regulated her bowels with calomel and enemata of hot water and soap.

On examining her after confinement, I was struck with the amount of pigmentation present - not only on the face, but in large patches on the breasts, from the axilla to the waist, and round the abdomen and loins. In fact, as I said before, it looked like a case of Addison's disease.

A month afterwards, I examined the urine, but found no traces of either albumin or sugar. The patient made a perfect recovery.

Case IV.

Mrs. R., aged 22, the wife of a farmer: primipara. Douglas.

On February 28th, 1902, I was called out to see this patient; and when I arrived, about 5.30 p.m., I found that she had taken a fit three-quarters of an hour before. I found her in a slightly dazed condition, but conscious and perfectly able to answer my questions sensibly. She told me that she had had been ailing for a few days previously; but, as she was expecting her confinement at any time, she did not pay much attention to her condition. She had been troubled for a fortnight with swelling of the feet and legs; and, during the last day or two, she noticed her eyes slightly puffed when she rose in the morning - this disappeared as the day wore on. A week before, she noticed that she became worse after she had been working very hard for two days - washing and preparing, as she said, for her confinement. She had also had slight attacks of vomiting and headache. During the early months of pregnancy she appears to have enjoyed the best of health. The night before I saw her, however, she felt really ill; the headache had become severe, and she could not see very well. Her mother told her that she had caught a chill, put her to bed early, and gave her a hot gruel which, however, the patient vomited an hour later. The headache had got gradually worse till her condition culminated in a convulsion; this greatly alarmed everyone, and was the cause of my being sent for.

The patient is a robust, stout, healthy-looking girl. Her face is slightly puffed, especially about the eyes, while her legs are very oedematous and pit deeply on pressure. She had passed no urine for twenty-four hours, so I drew it off with a catheter; there was about eight ounces of it; examined at home, it was seen to be absolutely solid with albumin, and to contain tube-casts in great variety.

I had not been in the house for more than fifteen minutes before she took another fit. When she came out of it, I resolved, as her pulse was full and bounding and her condition such as to justify, in my opinion, venesection, - to bleed her; and this I did to the extent of taking about twelve ounces from her arm. When she was conscious, I gave her six grains of calomel and one-third of a grain of morphia, and began to wash out the bowel with copious enemata of hot water and soap. I may add here that she had taken a good dose of castor oil, two days before, which had moved her bowels thoroughly. In half an hour, as she was very restless, I gave her twenty grains of chloral by the mouth; and this she retained. I waited with her for two hours, during which time she had no other fit. Before I left her, I gave her 15 grains of chloral, and allowed a large quantity of hot saline solution to flow into the large bowel. I took her temperature after the second convulsion, and found it to be 99.8°F. I had made a vaginal examination of the patient when I arrived at 5.30, when I found the os large enough to admit my finger. During this time I was with her, she had several sharp pains; but, on examining the os when I left at 8 o'clock, I found the condition of the os much the same. I questioned her as to movement, and she told me that the movements of

the child had become much feebler - on examination it was difficult to detect them, but the heart sounds were perfectly audible.

I returned to the patient in the afternoon; and the nurse told me that she had been very quiet for four or five hours after my departure, but that she was beginning to get restless again. I gave her an injection of one-third of a grain of morphia, and 15 grains of chloral by the mouth, as well as another large enema of saline solution. I then examined her, and found that the os was about the size of a crown-piece and dilatable. I then resolved to allow labour to go on without interfering in any way; but I had not been long in the house before she had another fit - the worst and most prolonged she had ever experienced. As the os appeared soft and dilatable, I put her under chloroform and dilated the part slowly with my fingers. She had several good pain during the process of dilatation, which helped her considerably; and I had no great difficulty in effecting dilatation, when I delivered her with forceps in the usual way. During the manipulations she had a severe convulsion, but this was the last. The child was dead. The patient made a good recovery.

This case presented little of interest, unless it be the fact of there being only four fits, - which, however, proved sufficient to destroy the child, - and the manner in which the patient reacted to the treatment adopted.

Case V.

Eclampsia in an Epileptic. Mrs. T., aged 25, primipara, and resident in Newmains.

On April 12th, 1904, I received an urgent call to go down and see this patient, who appears to have fallen down in a fit whilst conversing with a neighbour. On my arrival, I found that she had practically recovered from her fit. I found her temperature normal, and that the pulse was 86 per minute. There were no signs of oedema anywhere. I got her to send up a specimen of her urine, which on examination showed no traces of albumin or sugar. The woman who was with the patient when she took the fit told me that she screamed out, and then fell suddenly to the ground. On questioning the patient next day, I found that she had been in the habit of taking fits periodically since she was thirteen or fourteen years of age; and I obtained from her the following history of her pregnancy: She had taken an epileptic fit a month after conception (about her menstrual period), another when 4 or 4½ months pregnant, and she was now 5½ months advanced in that condition. That is to say, she had taken three fits since she became with child. Before each fit she always feels sudden giddiness and a tingling in her right arm. During the early months of pregnancy, she had been under the care of a physician in ~~Town~~, as she had severe vomiting lasting for a considerable time. In spite of all his treatment, it became so bad that the practitioner in question told the patient that he was afraid he would require to bring on an abortion - however, this vomiting gradually became less, and as she was in fairly good health when I first saw her, she must have recruited her health considerably.

Towards the end of May, she came to see me, as she was alarmed about her legs swelling, and also because her face had been very puffy in the mornings for the last day or two. She was now nearly seven months pregnant. She also complained to me of considerable frontal headache, of feeling unwell, dull, "not like herself", and of wandering pain about the abdomen. I ordered her to go home to bed, gave her a powder of jalap (gr. xxvi) and calomel (gr. v), and told her to keep to a strictly milk diet.

About twelve hours afterwards, I received an urgent message to go to her, as she felt very ill. When I got down, I found that she had had a severe fit, and also that she had been very ill before it came on, complaining of severe headache, giddiness, and ringing in the ears. The abdominal pain, of which she had complained on her visit to me, appears to have become so much worse as to make her cry out. She had also had two or three severe attacks of vomiting. Her whole body was very much swollen - the limbs, both upper and lower, pitting

deeply on pressure. She had passed no urine for nearly a day, but her bowels had moved pretty freely an hour before in response to the powder administered. I now had a hot pack prepared; and, after washing out the bowel with hot water and soap and a little turpentine, put her into it. I then gave her 40 grains of chloral, beaten up with an egg, by the rectum: this she retained. Shortly afterwards, she had another convulsion, but not very severe, and the coma which followed it quickly disappeared. I drew off the urine, and found it very scanty (about six ounces), very highly coloured, and containing a large quantity of albumin and tube-casts. Her temperature was 100° F. I now gave her a large saline injection into the bowel; and, as she was quite conscious, 15 grains of chloral and 60 grains of potassium bromide by the mouth. I stayed with her for an hour - when she appeared to be very much better, her headache not so severe, no vomiting, and much less abdominal pain. Before leaving, I examined her per vaginam: there were no signs of labour, the os was tightly closed, and the child appeared to be alive. An hour later, I received another call; and on going down found that she had just had another convulsion which, however, was the mildest she had ever yet experienced. She was quite conscious when roused, and I got her to swallow 15 grains of chloral, and gave another saline enema. She had no return of the fits, and when I saw her next day she appeared to be very much better. I ordered her to stay in bed, keep to milk, and to take one of my 2-gr. calomel tabloids every night and a saline in the morning. She was also directed to take occasional hot baths, with the usual precautions against chills; and I gave her an occasional saline enema after a preliminary washing-out of the bowel with hot water and soap. She carried out my orders faithfully, and remained in good health.

On July 14th, I was sent for, as she was now in labour. I happened to be out at the time, and so did not see her for an hour and a half after. When I arrived, the district nurse - who was attending her - told me that the patient had had two fits. I had not seen the patient for a week - during which time she had not been so well, complaining of headaches, and occasional attacks of vomiting, while the swelling, which had almost disappeared from her legs and face, had become worse. On examining her, I was surprised to find the os well dilated. The presentation being transverse, I turned the child and soon effected its delivery; it was dead, and appeared to be about 5½ months. While I was expressing the placenta, the patient had another fit. I gave her a large enema of hot water, and her bowels moved shortly afterwards. I then thoroughly washed out the bowels with a soft tube and funnel, and forthwith injected a considerable quantity of saline solution therein. After that, I gave her 40 grains of chloral per rectum. During the next three hours, she took four fits; but each one was less severe than the preceding, and the coma was also less marked. On my next visit, as she was rather restless, I gave her bromide of potassium (60 grains), chloral (20 grains), and liq. morph. hydrochlor. (3i) by the mouth. Her temperature at this time was found to be 100.8° F. In an hour she had another fit - this was the mildest, and it proved to be the last. I saw her ten minutes after its occurred, and found that her temperature was now 101° F. After that the nurse took it frequently, and it soon came down to normal.

The patient made an excellent recovery. She passed increased quantities of urine every day; while the albumin, which was present in such quantity as to cause solidification on boiling during the time that the fits were in progress, gradually became less, and disappeared entirely fourteen days after delivery. On getting up on the eighth day, she gave a scream and fell on the floor in a fit. I was immediately summoned, and examined her carefully - with the result that I came to the conclusion that the fit was epileptic and not eclamptic. She passed a quantity of urine shortly after it; and, although albumin was present, it was only so in small quantity. Indeed, it had never disappeared since her confinement, although it was certainly progressively diminishing. I took her temperature: it was normal. The patient was in excellent health before it came on, and continued well after it: in fact, she assigned it to "one of her old turns".

This case was all the more interesting as epilepsy presents a striking resemblance to eclampsia; and here the latter developed in a patient tainted with the former dyscrasia. There was no doubt

whatever in this case about diagnosis - the history, signs, and symptoms all pointing to eclampsia. The only doubtful point is whether the last fit, which came on eight days after delivery, was eclamptic; but here everything pointed to an epileptic origin.

Other interesting features of this case were that the patient had three eclamptic seizures about the seventh month of pregnancy; that these disappeared without giving rise to miscarriage; that the child survived them; that the patient went on to 8½ months, when labour set in; the subsequent occurrence of eclampsia; and that at this time the seizures resulted in the death of the child.

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(NOTE.- All the remarks enclosed by brackets are my own.- D.D.I.)