(5) The head cannot be made to engage at all. Cæsarean Section is necessary. It is to be remembered that it is difficult to make a badly flexed head engage. If, therefore, there is any doubt, an examination under anæsthesia, and the employment of Munro Kerr's method is indicated.

The Examination of the Urine.

This should be tested every month for the first six months, every fortnight in the seventh and eighth month, and every week during the last month. The patient should be instructed to pass the morning specimen of urine in two portions after carefully swabbing the vulva from before backwards. A specimen of the second portion passed should be poured into a carefully cleaned bottle and sent to the doctor. If albumin is found, confirm by a catheter specimen, and take the blood pressure. If sugar is found a blood sugar estimation should be made. If between .09 and .18 milligrams per cent. it can be neglected. A history of venereal disease requires careful investigation.

Syphilis should be suspected if there is a history of miscarriages and premature still births. This should be confirmed by determination of the Wassermann reaction of *both* parents. If a patient is treated during the last three months, the baby will probably be healthy. Any purulent discharge should be investigated by taking slides and cultures.

ALBUMINURIA AND ECLAMPSIA.*

By ANDREW McALLISTER, F.R.C.S.,

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For the purposes of this paper I shall consider 179 cases of toxic albuminuria of pregnancy and 78 cases of eclampsia which have been treated in the Maternity Wing of Queen Mary's Hospital for the East End during the past twelve years. I have to thank my colleagues Mr. Carnac Rivett and Mr. Alan Brews for allowing me to include their cases in my own series, a step which is valuable since the treatment of these conditions has been uniform throughout the department. During the same period, I have had under my care 122 other cases of albuminuria associated with pregnancy, *e.g.*, cases of undoubted chronic nephritis and cases where the albuminuria has been so slight and transient as not to be considered by myself as being of toxic origin.

In the autumn of 1927 I embarked on a survey of all the cases admitted up to that time with a view to determining the frequency of recurrence in the same patient, for I much doubted that the then generally accepted view was true, namely that albuminuria of pregnancy rarely recurred. The result of my enquiry satisfied me that recurrence was the rule rather than the exception. About that time I became stimulated by the able writings of Dr. James Young of Edinburgh

* Being in somewhat modified form, a Lecture delivered for the Fellowship of Medicine in July 1933.

and Mr. G. F. Gibberd of London, which led me to continue my survey for another five years. The result has gone to prove that whereas the recurrence of eclampsia is very rare, that of toxic albuminuria should be anticipated in every subsequent pregnancy.

Table I shows the allocation of the cases. The untraced ones are those who did not come for review in response to the letter sent to them: they are, however, of value from the point of view of their past history and of their pregnancies and labours observed in the Hospital. I would point out that the 179 cases of albuminuria were with few exceptions, destined to have their labours in Hospital and had been attending the Antenatal Department whence they were admitted as soon as the toxæmia declared itself, whereas the 78 cases of Eclampsia with three exceptions, were admitted as emergencies from neighbouring practices and so must be considered as cases selected from the albuminuric cases of those practices. It is important to bear this point in mind, otherwise Eclampsia would appear to be far more common than it really is. Thus, the cases of albuminuria occurred in upwards of 10,000 women selected for delivery in Hospital whereas the cases of Eclampsia came from an East London and South of Essex population of nearly two millions. When we add to these figures the 122 cases of albuminuria not included in Table I we find that of 10,000 women delivered, 4 per cent. had albuminuria, a figure corresponding to the generally accepted figure of 3 to 5 per cent. throughout the country. I am inclined to think that the true incidence is much higher since it is probable that a great many deliveries take place in private houses where the albuminuria has been undetected or at best unrecorded.

When a woman suffers from toxic albuminuria of pregnancy she is faced with the following risks:—

- (1) Abortion, maceration and stillbirth;
- (2) Neonatal death of the baby, largely due to præmaturity;
- (3) Recurrence of the toxæmia in future pregnancies, perhaps with increasing severity;
- (4) Permanent damage to the kidneys;
- (5) Eclampsia.

It would be well to consider each of these in turn.

1. Stillbirth and Neonatal death.—A study of the stillbirth rate proves to my satisfaction the unwisdom of prolonging a pregnancy under expectant treatment in the hope of a living child if by so waiting the risks to the mother are increased. Table II shows the result to the foetus in the first toxæmic pregnancy. Only those cases where the pregnancy had reached the term of viability have been included, and too, cases of stillbirth which might fairly be attributed to some accidental cause such as forceps delivery are not considered here. Table II includes twins and one case of triplets, representing in all, 272 pregnancies. Of these no fewer than 35 per cent. were wasted. I would point out that of the babies born alive, roughly two-thirds of them were born prematurely, and of these, less than half were induced. The same applies to the stillbirths and macerated foetus. Thus we learn that in toxæmia, labour is premature in the great majority of cases, and from this we may draw a strong indication to induce

labour and so cut short the toxæmia, and if possible to induce at such time as will result in a living baby. It is disconcerting to have a foetus alive at say the 36th week and to have it born macerated at the 38th week; and this common experience offers a strong argument against prolonging expectant treatment especially when it is remembered that during such treatment the kidneys are exposed to the action of the toxins even though the degree of toxæmia may be lessened under treatment.

Table II shows other important points for remembrance:—

(1) That in toxic albuminuria of pregnancy, roughly 1 foetus in 4 is lost; in Eclampsia, 1 in 2.

(2) That in primigravidous albuminuria the foetal wastage is almost twice what it is in multigravidæ toxæmic for the first time. This may in part be explained by the fact that in primigravidæ the stress of labour is so much greater, and this stress is ill-borne by a premature foetus.

(3) That in multigravidous Eclampsia the risk to the foetus is almost twice what it is in primigravidous cases. This is not surprising when it is remembered that many of the multigravidæ had had previous toxæmic pregnancies, and if at this stage we may fairly anticipate and assume that in those toxæmic pregnancies the kidneys received permanent damage, then we may assume that in the pregnancy under consideration the foetus was exposed to the toxæmia for a prolonged period prior to being called upon to withstand the stress of the eclamptic convulsions. In the case of primigravidæ it is different, for in them the disease is generally more acute and of shorter duration and the onset of Eclampsia more sudden.

These points seem to prove that if there be an added maternal risk in prolonging a pregnancy even under treatment, in the hope of having a living baby and one which will live, the chance of a successful issue from the foetal point of view is too poor to justify our taking such a risk.

2. The Risk of Recurrence in Future Pregnancies.—In order to gain some idea as to the risk and the frequency of recurrence, I have studied the subsequent histories of traced cases up to the end of 1930. I have omitted the cases of 1931 and 1932 on the ground that the time might be too short to allow of a fair proportion becoming pregnant again. Thus out of 78 primigravidæ reviewed 16 became pregnant again. Eleven of these showed recurrence of toxæmia and two of them died after their second confinement, one ten days after and the other three weeks after. The postmortem findings in both cases were those of pregnancy kidney. Thus there was a recurrence rate of 68.75 per cent. in these women which compares with the figure of 68 per cent. reported by my friend Mr. G. F. Gibberd from his own series. In some of these 11 women the toxæmia recurred on more than one occasion, for between them they had 24 future pregnancies. Of these, 9 were normal and 12 were definitely toxic and there were 3 miscarriages. That is to say that 50 per cent. of these pregnancies were associated with toxæmia, or if we may include the miscarriages in this category, 62.5 per cent. It is of interest to note that only 16 of the 78 primigravidæ became pregnant again, the time of review varying from 10 to 2 years after their confinement. Of the others, 2 had separated from their husbands, 3 admitted the practise of contraception whilst all the others denied it. On this, could we be justified in assuming that toxæmia of pregnancy may in some way contribute to relative sterility?

Of the 44 multigravidæ reviewed for the same purpose, no fewer than 22 gave a history suggestive of former toxæmia if we may include some of the miscarriages as due to this cause. They had had between them 107 previous pregnancies of which 73 were assumed to be normal, 15 were frankly toxic, and there were 19 miscarriages. Thus, 31.7 per cent. of their previous pregnancies were toxæmic if we may include the miscarriages under this heading. Moreover, of these 19 miscarriages no fewer than 12 preceded the pregnancy first known to be toxæmic. The later history of these multigravidæ is that 9 of them became pregnant again and in 4 there was a recurrence. These 4 had 12 pregnancies of which 10 are known to have been toxæmic and there were 2 miscarriages.

The Risk of Eclampsia.

In my earlier years I was accustomed to see labour induced in cases of albuminuria in order to prevent Eclampsia. When the opportunity came to study a greater amount of clinical material I formed the opinion, and still hold it, that the risk of the supervention of Eclampsia in any given toxæmic pregnancy is extremely small. The reason for this is doubtless that where the toxæmia has become extreme the chance of premature labour is infinitely greater than the chance of Eclampsia. Thus, in my series only one primigravida and one multigravida developed Eclampsia whilst under observation, yet I am not prepared to attribute this low incidence to the efficiency of the treatment, rather would I enter the realm of controversy and suggest that Albuminuria of Pregnancy and Eclampsia are two distinct diseases brought about by different agents. And when we come to examine the previous histories of multigravidous eclamptics we find that 50 per cent. of their previous pregnancies were toxæmic: surely in these women we might well expect the next pregnancy to be again eclamptic if Eclampsia be the prime risk in toxic albuminuria, yet recurrent Eclampsia is acknowledged to be extremely rare. Nevertheless when Eclampsia attacks primigravidous women the kidneys are so badly damaged that 33 per cent. of these women have albuminuria in future pregnancies and in primigravidous albuminurics there is a recurrence rate of 68 per cent., more than twice as great. The only explanation I can offer for this is that in Eclampsia the toxæmia is of short duration whereas in albuminuria it is of long duration, long because we allow it to be long by employing eliminative measures instead of inducing labour, during which time the kidney is undergoing prolonged exposure to the toxin and consequently there is more extensive permanent damage to the renal structure than in the case of Eclampsia where our treatment is immediate and drastic and labour is soon over.

The Risk of permanent damage to the kidneys.

We have now to consider whether the kidneys receive permanent damage and what is the nature of that damage. It is stated by most observers that 4 per cent. of pregnant women suffer from albuminuria. If it be a fact that toxæmia causes no permanent damage to the renal structure, then the chances of a woman, so affected and being cured, of falling into this class again is extremely small. Yet my cases show that 68 per cent. of primigravidæ have a recurrence in future pregnancies and 50 per cent. of multigravidæ have had toxæmia before. This is more than a coincidence and suggests three hypotheses:—

(1) That the woman affected has some in-born idiosyncrasy to the pregnant state. This is refuted by the fact that it is not uncommon to find one or more normal pregnancies interspersed between two toxic ones.

(2) That the uterine contents are toxic on some occasions and not on others in the same woman. It is impossible to prove or disprove this.

(3) That the kidney completely recovers in one-third of the cases and is permanently damaged in two-thirds.

To my mind this last is more than a hypotheses ; it is an established fact ; for in a follow-up clinic, we are able to place the patients in three groups :—

(a) Those in which the kidney conditions clears up quickly and toxæmia does not recur in future pregnancies. These are cases where the toxæmia was of short duration.

(b) Those who have a recurrence in future pregnancies but remain free of albuminuria in the intervals. These are cases where the toxæmia has been of long duration.

(c) Those in which albuminuria is permanent and which may be considered as cases of chronic nephritis secondary to the toxæmia.

It is impossible to say what is the exact nature of the pathological change in the kidneys. In fatal cases the epithelium lining the renal tubules is seen to be extensively degenerated, and it is reasonable to infer that in non-fatal cases the change is the same but of less extent, and whether there is complete regeneration is a moot point. It is difficult to believe that the renal epithelium, so delicate and high specialised, changing, as it does, in character and function in different parts of the renal tubule, can ever completely recover from the effects of exposure to a toxin such as we are considering. It is more reasonable to suppose that the damage done is permanent though limited in extent and that the remaining healthy portion of the kidney, though enough for normal function in the intervals between pregnancies, is insufficient to meet the demands of a future pregnancy. Thus the evidence of toxæmia will appear at an earlier date in successive pregnancies, further damage is done to the kidneys and the time arrives when the patient is in a condition of chronic degenerative nephritis.

Treatment.

When we face the fact that one pregnancy in four is wasted, and this does not include miscarriages, we must ask ourselves whether, knowing that the kidneys receive permanent damage in 68 per cent. of cases, we are justified in allowing a toxæmic pregnancy to continue. The review of my cases has convinced me that the expectant treatment I have hitherto pursued has been a mistake and that labour should be induced in all cases. We have then to consider the most suitable time to induce, and how long can we safely leave the kidneys exposed to the action of the toxins. In this latter connection I would put the limit of safety at two weeks, for Gibberd has established the useful fact, that where the toxæmia lasts for three weeks or more, a recurrence in future pregnancies is almost certain. Now, it is unusual for a true toxæmia of pregnancy to show signs until about the 35th week in the first instance, and we could safely employ expectant treatment until the 37th week and then induce labour. When we induce for contracted pelvis, we can at the 34th week get a living baby of about 4 pounds and one which has a good chance of survival, yet it is a noticeable fact that in toxæmia of pregnancy the premature baby is generally smaller than it should be for the

period of pregnancy and therefore if we can delay the induction for a while without great risks to the mother we might gain much. I would limit this delay to a fortnight, and for this reason. It has been a common experience to continue with expectant treatment because the case has appeared to be doing well and the foetus has been alive at say the 37th week, and at the 39th week the patient has gone into labour and has been delivered of a macerated foetus. Such a foetus would have been saved by a timely induction and the maternal kidneys would have been spared much damage. I would therefore advise that in all cases labour should be induced at the 36th week or earlier if the toxæmia has been in evidence for a fortnight.

I will turn now to a consideration of the cases of Eclampsia. Table 3 shows the number of cases and the results. So far as Hospital practice is concerned a death rate of 14.6 per cent. might well be improved if there were less delay in their admission. Frequently they are not sent to Hospital for many hours after the first fit and the all important opportunity of beginning the treatment immediately after the onset of the disease has been lost. I have shewn that in multigravida Eclampsia 50 per cent. of the women have had previous toxic pregnancies, and it is only reasonable to suppose that the eclamptic pregnancy has shown signs of toxæmia for a considerable length of time before the onset of fits and the opportunity of forestalling them has been lost. With proper ante-natal care of these women Eclampsia is so easily prevented. It is, however, different in the case of primigravida, for with them the apparent improvement in health so common in primigravida, often masks one or more of the important prodromal symptoms. It is usual to hear that they had never been so well until the first convulsion. It is important therefore to consider the warning signals. The chief ones are as follows:—

Variations in the amount of Albumin.

The amount of albumin as a rule increases with the toxæmia, beginning with a trace and increasing until the urine boils solid. Conversely, on a water diet we can reduce the amount almost to a trace again. If under such treatment the albumin remains stationary in amount or increases, or worse still, if there be a sudden increase, we are faced with a sign of the greatest importance.

A rising Blood Pressure.

The blood pressure is of more importance than the amount of albumin yet commonly it is apt to receive less attention. Pregnant women are for the most part young and their blood pressure should not be above 130. Variations between 130 and 160 in cases of albuminuria are indications for a very careful watch over the patient. In many of my cases the tension has been as low as 140 but the average has been around 170 and there have been a few up to 200. To my mind, a steadily rising blood pressure is of graver significance than a high one which is stationary.

Oedema.

The amount of oedema is no criterion to go by, for in many of the severe cases of albuminuria and in some of the fulminating cases of Eclampsia, oedema of the legs has been slight or even absent. But if oedema be present and it is extending to the vulva and abdominal wall we have an important sign, and still more so if there be oedema of the face, if only of the eyelids.

Headache.

An intractable frontal headache is a sure indication that the case is growing steadily worse.

Vomiting.

Vomiting is uncommon in the ordinary albuminuria of pregnancy but in about half the cases of Eclampsia it has preceded the first fit. It is usually thought to be indicative of liver damage which in itself is responsible for another very important danger signal, namely epigastric pain.

The onset of fits is the first indication that the case is one of Eclampsia. The first fit is sudden and unexpected and often in the case of primigravidæ is the sign noticed that anything is wrong. Nevertheless, with the first fit the case has taken a serious turn for the worse, for at this time, the patient not only has a lethal amount of toxin in her system but also has to contend with the added strain of the fits with the attendant cyanosis and perhaps too œdema of the lungs, all of which contribute towards cardiac failure or cerebral hæmorrhage.

There are certain features concerning the fits which give some indication as to prognosis although it must be admitted that a case which appears to be progressing may suddenly become worse and die. These features are as follows:—

- (1) The relation of the fits to the onset of labour;
- (2) The number, frequency and intensity of the fits;
- (3) The response of the fits to treatment;
- (4) The degree of Coma.

The Relation of the Fits to the onset of Labour.

Table IV shows this relationship and points out that where the fits began before labour the death rate was three times what it was where the onset was intrapartum and twice what it was in postpartum cases. To this Table I append the corresponding though dissimilar figures of the London Committee on Eclampsia (1922). These two sets of figures show one striking feature, namely the high death rate in postpartum Eclampsia, namely 11.7 per cent. in my series and 27.6 per cent. in that of the Committee. I think these two percentages give an erroneous idea as to the gravity of the postpartum cases, for it has generally been taught that postpartum Eclampsia is the least fatal. I believe this to be true for the reason that a delivered woman is less likely to be sent to Hospital than one undelivered, and I suggest that if it were possible for us to see or hear of all postpartum cases instead of only those sent to Hospital, we would find the death rate is lower than in either of the other two groups.

The Number, Frequency and Intensity of the Fits.

In 65 of the cases the number of the fits has been recorded. It is interesting to note that 40 had no more than 7 fits with only two deaths, whereas 25 had more than 7 fits and amongst these there were 9 deaths. This suggests that if the degree of toxæmia be so slight, or our treatment so efficient, as to permit of not more than 7 fits, then the chances of a happy result are good. Of the two fatal cases having less than 7 fits, one was a primigravidæ who had 5, the other was a multigravidæ who had 6. Both were

fulminating cases without any return to consciousness between the fits. Table V. shows the average number of fits in the survived and fatal cases. It seems to suggest that 7 fits may be considered a maximum for comparative safety. This table also shows the number of fits endured by the fatal cases and I have placed a cross against those who died undelivered and it may be noticed that of six women who died undelivered, four had fewer than ten fits. These were cases of extreme toxicity who made no response to treatment.

Table V also shows that the number of fits before the fatal issue in multi-gravidæ was just short of twice the number in primigravidæ mounting up to over thirty and in one case to 46. If we refer back to Table I we shall see that Eclampsia is half (and probably less) as common in multigravidæ as in primigravidæ, yet in them the death rate is more than twice as great and they have a greater number and greater average percentage of fits. Now, although the toxæmia is the ultimate cause of the fits, and although the fits are for the most part the cause of death in some way or other, nevertheless the two act in conjunction, each contributing its own quota. We can, therefore, conceive of two possibilities bringing about the fatal issue:—

- (1) A high degree of toxæmia with relatively few fits *e.g.* in primigravidæ.
- (2) A low degree of toxæmia and many fits, *e.g.* in multigravidæ.

This latter possibility is merely on all fours with what has already been stated, namely that half of the multigravidous eclamptics have had toxæmia of pregnancy in former pregnancies and it is not unlikely that many of the other half have had albuminuria which has been overlooked. Thus the multigravidæ start off with an adverse handicap, namely a badly damaged kidney, and with such a condition it would only require a low concentration of the necessary toxin to bring about the vasospasm, the suppression of urine and the convulsions, all essential features of the eclamptic seizure. And it may not be wrong to assume that it requires a good many hours before the toxin reaches such a degree of concentration in the blood stream as to become itself a menace to life, during which time the fits are steadily mounting in number and increasing in severity, and so reducing the patient's strength quite apart from the ever present danger of cerebral hæmorrhage.

This view is in no way opposed to my opinion already expressed namely that Eclampsia is not the prime risk in any given case of albuminuria of pregnancy; but where we have a toxæmic pregnancy neglected or there is a recurrence of the condition, we have a good foundation for Eclampsia in a future pregnancy.

The Response of the Fits to Treatment.

When we consider how much the fits sap the vitality of the patient, and add to this the risks they entail of cardiac failure or cerebral hæmorrhage, it follows that if they can be controlled or completely abolished our chances of success are much greater. Yet the cessation of fits does not mean a cessation of the toxæmia, but it does mean that the general vasospasm has been overcome and that the kidneys may restart excreting urine. Re-excretion of urine is a favourable sign.

In my series of cases, of those who recovered, 25 ceased having fits before the onset of labour. Of these, 10 were delivered on the following day, and the

rest went varying periods up to 9 days and one as long as 17 days before delivery. In 14 the fits ceased with delivery and in 13 they continued after labour. It is well to bear in mind that it would seem that a persistence of the fits after delivery has no bearing on the mortality which is the same as the general mortality of severe cases. (London Committee's Report 1922.)

The Degree of Coma.

The degree of coma is perhaps the most important factor in prognosis. Except in the fulminating cases, coma is never extreme at the onset yet it is progressive, and what might appear to be a favourable case at the beginning may steadily become more comatose even under treatment. We can classify the cases into three groups in relation to the degree of coma:—

(1) Where the fits are at long intervals and between them the patient recovers complete consciousness, or at most in the intervals there is drowsiness from which she can be easily roused. Most of these recover;

(2) Where the fits are frequent and the patient does not recover consciousness between them, yet she may respond to stimuli. About half of these recover;

(3) Where the coma is profound, the breathing stertorous and even the corneal reflex may be absent. These cases rarely recover.

Having considered the chief features of the disease we are able to group the cases into mild and severe. The mild cases have the following characteristics:—

Number of fits less than 7.

Consciousness between the fits.

Excretion of urine.

Low blood pressure.

Normal temperature and slow pulse.

It must not be overlooked that a mild case may steadily progress in severity.

The severe cases show the following signs:—

More than 7 fits.

Profound coma.

Suppression of urine.

Blood pressure of 170 or more.

Temperature above 101.

Rapid pulse.

The outlook in patients exhibiting any two of these signs is grave.

Treatment.

Space does not allow me to enter into a detailed description of the treatment which has been employed in my series; suffice it to say that it has been, so far as circumstances would permit, that advocated by Professor Studgaroff, and with a death-rate of only 14 per cent. (as compared with 10 per cent. of Studgaroff) I see no reason to change it, except in the matter of closer attention to details. Operative treatment has no place in eclampsia, for *accouchement forcé* gives a death-rate of over 60 per cent., and with Cæsarean section the death-rate is double what it is in expectant treatment in both mild and severe cases. What operation can be done when eclampsia is post-partum in its onset? The advocates of surgical treatment find difficulty in answering this question.

(a) ALBUMINURIA—

TABLE I.

					Died.
1. <i>Primigravidæ</i>					
Traced	...	102	...	1	
Untraced	...	21	...	—	
2. <i>Multigravidæ</i>					
Traced	...	44	...	1	
Untraced	...	12	...	—	
Total	...	<hr/> 179	...	<hr/> 2	

(b) ECLAMPSIA—

1. <i>Primigravidæ</i>					
Traced	...	41	...	5 = 10.4%	
Untraced	...	7	...	—	
2. <i>Multigravidæ</i>					
Traced	...	28	...	6 = 20%	
Untraced	...	2	...	—	
Total	...	<u>78</u>	...	<u>11 = 14%</u>	

TABLE II.

Results where the first toxæmic pregnancy is:—

(a) Primigravidous.				(b) Multigravidous.	
Baby lived	...	91	...	...	50
Neonatal death	...	16	...	...	2
Stillborn or macerated	...	27	...	...	10
Unrecorded	...	1	...	...	2
<i>i.e.</i> , 43 or 32% wasted.				<i>i.e.</i> , 12 or 19% wasted.	

Results where the first eclamptic pregnancy is:—

(a) Primigravidous.				(b) Multigravidous.	
Baby lived	...	28	...	...	8
Neonatal death	...	7	...	...	4
Stillbirth	...	12	...	...	11
Died undelivered	...	2	...	...	4
Unrecorded	...	2	...	...	3
<i>i.e.</i> , 21 or 42.8% wasted.				<i>i.e.</i> , 19 or 70% wasted.	

That is, in the equivalent of 272 pregnancies 95 were wasted or 35%.

TABLE III.

CASES OF ECLAMPSIA.

Primigravidæ	...	48	...	Died	5 = 10.4%
Multigravidæ	...	30	...	Died	6 = 20%
Totals	...	<u>78</u>	...		<u>11 = 14%</u>

FATAL CASES.

Primigravidæ	...	2 of 5 died undelivered	
Multigravidæ	...	4 of 6 died undelivered	
Totals	...	<u>6 of 11 = 55.5%</u>	

TABLE IV.

ECLAMPSIA.

Relation of onset of fits to onset of labour.

	A.P.	Died		I.P.	Died		P.P.	Died
Primigravidæ	18	3	...	18	1	...	10	1
Multigravidæ	12	4	...	9	1	...	7	1
Unrecorded ... 4								
Totals ...	30	7 = 23.3%		27	2 = 7.5%		17	2 = 11.7%

London Committee ... 1922	287	59 = 20.5%		84	14 = 16.6%		76	21 = 27.6%
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TABLE V.

Average number of Convulsions:

	Survived.	Fatal.
Primigravidæ ...	6.9	13.4
Multigravidæ ...	8.9	25.1
Average ...	7.6	19.8

Number of fits in fatal cases:

Primigravidæ	5x	9x	15	16	22	
Multigravidæ	6x	8x	26x	31	34	46x

THE TREATMENT OF ANTE-PARTUM HÆMORRHAGE

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For all practical purposes, vaginal hæmorrhage occurring during pregnancy may be taken as being due to premature separation of the ovum from its uterine attachments. The term Ante-partum Hæmorrhage is restricted to such hæmorrhage coming on after the twenty-eighth week, that is, after the child is viable. As the only part of the uterine contents to have a vascular connection with the uterine vessels is the placenta, ante-partum hæmorrhage is caused by the premature separation, in whole or in part, of the placenta. In the great majority of cases the blood passes through the cervical canal and appears as vaginal bleeding, but, in a small number, the blood is retained in the uterus. The circumstance which brings about this retention is almost invariably severe toxæmia and in such a case toxæmia rather than loss of blood may be the more important complication.

Ante-partum Hæmorrhage may be divided into the two classical types, Unavoidable and Accidental. The former signifies that for delivery to take place per vaginam, separation of the placenta is unavoidable because the placenta is situated in whole or in part on the lower uterine segment which must dilate. The

And lastly, there is the case of the general practitioner or family doctor, who is certainly not least worthy of consideration in any post-graduate scheme of education, especially when we remember that he is not only the back-bone of the profession but that he forms by far and away the majority of its members. No doubt the courses referred to in the last paragraph meet his wants, but to attend them means as a rule, on account of the time and expense involved, the foregoing of his much-needed annual holiday. Hence it is an experience which cannot be often repeated.

One cannot admire too much the doctor who sacrifices his holiday for the purpose of making himself more useful to the community and it is in the easing of the burden of this altruistic urge that we see the virtue of the special intensive week-end courses about to be inaugurated, and for which we are sure there is a great future. They have the advantage that the time consumed is not more than even the most busy practitioner can occasionally spare, the time is fully and beneficially employed and the survey embraces all branches of his work so that variety will lessen the strain involved. To us one particularly fortunate arrangement seems to be that all the members attending the course will during its progress live together so that they will obtain that most valuable stimulus which comes from mutual discussion of the day's work.

ERRATA.

We regret that through an oversight in the proof-reading, the following appeared on page 286 of the August number of the Journal (9th line from the bottom of the page) "Refusal of a foetus to remain as a *breech* often implies a contracted pelvis".

The author has requested us to correct this.

For the word "*breech*" read "*vertex*".

To the Editor,

Sir.—I would be grateful if you would allow me space to correct an error in my Lecture on "Albuminuria and Eclampsia" which you published in the August number. In the last paragraph the name of Professor Stroganoff appears as "Studgaroff"

and I regret that this escaped my notice during the revision of the proofs.

Since the publication, I have received a letter from Dr. Perott, of South Kensington, telling me that his former master, Professor Stroganoff has been publicly disgraced in Russia and sent to exile. I am sure the news of this tragedy will be received with the greatest sorrow by all readers of this Journal.

It is said that prophets have no honour in their own country, and I cannot help thinking that Professor Stroganoff does not lose much, for, outside Russia, he is honoured and revered by Obstetric Surgeons throughout the world.

Yours faithfully,

ANDREW McALLISTER.