BIOCHEMICAL STUDIES

IN

THE TOXAEMIAS OF PREGNANCY.

By

M.D. CRAWFORD,

M.B., Ch.B.

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University of Glasgow.

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P R E F A C E

The investigations on which this thesis is based were carried out in the Research Department and Wards of the Royal Maternity and Women's Hospital, Glasgow, during the tenure of the Faulds Fellowship, Glasgow University.

Two papers, entitled "The Effect of Labour on the Plasma Uric Acid and Urea" and "Changes in Blood Concentration in Normal and Toxaemic Pregnancy", have been published in the Journal of Obstetrics and Gynaecology of the British Empire, 1939, 46, 540 and 1940, 47, 63, respectively, from data recorded in this work; a third paper, entitled "Changes in Plasma Uric Acid and Urea in Eclampsia", is at present in press.

I have much pleasure in acknowledging my indebtedness to Dr. H.L. Sheehan for his constant encouragement and helpful criticism throughout the course of the work. Thanks are also due to Professor S. Cameron, Professor J. Hendry, Dr. J. Hewitt and Dr. R.A. Lennie for permission to investigate cases in their wards in the Royal Maternity and Women's Hospital, Glasgow, and to Dr. A. MacNiven for permission to study cases in his wards at Gartnavel Mental Hospital.

SECTION I

INTRODUCTION.

About ten per cent of all pregnant women are found to suffer from one or other of a group of disorders peculiar to pregnancy which are referred to as the "toxaemias of pregnancy". Under this term "toxaemias of pregnancy" there are included - vomiting of pregnancy, acute yellow atrophy of the liver, pre-eclampsia, eclampsia, low reserve kidney, etc. The term hypertensive toxaemia denotes those conditions associated with high blood pressure, albuminuria and oedema, either together or singly. It is to be specifically noted that, throughout the present work, the word "toxaemia" is used as a clinical description without any implication that hypothetical toxins are circulating in the blood.

The hypertensive toxaemias of pregnancy have been the subject of endless research and controversy for many years; in many textbooks of obstetrics a whole chapter is given to the discussion of the various theories of the etiology of eclampsia and pre-eclampsia.

In 1843 Lever discovered albumen in the urine of eclamptic women and for some years after this the disease was thought to be allied to uraemia. This theory soon fell into disfavour but was supported by Volhard (1924) who regarded eclampsia as a form of acute uraemia and proposed

the term "eclamptic uraemia". He considered that general vasoconstriction with resulting capillary changes was a primary factor. Many workers agree concerning the importance of capillary damage; Hinselmann (1924) and Heynemann (1924) have been able to demonstrate marked changes in the capillaries in cases of hypertensive toxaemia and eclampsia; they found widening and lengthening of the skin capillaries with resulting stasis of blood flow - these changes they considered were secondary to spasm of the arterioles. In 1919 Zangemeister advanced his "Hydrops Gravidarum" theory which postulates increased capillary permeability as the fundamental lesion of eclampsia. He considers that oedema, raised blood pressure, albumenuria and other signs are all secondary to increased transudation of fluid to the tissues and that cerebral oedema is the cause of the convulsions, headaches and retinal changes. He concludes that "the long sought eclamptic toxin is water". Foetal elements (Veit, 1905), foetal metabolic products (Dienst, 1902 and 1927), placental (Liepmann, 1906; Young, 1927; Cruickshanks, Hewitt and Couper, 1927) and intestinal (Solomons, 1922 and 1933) toxins have been suggested as causal agents of eclampsia while some workers advance theories relating to endocrine imbalance (Hofbauer, 1918 and 1933; Hoffman and Anselmino, 1931; Cushing, 1934; Smith and Smith 1933 and 1934) and anaphylaxis (Rosenau and Anderson, 1908).

Almost all the theories have one point in common

and that is, the recognition of a general metabolic upset in these cases. This metabolic upset is evidenced by changes in the biochemical findings of the various body fluids. Investigations of these changes have thrown some light on the nature of the disease, but there has been much divergence of opinion even in regard to some of the findings. In particular, reported results from hypertensive toxaemic patients disagree with regard to the question of retention of non-protein nitrogen fractions. Low blood urea values have been found by many workers, (Zangemeister, 1903; Williams, 1921; Hellmuth, 1923; Heynemann, 1924; Plass, 1924; Stander, 1924; Stander, Duncan and Sisson, 1925; Cruickshanks, Hewitt and Couper, 1927; Dieckmann, 1933; Stander and Cadden, 1934b); while the results of others show a considerable retention in the blood of all non-protein nitrogen fractions (Caldwell and Lye, 1921; Killian and Sherwin, 1921; Walthard, 1922; Frey, 1923; Bokelmann and Rother, 1925). One of the reasons for these differences is that, in most cases, determinations were made on groups of different patients, either at different stages in pregnancy or with varying degrees of toxaemia, and the average results compared. When the results are examined in detail, it appears that those workers whose average figures lie within normal limits had several patients with values much above normal and, conversely, in the results of those workers in favour of a non-protein nitrogen retention, there are patients with low non-protein nitrogen and urea values. Very few investigators have attempted to follow, in any detail, the changes occurring in the non-protein nitrogen

fractions of the blood in individual patients and thus the factors influencing the blood non-protein nitrogen fractions in these hypertensive toxaemic cases have not been recognised.

Renal function tests in hypertensive toxaemic cases before and soon after delivery have been found to be very unsatisfactory both from a diagnostic and from a prognostic point of view. Stander, Ashton and Cadden (1932) found the urea clearance and creatinine excretion tests of some value in differentiating mild nephritis from low reserve kidney cases, but give no results for pre-eclamptic cases. Dieckmann (1935), on the other hand, in a detailed investigation of renal function in convulsive and non-convulsive, acute and chronic hypertensive toxaemic patients, found that the urea clearance test was of little value during pregnancy or soon after delivery because of the wide range found in the normal pregnant cases. Cadden and McLane (1934) found that only the urea clearance test was sensitive enough to differentiate true chronic nephritis from the other toxaemias; the average results from pre-eclamptic and eclamptic cases were lower than the average results from normal cases, but the range in each group was so wide that the test was of no diagnostic value. Elder, Sinclair and Rogers (1936) in a similar investigation also stressed the wide range of the normal values for renal function tests in pregnancy and indicated that this was partly due to wide variations in blood urea values in different cases, the values in some normal cases being very low. It is, therefore, of great importance that

any factor causing changes in the blood urea, in normal and hypertensive toxaemic cases, and so contributing to the wide variations, should be investigated and eliminated.

During recent years much attention has been drawn to the raised uric acid content of the blood in pre-eclamptic and eclamptic cases. An increased uric acid content, decreased CO_2 combining power and lowered pH of the blood are now generally accepted as being the outstanding findings in the blood chemistry in eclampsia. Raised blood uric acid in certain cases is often considered to be diagnostic of eclampsia while some workers (Stander and Cadden, 1934b) regard the height of the uric acid value as an index of the severity of the disease. It will be shown here, however, that several factors, apart from the toxaemia, cause marked changes in the blood uric acid level, and unless these are understood the actual uric acid value in an individual case may be very misleading.

S E C T I O N II

THE EFFECT OF LABOUR ON PLASMA URIC ACID AND UREA IN NORMAL AND NON-CONVULSIVE TOXAEMIC PATIENTS.

The present study deals with the changes in uric acid and urea in the blood of individual patients during the last few weeks of pregnancy, during labour and throughout the puerperium. Blood samples were collected daily in most cases and, during labour, at intervals of a few hours. Each specimen was examined for plasma uric acid content by Benedict's direct colorimetric method. (1922 a and b). The accepted normal range by this method is 2 to 4 milligrams per 100 c.c. of plasma, and all normal cases, in non-pregnant patients or in those in early or mid-pregnancy, studied here have fallen within this range. Blood-urea was determined by the gasometric method of Van Slyke and Kugel. (1933).

The series consisted of 93 women:

A. Twenty-five normal cases. Patients at term without any albuminuria, hypertension or oedema at any time during pregnancy. Most were primigravidae, a few having slightly contracted pelves. (See Appendix - Cases 1 to 25.)

B. Fifty-six pre-eclamptic cases. Practically all the patients were primigravidae having toxaemic signs of varying degrees of severity but without any convulsions. The term, pre-eclamptic, is used here, as by most authors, to define a specific type of hypertensive toxaemic patient; it does

not imply that these patients later developed convulsions. The patients have been sub-divided from a clinical point of view as mild or severe. The mild pre-eclamptic patients correspond to the low reserve kidney cases of American writers; they were free from symptoms and had only mild hypertension, albuminuria or oedema. (Cases 26 to 32.)

Patients with eclampsia have been excluded from consideration in this section.

C. Twelve cases of chronic toxaemia. A rather miscellaneous group of multiparae who had toxaemic symptoms and signs during pregnancy suggestive of the conditions often referred to as essential hypertension or nephritic toxaemia. The diagnosis was based, among other things, on the previous history, multiparity and advanced age.(Cases 83-94)

Findings Before Onset of Labour.

The findings for plasma uric acid and urea in these patients at term, but before the onset of labour, are shown in Table I.

These findings are essentially similar to those of many other workers (Caldwell and Lye, 1921; Killian and Sherwin, 1921; Frey, 1923; Hellmuth, 1923; Plass, 1924; Stander, 1924), the only noteworthy difference being the somewhat raised uric acid values in the normal cases. Of these, the patients with values 4.0 milligrams or more per 100 c.c. before the onset of labour were all primigravidae. Many of them had been examined a few weeks earlier and at that time the uric acid values had been within normal limits.

The probable cause of this rise of uric acid in primigravidae in the last few weeks of pregnancy is discussed later.

Table I.

Plasma uric acid and urea before onset of labour.

Class of Patient	Uric acid (milligrams per 100 cubic centimetres of plasma)		Urea (milligrams per 100 cubic centimetres of plasma)	
	Range	Mean	Range	Mean
Normal patients	2.5 to 5.2	3.9	16 to 21	18
Mild pre-eclamptic patients	3.0 to 5.3	4.5	14 to 32	21
Severe pre-eclamptic patients	3.6 to 9.7	6.1	14 to 55	30
Chronic toxaemic patients ...	2.7 to 7.3	5.3	14 to 55	26

In the pre-eclamptic and chronic toxaemic patients great difference in the uric acid content of the plasma was found from case to case, during the last month of pregnancy. In the mild pre-eclamptic patients the value was rarely much above normal limits (only 5 patients had values over 5 milligrams per 100 c.c.). Patients with high values were usually of the severe clinical type, but the reverse did not hold; not all patients with severe symptoms had a high uric acid. Many of these pre-eclamptic and chronic patients were under observation for 2 to 3 weeks before the onset of labour and it was found that, although a definite conclusion could not be made from a single determination, the rise and fall in uric acid values, in individual patients, accompanied changes

in the clinical severity of the conditions. Stander and Cadden (1934b) found much the same relation. However, while a high uric acid value was found to indicate a severe toxaemia, it did not appear to have any relation at all to impending convulsions.

The blood urea value in the normal patients were found to be very low as shown in Table I. In the majority of the toxaemic patients the blood urea values obtained lay within the normal limits for non-pregnant cases, but were definitely higher than the results from the normal pregnant cases. Similar results have been recorded by Zangemeister (1903), Williams E.C.P. (1923), Hellmuth (1923), Cruickshanks, Hewitt and Couper (1927), Dieckmann (1933), etc. It has not, perhaps, been sufficiently stressed that, because of the low normal values in pregnancy, any case with a blood urea value of more than 25 milligrams per 100 c.c. before the onset of labour, should be regarded as abnormal.

Changes During Labour and the Puerperium.

When these same patients were followed throughout labour and the puerperium it was found that in practically every case a rise in uric acid, and in many cases a rise in urea also, occurred during labour, with a return to normal during the puerperium. In some patients the rise was slight and would have been overlooked if blood-specimens had not been collected at intervals of a few hours before and after the time of delivery. In other patients, however, the rise was considerable, the values mounting well above normal limits

CHART 1.

Relation of Length of Labour to Rise of Uric Acid in all Cases

even in non-toxaemic patients. The highest value reached among normal patients was 10.4 milligrams of uric acid and 104 milligrams of urea per 100 c.c. plasma. (Case I)

Uric Acid Findings.

When the results from the whole series were studied it was found that the extent of the rise in uric acid, above the ante-partum level, depended directly on the length and severity of the labour. The evidence for this is shown in Table II and Charts I to IV.

Table II.

Relation of length of labour to uric acid rise.

Hours in Labour		Maximal rises in uric acid (milligrams per 100 cubic centimetres of plasma)	
Range	Mean	Range	Mean
0 to 4	3	-0.5 to 0.5	0.1
5 to 12	7	-0.1 to 1.7	0.5
13 to 24	18	0.1 to 3.0	1.1
25 to 48	35	1.0 to 4.5	2.4
49 to 120	73	3.1 to 6.4	4.6

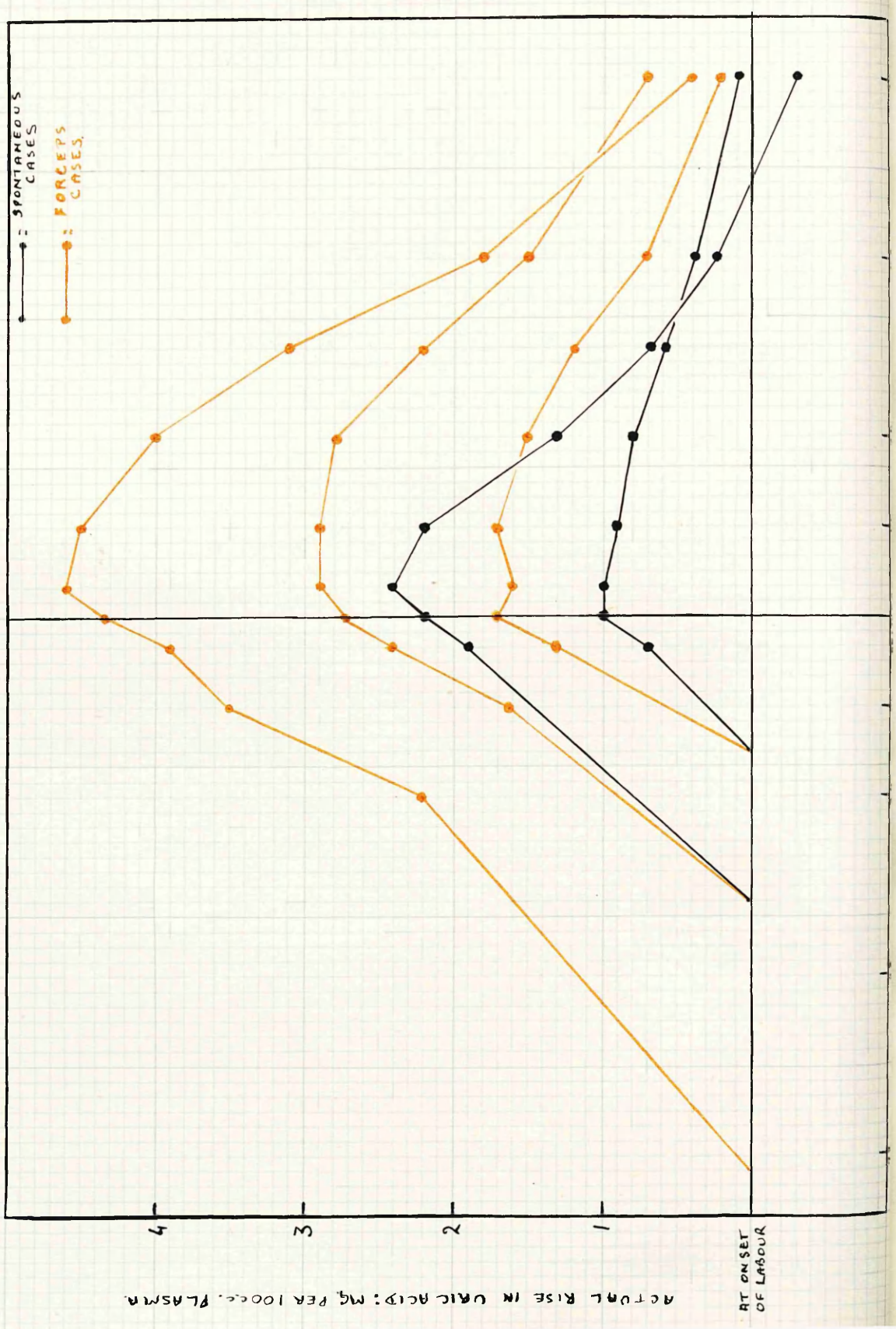
In Table II all patients have been arranged into groups according to length of labour, without regard to classification as normal or toxaemic. The mean rise for each of these groups at different times throughout labour and early puerperium is shown in Chart I. The base line of the graph represents the original uric acid value of each

group before the onset of labour, while the points plotted show the actual rise, in milligrams of uric acid per 100 c.c. plasma, above the antepartum level. The point of origin of each curve from the base line indicates the mean length of labour for patients in that group.

From this chart it can be seen that, not only does the uric acid increase during labour, but that the extent of the rise is strictly proportional to the length of the labour. It will be noticed that the rise during labour is gradual but slightly more rapid towards the end, i.e. in the second stage of labour. The maximal value was usually found at about 4 hours postpartum, although variation was found from case to case, some patients reaching their maximum at delivery and others continuing to rise till 24 hours post-partum. In most patients the values returned to an absolute normal between the third and fifth days post-partum; cases which started with high ante-partum values thus show a more rapid post-partum fall, which is seen in later charts. Patients having a severe labour showed a greater rise than patients with a mild labour of the same duration; this fact accounts for the range of the rises in each group shown in Table II. The estimation of the severity of the labour depended merely on clinical observation and, therefore, accurate figures cannot be given to show this point. In the great majority of patients, however, the length of the labour gives a fairly satisfactory index of its severity. As one would expect, the majority

CHART II.

Relation of Length of Labour to Rise of Uric Acid in forceps and non-forceps cases.



of the patients having a long labour were primigravidae while many of the patients with a short labour were multiparae, but the rise in plasma uric acid was found to be unaffected by the parity of the patient, depending only on the length of the labour.

In Chart II the patients delivered by forceps, and those not so delivered, are plotted separately. In the group of patients with labour over 48 hours, there was only one spontaneous delivery and, therefore, a curve for the patients not delivered by forceps at that time has not been drawn. The results from the other two groups, however, show a greater ante-partum rise in patients delivered by forceps and a slight delay in the post-partum fall to normal. It is obvious that neither delivery by forceps nor the administration of an anaesthetic can affect the ante-partum rise in uric acid, except to shorten it by terminating the labour. The greater rise in patients delivered by forceps is most probably due to the greater severity of the labour; forceps were applied in most cases because of a prolonged second stage and maternal distress. The delay in the post-partum fall is not marked and may be due either to the more severe labour or to the anaesthetic. Patients who have had a similar anaesthetic (gas-oxygen-ether) apart from labour, e.g. for the performance of external versions, have shown a slight rise in plasma uric acid (mean 0.7 mg., range 0.1 to 1.5 mg. per 100 c.c.) lasting for rather more than 24 hours. The delayed post-partum fall is, therefore, probably due to the anaesthetic at delivery.

CHART III.

Relation of Length of Labour to Rise of Uric Acid in Toxaemic and non-toxaemic cases.

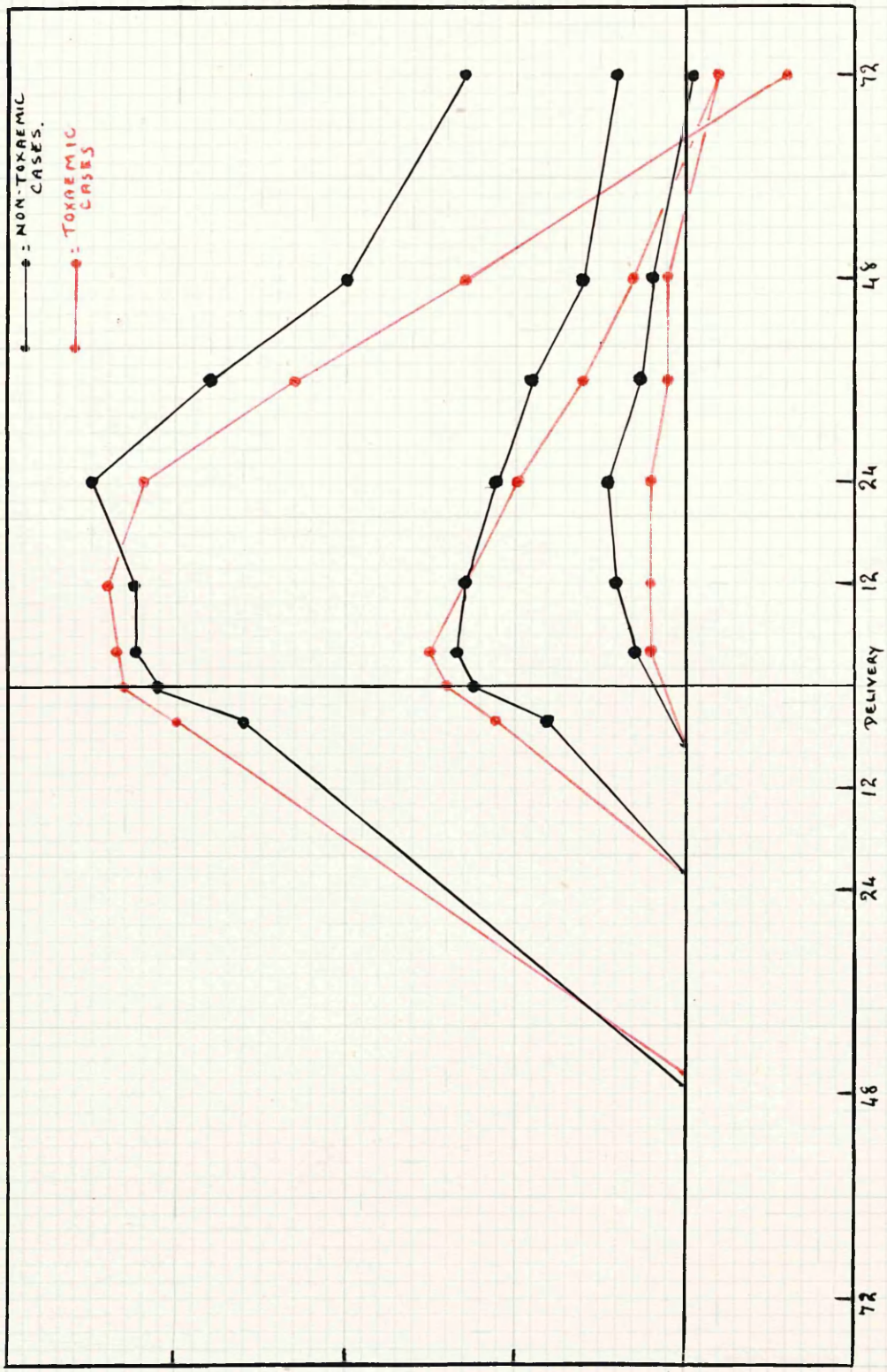
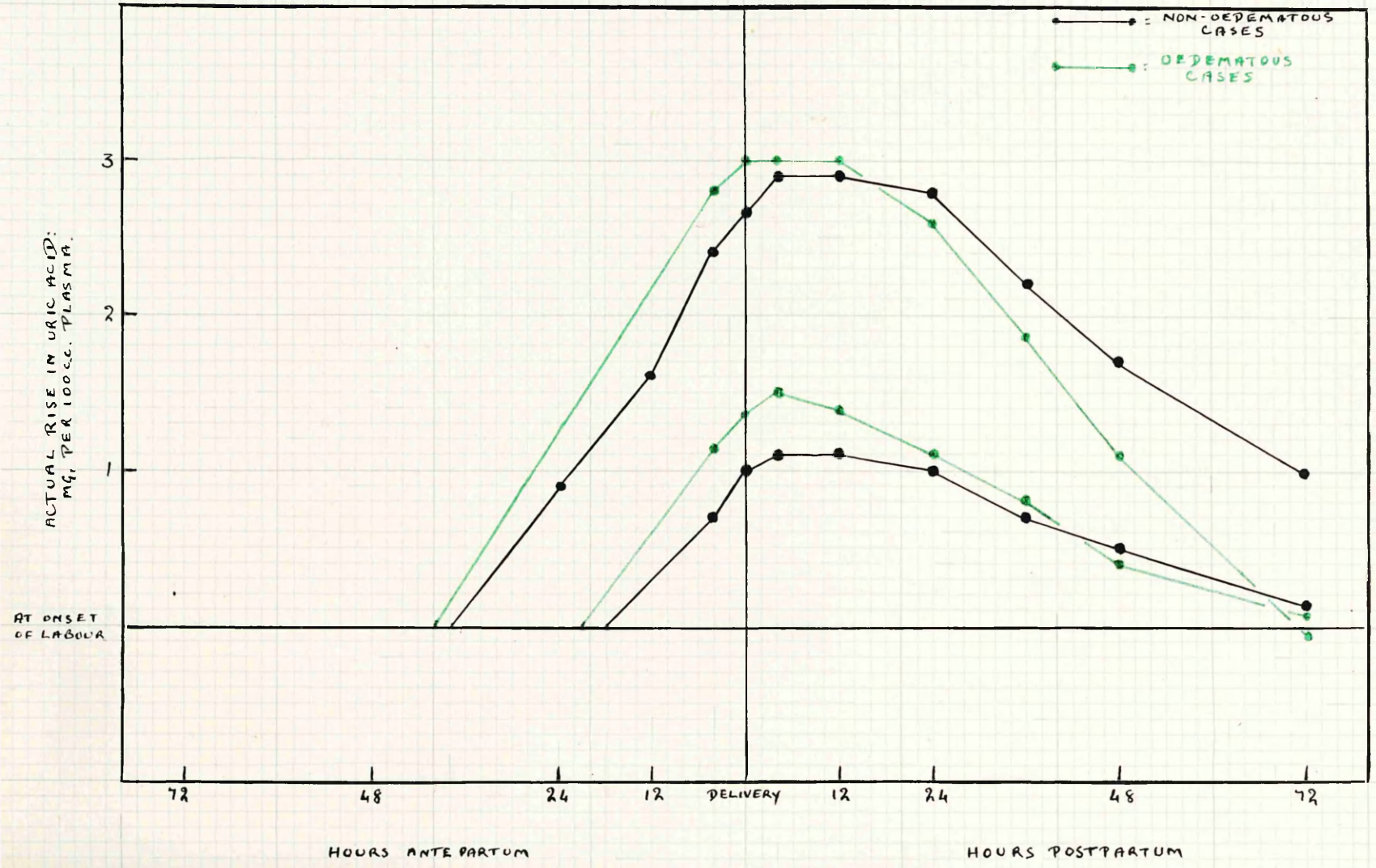


CHART IV.

Relation of Length of Labour to Rise of Uric Acid in oedematous and non-oedematous cases.



In Chart III the combined results from chronic toxaemic and pre-eclamptic cases have been plotted against the normal cases, but still in groups according to length of labour. The toxaemic cases have been combined, as there is not any significant difference between them. In the longest labour groups, only patients delivered by forceps have been included, so that a fair comparison can be made. It will be seen that, when the mean length of labour is the same, the rise in uric acid is not higher in these toxaemic cases than in the normals. The post-partum fall in the toxaemic cases is greater and more rapid than in normal cases because the uric acid is falling from a higher original ante-partum value to an absolute normal value after delivery.

In Chart IV the results from oedematous patients are charted against the results from those not oedematous. The oedematous patients form the majority of the severely toxaemic patients. The curves are essentially similar; the slight differences in height can be accounted for by the differences in the length of labour of each group.

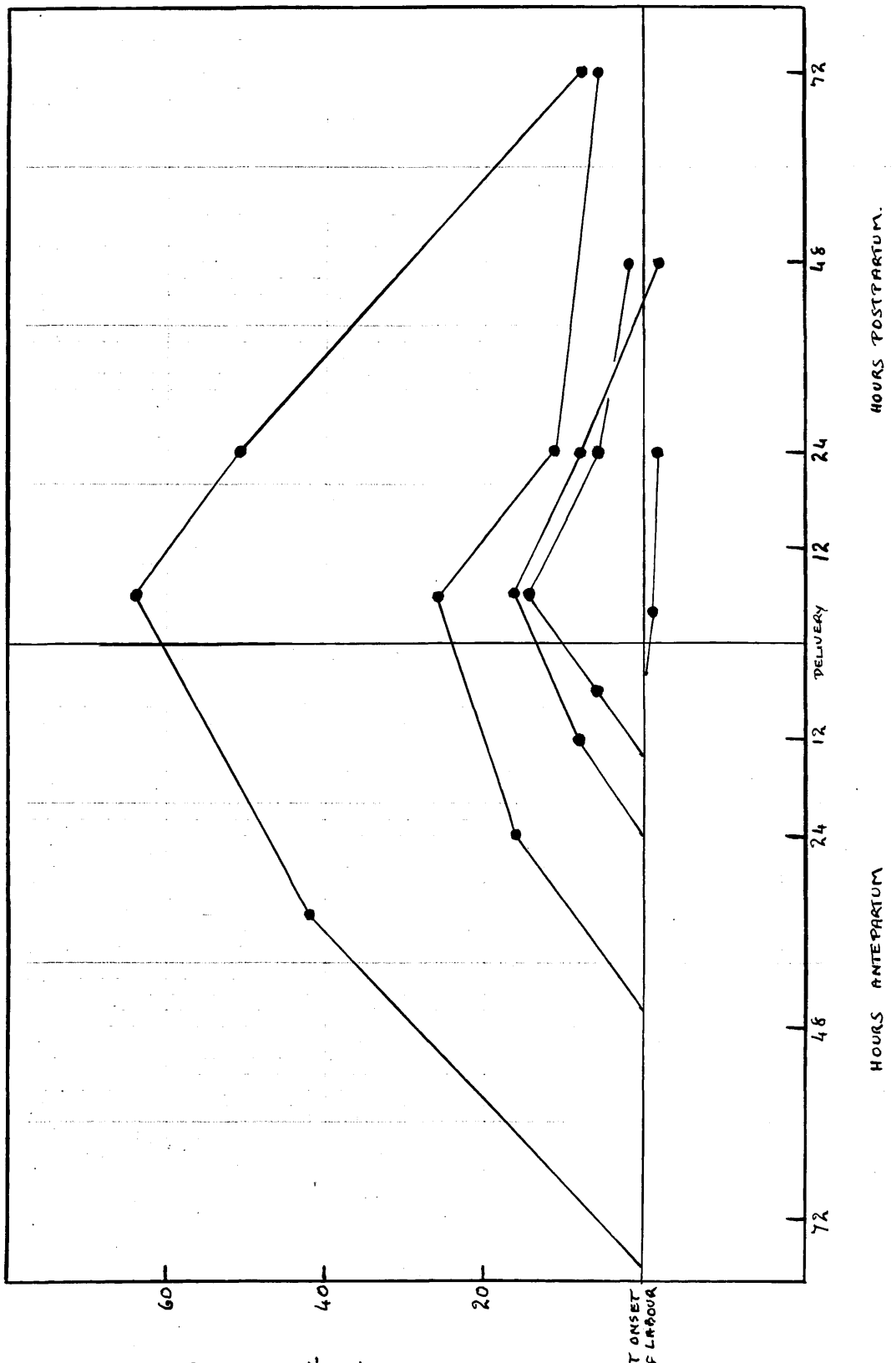
It may, therefore, be concluded that the rise in plasma uric acid which has been shown to occur during labour is neither related in any way to the height of the original ante-partum value nor to the classification of the patient (normal or toxaemic) nor to oedema, nor to the method of delivery. It appears to be related only to the length and severity of the labour.

Urea Findings.

In most of the patients plasma urea determinations

CHART V.

Relation of Length of Labour to Rise of Plasma Urea.



were made at the same time as those of uric acid, and a similar rise was usually found during labour. The rise was most marked in long labours, while patients with normal labours showed little or no rise.

Chart V shows the mean urea rise during labour and the early puerperium in patients grouped according to length of labour. The similarity to the uric acid changes will be noted, the same relation to the length of labour being seen. The rise in urea occurred later in labour than the uric acid rise. In many patients there was not a complete return to the ante-partum level; this is probably due to the fact that ante-partum urea values are below the normal non-pregnant level, and that there is a physiological rise in urea in the puerperium. (Williams, 1923, Cadden and Faris, 1938).

It was found, however, that, although the urea and uric acid both showed changes in the same direction and at the same time in individual patients, a constant proportion did not exist between their values from case to case. As with uric acid the rise was not obviously related to toxaemia, forceps, or anaesthesia, though the data are less complete in the case of urea than in that of uric acid.

Discussion.

The findings recorded here show that labour can produce a marked elevation of the uric acid and urea in the plasma. It is obvious, therefore, that any estimations of these substances, or other non-protein nitrogen fractions reported in women at or about delivery or in the early

puerperium, have little significance unless the length of the labour is known and the appropriate rise accounted for.

The rise in uric acid and urea during labour is probably due to diminished excretion by the kidney, and related to the oliguria which occurs so commonly during labour. The impairment of renal function is presumably due mainly to the pressure of the child's head on the base of the bladder and lower end of the ureters. Such a local pressure effect is of course to be expected from a priori considerations, and it is proved pathologically by the invariable finding on post-mortem examination of oedematous, haemorrhagic or ulcerated areas at the base of the bladder in fatal cases following a protracted labour.

The rise in uric acid to values above normal, which was noted in some non-toxaemic primigravidae during the last few weeks before term, may also be due to pressure on the lower ureters since it occurs after the fixing of the head in the pelvis.

A survey of the literature on blood-nitrogen, in normal and toxaemic patients at about the time of delivery, or in the puerperium, reveals that only a few workers made serial investigations on individual patients, and of those none made examinations of the blood regularly at short intervals of time. As a result, the effect of labour on the non-protein nitrogen fractions of the blood has not been clearly shown and a reference has not been found which relates raised non-protein nitrogen values to the length of labour.

In most cases the estimations were made only at intervals of 2 to 7 days, and figures are not given for duration of labour. Owing to lack of information on this point, and also in view of the relatively infrequent examinations, it is difficult to assess the significance of these findings in general.

The most detailed work in this connection appears to be that of Cadden and Faris (1938), who studied total non-protein nitrogen, urea nitrogen and rest nitrogen in 40 patients during the first stage of labour, at delivery and on alternate days throughout the puerperium. They found a slight increase in the mean results for the series during labour and in the first day post-partum. During the 2nd and 3rd day post-partum the levels remained steady or showed a slight fall; this was followed by a rise of all values reaching normal non-pregnant levels by the 10th day post-partum. When their findings in individual cases are studied, however, wide variations are found; 8 out of 40 cases showed a decrease in total non-protein nitrogen from before delivery to after delivery, 5 out of 40 cases showed a decrease in urea nitrogen and 14 out of 40 showed a decrease in rest nitrogen, while 1 case showed an increase in non-protein nitrogen from 24.2 to 48 mg. per 100 c.c., in urea nitrogen from 8.2 to 25.6 mg. per 100 c.c. and in rest nitrogen from 16 to 22.4 mg. per 100 c.c. during labour. Their mean results tend to agree with the present findings, however, and it is likely that the factor of length of labour is responsible for the changes described and would explain the wide range of the findings reported.

Stander and Cadden (1934b), published biochemical findings from many pre-eclamptic and eclamptic cases. Determinations on pre-eclamptics were made "immediately before delivery" and "after recovery". The results show higher uric acid values before delivery with normal values at the second examination; they do not record the time of onset or the length of labour, however, and therefore their conclusions cannot be accepted at their face value. A rise in uric acid during labour can be seen in several of their charts, but no mention is made of it in their paper.

Siedentopf (1932), in an investigation on changes in blood chemistry during the three stages of labour, determined total non-protein nitrogen of the blood in several women. In most of his cases he found a slight rise during the first and early second stage and a greater rise during the late second stage of labour, but there were several marked exceptions: 11 out of 12 patients showed a fall in total non-protein nitrogen in the third stage of labour while 12 out of 14 cases showed a rise between the end of labour and the 5th or 6th day of the puerperium. The investigation appears to have been carried out on groups of different women at the various stages of labour and therefore the changes in individual patients cannot be followed. His findings prior to delivery agree with the present results but no notice is taken of the length of labour, and this probably accounts for the inconsistencies in his results.

The rapid fall in non-protein nitrogen fractions which he finds immediately after delivery has not been

found in the present study and is in direct disagreement with the findings of Plass. His method of recording changes as a percentage of their original value makes even small changes appear striking and exaggerates any experimental error.

Plass in 1924 examined a series of 24 patients, 7 normal, 7 eclamptic and 10 non-convulsive toxaemic, for non-protein nitrogen fractions during labour and the puerperium. In some cases the first specimen was taken off during labour, but in others the first specimen was taken off after delivery. A slight rise in all figures for total non-protein nitrogen, uric acid and urea was found to occur post-partum (maximum levels reached on 2nd or 3rd day post-partum) with a subsequent fall to normal. The rise was greatest in toxaemic cases and was usually found to coincide with the clinical improvement of the patient. Plass suggested that this increase in non-protein nitrogen fractions was due to dilution of the blood by tissue fluid containing higher non-protein nitrogen, urea and uric acid content. The present results, especially that from oedematous cases, do not support this theory and it seems likely that the rise which he demonstrated was merely the end of the rise which had occurred during labour.

Hellmuth (1923) examined a series of normal patients in labour and again once or twice in the puerperium, usually about the 7th day post-partum. He found values raised during labour and normal at later examinations. In 12 of his normal cases the uric acid was found to be over 3.5 mg. per 100 c.c. (highest 5.0 mg. per 100 c.c.). He explains these findings

as due to a metabolic upset in primigravidae at term causing difficulty in complete oxidation of proteins.

Frey (1923) determined blood non-protein nitrogen fractions in a series of normal and toxaemic pregnant women at delivery and later in the puerperium. In normal cases at delivery he found non-protein nitrogen, urea and uric acid at the upper limit of normal but well within normal limits later in the puerperium. In toxaemic cases he found that the non-protein nitrogen and urea were often much increased at delivery, but the values varied greatly from case to case. His uric acid findings were very inconsistent. It is likely that the high values found by these two workers, Hellmuth and Frey, in their normal and in many of their toxaemic cases can be accounted for by length of time in labour.

The relation between the length of labour and the rise of plasma uric acid and urea is not, in retrospect, a surprising finding. The rise occurs to the same extent in normal as in toxaemic patients and may be considered almost physiological; but in those cases with labour over 48 hours the nitrogen retention is so marked that the possibility of actual kidney damage as a result of the obstruction cannot be excluded. In a few cases the return to normal values has been slow and it may be that these patients have suffered some permanent renal damage. These findings are therefore another indication of the dangers of a protracted labour. The rise of plasma uric acid and urea, due to labour is also of importance, however, in that it can produce such great changes in blood chemistry as to invalidate any work which

does not take it into account.

Summary.

The changes in the plasma uric acid and urea occurring during labour and the early puerperium have been studied in detail in a series of normal and toxaemic women. A rise of plasma uric acid and urea is found to occur invariably during labour, the height of the rise being dependent on the length of the labour. The rise is independent of toxaemia and other factors. There is a corresponding fall in the early puerperium so that normal values are usually reached by the third day after delivery.

S E C T I O N III.

CHANGES IN PLASMA URIC ACID AND UREA IN ECLAMPSIA.

In the previous chapter it was indicated that wide variations of opinion existed, between investigators, on the question of non-protein nitrogen retention in cases of hypertensive toxaemia of pregnancy. It was shown that these varied opinions were frequently due to the fact that the data were obtained from different patients at different stages of pregnancy and that no account was taken of the influence of labour on the blood non-protein nitrogen fractions. In eclampsia most workers agree in finding raised uric acid values but exceptions have been noted and the height of the values recorded varies greatly.

Williams (1921) was one of the earliest workers to find high uric acid values in eclamptic patients while the blood urea values in the same patients were within normal limits. He examined the blood of 5 eclamptics during the acute stage of the disease; the average uric acid value from these cases was 7.8 mg. per 100 c.c. From this series he was unable to find a relation between uric acid values and clinical symptoms, labour or stage in pregnancy. Caldwell and Lye (1921) found a non-protein nitrogen retention, of varying degree, in the blood of non-convulsive toxaemic and eclamptic patients. They concluded that in cases with a

severe retention, the prognosis was bad. Killian and Sherwin (1921) found total non-protein nitrogen of the blood at the upper limit of normal in eclamptic cases - the urea percentage was low, while the uric acid was markedly increased. Each patient was examined once only and there was wide variation from case to case (range of uric acid values was 3 to 11 mg. per 100 c.c.). The increase in values was thought to be due to renal impairment. Hellmuth (1923) in a larger series of eclamptic cases also found high blood uric acid values while the blood urea was normal or only slightly raised. He concluded from his series that the height of the uric acid value in an eclamptic case was of no value in prognosis and that a high value in a pre-eclamptic case did not necessarily indicate impending fits. Frey (1923), on the other hand, found uric acid values within normal limits in eclamptic cases while blood non-protein nitrogen was often considerably raised. His uric acid findings in his different groups of patients, however, appear to be unusual - in the normal non-pregnant group the range is given as 12 to 15 mg. per 100 c.c. and it must be accepted that the method employed was not in any way as reliable as the modern methods. Plass (1924), as mentioned in the previous chapter, investigated non-protein nitrogen fractions in 7 eclamptic patients. He found uric acid raised, especially in the first two days of the puerperium, while urea values were within normal limits. Stander (1924); Stander, Duncan and Sisson (1925); Bokelmann and Rother (1925); Stander and Radelet (1926); Cruickshanks, Hewitt and Couper (1927); have all compared average uric acid findings

of different groups of pregnant women and found high values in eclamptic cases. More recently, Stander and Cadden (1934b), and lately Cadden and Stander (1939), made much more detailed studies of the biochemical changes in the plasma in cases of eclampsia. In the former of these papers they recorded the biochemical findings from 108 eclamptic and 40 pre-eclamptic cases and in the latter, composite charts were made from 132 eclamptic cases. Blood non-protein nitrogen and urea were found to be low in most cases, although in some cases a rise was found to occur late in the disease. The blood uric acid content was found to be raised in both pre-eclampsia and eclampsia and was regarded as a criterion of the severity of the disease.

In spite of the great volume of literature which has been written on the subject of the biochemical findings in eclampsia, it has again been found that little attempt has been made to follow in detail the changes which occur in individual patients. In the present study the changes occurring in the plasma uric acid and urea were followed in a series of 42 eclamptic patients throughout the course of the disease, and an attempt has been made to disentangle the various factors influencing these non-protein nitrogen fractions. In all cases the blood was examined daily or oftener, and in many cases determinations were made at hourly intervals during the acute stage. The results from the previous study of 25 normal pregnant and 56 pre-eclamptic patients are quoted here for comparison with the present findings. It is to be specifically noted that every patient

who developed eclamptic fits is classed here under the term "eclampsia", even in the stage before the onset of fits; the term "pre-eclamptic" again refers only to patients with toxaemia, characterised by hypertension, albuminuria and the other standard signs, who did not develop eclamptic convulsions. The eclamptic patients have been divided into 3 groups:-

(a) 18 Ante-partum eclamptics (Cases 104 to 121) - patients who had fits before the onset of labour, but not during labour or the puerperium.

(b) 16 Intra-partum eclamptics (Cases 122 to 137) - patients who had fits during labour; this group includes cases in which fits began before labour but continued after the onset of pains, and also cases with fits during labour which continued after delivery.

(c) 8 Post-partum eclamptics (Cases 138 to 145) - patients who had fits which began only after delivery.

Two of the ante-partum and one of the post-partum cases died during the acute stage of the disease.

Uric Acid Findings.

In the previous section it was shown that during labour there is a rise in plasma uric acid and urea, both in normal and in non-convulsive toxaemic patients; as will be shown here, precisely the same changes occur in eclamptic patients during labour. In addition, in pre-eclampsia and in eclampsia there are changes in plasma uric acid level resulting from toxaemia; and also, in eclamptic patients there are changes related to the onset of fits. The uric

acid findings in various types of eclamptic patients can be understood only when these three factors have been disentangled. For this purpose the cases studied here are considered under three headings.

(1) The findings before the onset of fits or of labour. This group involves only the effect of the preceding hypertensive toxaemia, the other two factors being automatically excluded.

(2) The changes which develop in relation to the fits. To exclude the changes which had developed previously as a result of the toxaemia the basic level is taken as that which had been found in each individual patient before the onset of fits; the rise due to the fits is calculated from that level. To exclude the changes due to labour, a correction has been introduced in those cases where the fits coincided with or followed the time of labour. This correction is calculated from the data which have been given in the previous section with regard to the influence of labour in patients without fits; the mean figure appropriate to the actual duration of labour is used in each case. The validity of this correction, in the present connection, is shown by the close agreement of the actual figures from eclamptic patients not in labour with the corrected figures from eclamptic patients in labour.

(3) The influence of labour. When the specific effects of the previous toxaemia and of the fits have been

analysed it is then possible to consider the actual findings in these patients with regard to labour.

Findings Before the Onset of Fits.

For comparison with the eclamptic cases, note may be made of the changes in plasma uric acid for a few weeks before the onset of labour, recorded in the earlier series of 25 normal and 56 pre-eclamptic patients, who did not develop eclampsia. The blood was examined at intervals of 1 to 2 days in each case. It was found that the uric acid values varied greatly from case to case. In the mild pre-eclamptic cases the value was rarely much above normal limits. Cases with high values were usually of severe clinical type, but the reverse did not hold; the cases with severe symptoms did not all have high uric acid values.

Ten cases from the present eclamptic series were examined before the onset of fits. (Cases 104, 122, 123, 124, 125, 126, 127, 142, 143, 144). In each case the last examination in this pre-convulsive stage was made shortly before or during the first fit. Contrary to what is generally stated in the literature (Stander and Cadden, 1934b; Cadden and Stander, 1939) the uric acid values at this time were found to be only moderately raised. In patients who developed eclampsia suddenly without any preceding stage of severe toxaemia, the uric acid value before the first fit was usually within normal limits (3.0 to 4.4 mg. per 100 c.c.) while in cases with evidence of severe preceding toxaemia the uric acid values were slightly higher (5.2 to 6.5 mg. per 100 c.c.). The mean value of the 10 cases was 5.5 mg. per 100 c.c. When

these figures are compared with those from the non-convulsive pre-eclamptic cases, it will be seen that much higher values are obtained among patients with severe pre-eclampsia who are not going to develop convulsions, as is shown in Table III.

Table III.

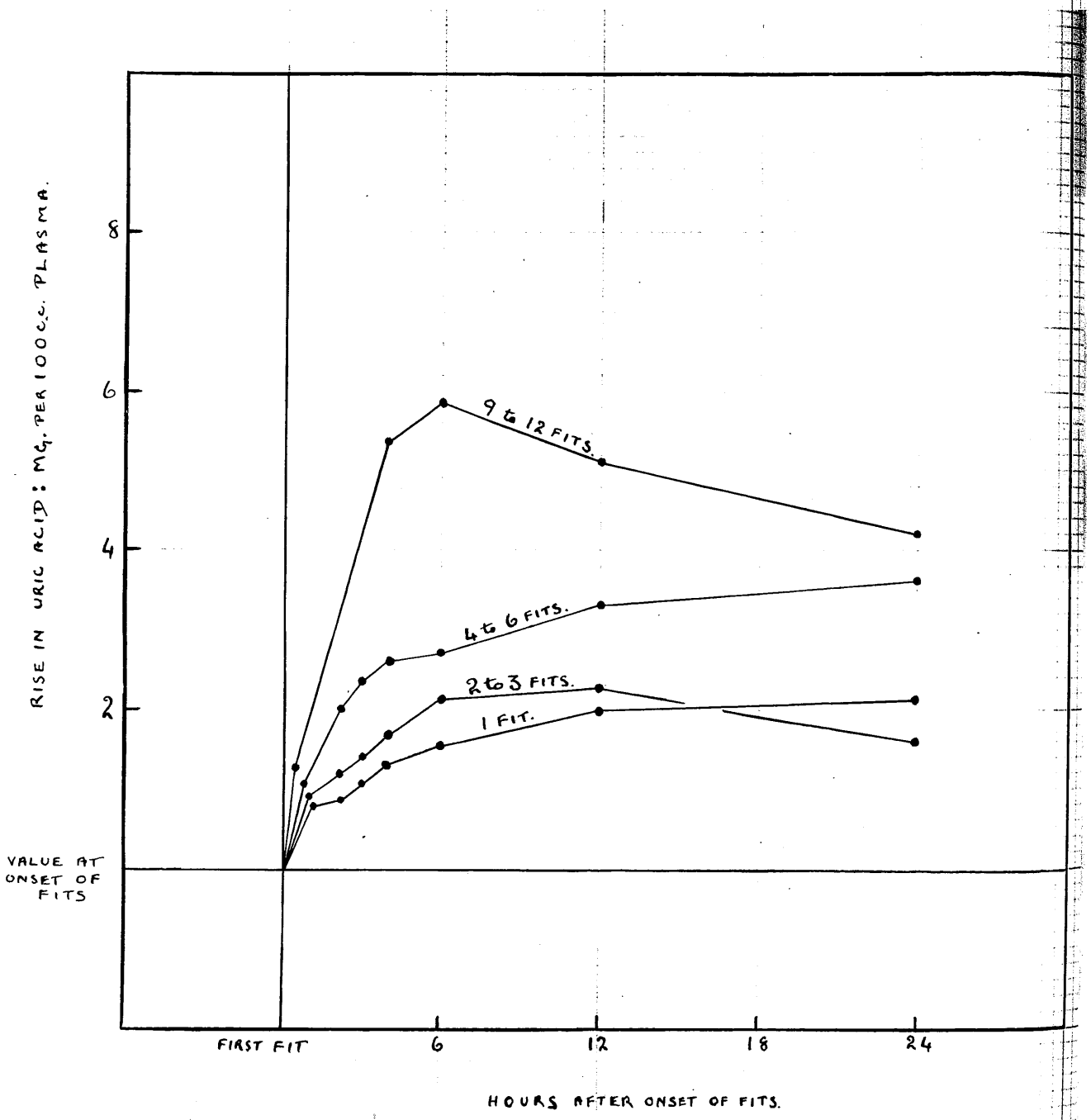
Uric Acid Values in Normal and Non-Convulsive Toxaemic Cases near Term and in Eclamptic Cases before the Development of Fits.

	Number of Cases.	Plasma Uric Acid: mg. per 100 c.c.	
		Mean.	Range.
Normal Pregnant.	25	3.9	2.5 to 5.2
Mild Pre-eclamptic.	29	4.4	3.0 to 5.4
Severe Pre-eclamptic.	27	6.4	4.8 to 9.7
Eclamptic before Fits.	10	5.5	3.0 to 6.5

It is therefore clear that a very high uric acid value in a pre-eclamptic patient, while of importance in indicating a severe metabolic disturbance, is not in any way an index of impending eclampsia. From the present findings it appears that eclampsia is actually more likely to supervene in a patient with only moderately raised uric acid (under 6 mg. per 100 c.c.) than in one with a higher uric acid value. This lack of relation between the severity of the previous biochemical findings and the development of fits has a clinical parallel; the severity of the preceding toxaemic signs and symptoms in the present series did not have any relation to the severity of the eclampsia.

CHART VI.

Graph showing Rise in Plasma Uric Acid after Fits.



The Effect of Fits.

In every one of these eclamptic patients a very rapid rise of plasma uric acid was found to occur immediately after the onset of fits. The 10 cases, in which biochemical studies had been made before the first fit, were examined frequently - some at hourly intervals - after the first fit. A steady rise of plasma uric acid of about 2 to 5 mg. per 100 c.c. occurred during the few hours after the first fit; the average rise was about 1 mg. per hour. The rise continued for 4 to 6 hours, even in patients without any subsequent fits, but a greater rise was found in those who had several fits. In Chart VI. the mean uric acid values from these 10 cases and from 8 others have been plotted to show the changes during the 24 hours following the first fit. The cases have been divided into groups according to the number of fits. The base line of the graph represents the uric acid value before the first fit in each case, while the curves indicate the mean rise in uric acid above the original value. The 8 additional cases that have been included were patients who had not been examined actually before their first eclamptic fit, the original estimation being made soon after the first fit ; the longest time being 3 hours. In these cases an allowance has been made for the initial rise before the first estimation, based on the time which had elapsed after the first fit and the mean rise during that time in comparable cases of the main series. In any of the cases in which labour coincided with the fits, the appropriate correction has

been made, as explained earlier.

This chart shows the rapid rise in uric acid which occurs in all cases after the commencement of the fits and demonstrates a clear relation between the extent of the rise and the number of the fits. This rapid rise for 4 to 6 hours after the first fit was a constant finding in every case, but subsequent changes in the uric acid values varied greatly from case to case depending on the course of the disease. High values were always maintained until the fits ceased, but there was no relation between the number of fits and the duration of time that the high values were maintained; this is probably because the time elapsing between fits varied greatly in different patients. After the fits had ceased, the findings were of two main types. In some cases the uric acid values began to fall a few hours after the last fit, but in other cases the values were maintained or even increased for a further 24 to 48 hours. The persistence of high values was often related to a period of oliguria. In the 3 fatal cases (Cases 120, 121 and 145) the uric acid value rose steadily till death even in the absence of fits; the values at death were 11.0 mg., 14.2 mg. and 16.4 mg. per 100 c.c.

In case 121, an ante-partum eclamptic, the first fit occurred at 4.30 a.m. 2 hours later - after 4 fits - the blood examination showed - uric acid 11.8 mg. per 100 c.c., urea 40 mg. per 100 c.c.; the plasma was very red in colour and gave a negative reaction to the indirect van den Bergh test. The patient had no more fits, but remained comatose. 6 hours after the first fit the plasma uric acid was 16.4 mg. per 100 c.c., urea 61 mg. per 100 c.c., the plasma was dark orange in colour and gave a positive reaction to the indir-:ect van den Bergh test - bilirubin 12.6 units. The patient died 2 hours later. At the autopsy performed half-an-hour

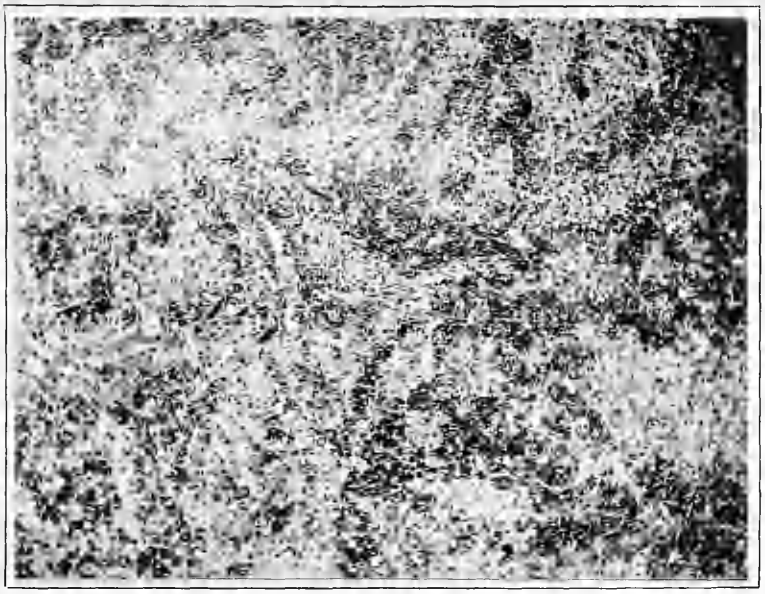


PLATE I.

Liver showing gross eclamptic haemorrhages.

(Case 121)

Low power view.

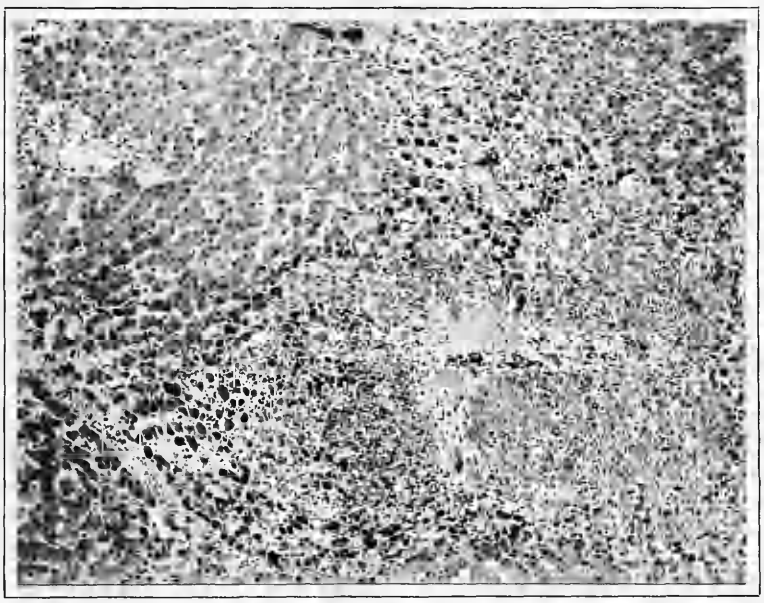


PLATE II.

Liver showing patchy necrosis and periportal eclamptic haemorrhages.

(Case 145)

Low power view.

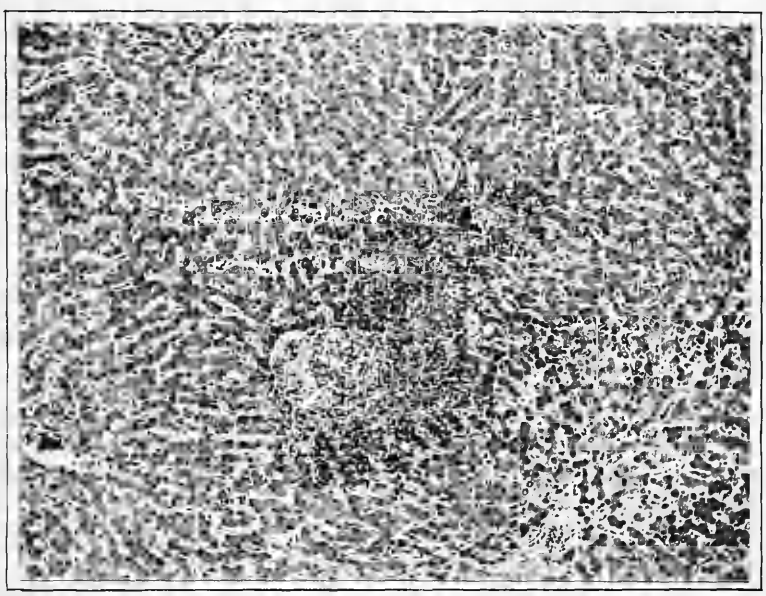


PLATE III.

Liver showing small periportal eclamptic haemorrhages.

(Case 120)

Low power view.

after death the liver showed very extensive areas of haemorrhage. (See plate I.). There were also petechial haemorrhages in the cerebral cortex and in the internal capsule there was a large haemorrhage which had burst into the third ventricle.

In case 145, a post-partum eclamptic with 3 fits before admission to hospital, the plasma uric acid value 12 hours after the first fit was 8.3 mg. per 100 c.c., urea 57 mg. per 100 c.c. The patient died 36 hours after the first fit and the plasma uric acid at death was 14.2 mg. per 100 c.c. although she had no fits after admission. B. urea 112 mg. per 100 c.c. At the autopsy the liver was yellowish and there were several large deep red areas of haemorrhage. Microscopically there were numerous periportal haemorrhages and a few areas of coagulative necrosis affecting several lobules. (See plate II.).

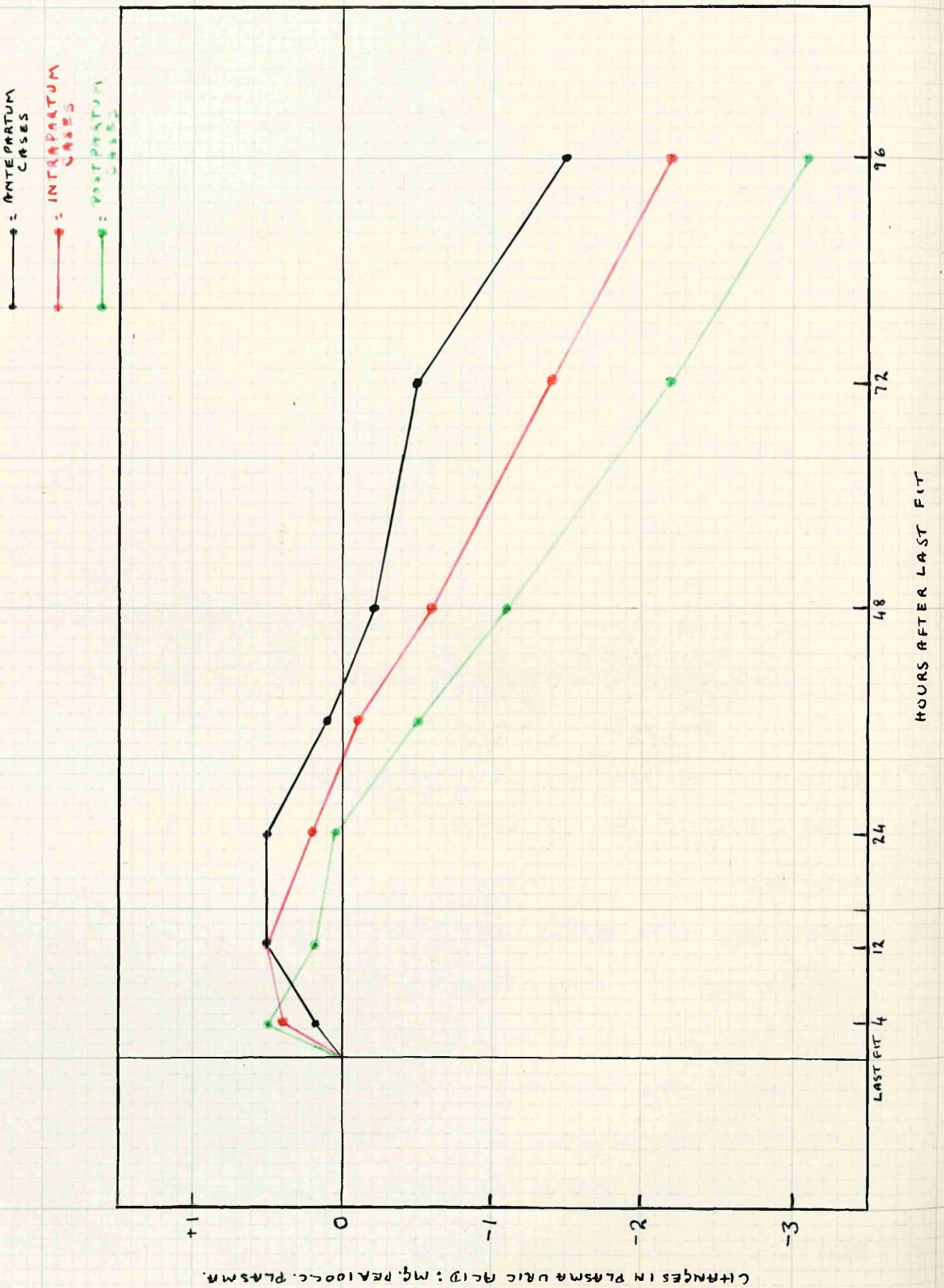
In the third fatal case, case 120, the plasma uric acid value 2½ hours after the first fit and after 4 fits was 6.2 mg. per 100 c.c., B. urea 31 mg. per 100 c.c. This patient had 6 fits during the next few hours but did not die until 21 hours after the first fit. The plasma uric acid value at death was 11 mg. per 100 c.c., urea 69 mg. per 100 c.c. The liver at the post-mortem examination in this case showed numerous tiny haemorrhages throughout its substance, and microscopically only small periportal haemorrhages were found. (See plate III.).

In these three fatal cases the plasma uric acid values at death were proportional to the extent of the liver damage. The blood urea values rose steadily after the fits had ceased.

The fall of the plasma uric acid towards normal values, after the last fit, began earliest in post-partum eclamptics and latest in ante-partum cases. The mean uric acid values for all cases, grouped as post-partum, intra-partum and ante-partum, are plotted in Chart VII. to show the changes occurring after the last fit. As before a correction has been made for the changes due to the duration of labour. The base line of the graph represents the uric acid value immediately after the last fit in each case. It will be seen that a mean rise of 0.5 mg. uric acid per 100 c.c. occurs in each group after the last fit, and that

CHART VII.

Graph showing Changes in Plasma Uric Acid after last fit in Antepartum, Intrapartum and Postpartum Cases.



in post-partum cases the fall begins 4 hours, in intra-partum cases 12 hours and in the ante-partum cases 24 hours after the last fit. There are corresponding differences in the rate of the subsequent fall, probably related to the greater ease of excretion of uric acid after delivery.

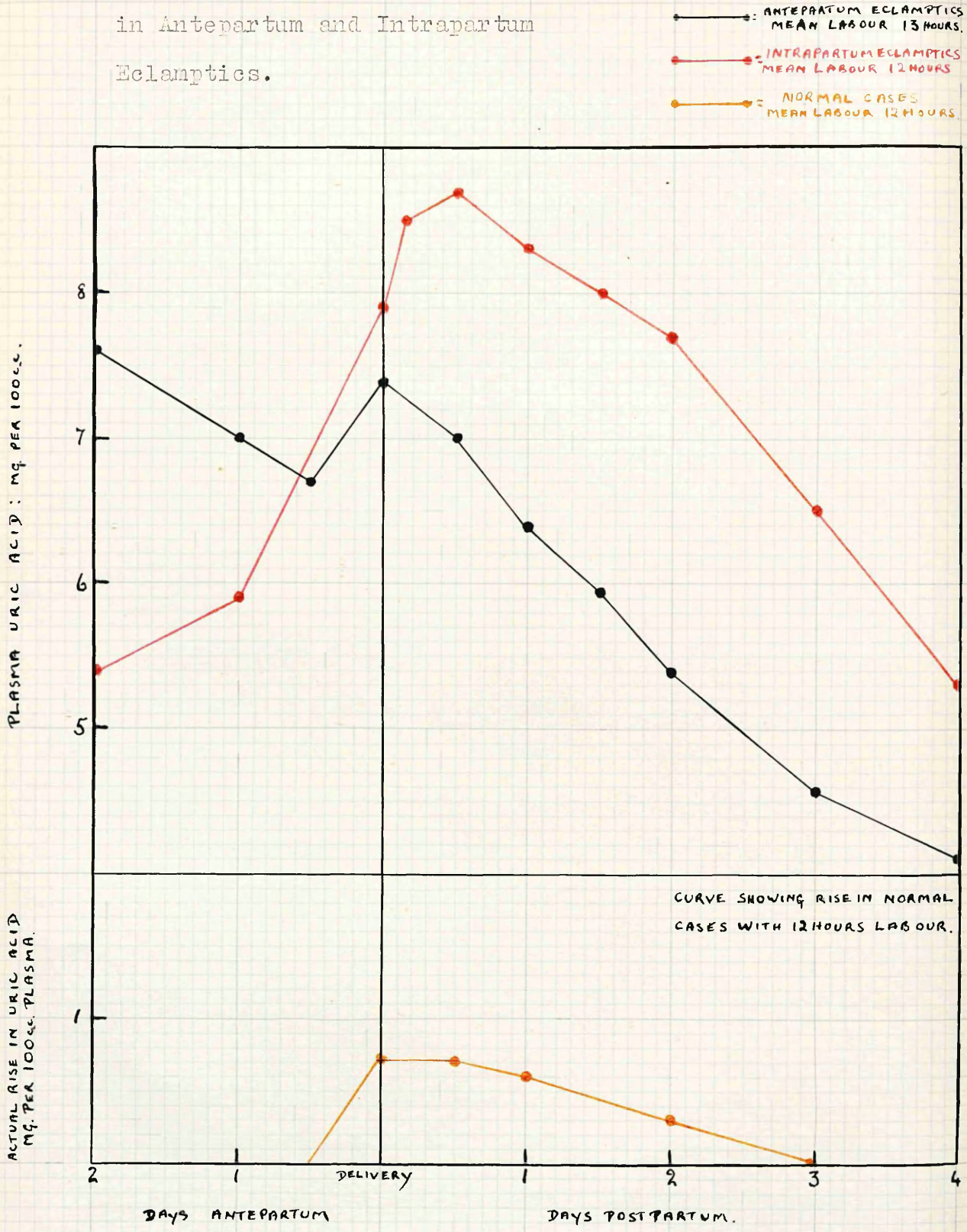
The Effect of Labour.

The effect of labour, apart from fits, is most clearly demonstrated in the patients who recovered from ante-partum eclampsia and did not go into labour until some days later. Most of the ante-partum cases in this series were not admitted to hospital until several hours after the onset of fits and, in these cases, the uric acid values obtained on admission were therefore very high. The mean uric acid value for the 16 ante-partum cases measured after the fits had stopped, but before the onset of labour, was 8.4 mg. per 100 c.c., with a range of 6.2 to 11.5 mg. per 100 c.c. Several of these patients improved clinically after the fits and the pregnancy was allowed to continue for a few days. Parallel to this clinical improvement there was a steady fall in uric acid values until the onset of labour. During labour the fall in uric acid was arrested and in many cases there was an absolute rise.

Chart VIII. shows the changes occurring in the plasma uric acid for a few days before and after the time of delivery in 10 of these ante-partum eclamptic cases. The points plotted represent the mean of the actual uric acid values. The chart shows the rapid fall in uric acid after

CHART VIII.

Graph showing Changes in Plasma Uric Acid during Labour in Antepartum and Intrapartum Eclamptics.



the fits, interrupted by a short rise due to labour. This rise occurs on a falling curve. The real extent of the rise is of course to be calculated from the continuation of the preceding part of the curve on through the time when labour, delivery and the first three days of the puerperium actually occurred. The rise is then found to be almost identical with that which occurs in normal patients who have the same duration of labour, as is shown at the bottom of Chart VIII. Consideration of the individual cases gives the same conclusion; in each case the extent of the rise in uric acid during labour is proportional to the length of labour and equal to that which occurs in normal cases with the same length of labour.

In the intra-partum cases the effects of labour and fits on the plasma uric acid are superimposed. In Chart VIII. the mean uric acid values for 6 intra-partum eclamptic cases are plotted to show the changes occurring before and after delivery; the only cases used are those in which values were obtained before the onset of the fits and of labour. The original value in these cases was only moderately raised (mean 5.4 mg. per 100 c.c.), but the usual rapid rise occurred in relation to the onset of fits. When the cases are arranged according to the length of labour the uric acid rise during labour is found to be much greater than that of normal patients with the same length of labour; the difference being the rise due to the fits.

Post-partum eclamptics show similar uric acid changes related to labour and fits. The rise during labour

is similar to that in normal patients with the same length of labour but, after delivery, the usual rapid rise begins after the first fit.

It may therefore be concluded that labour causes the same rise in plasma uric acid in eclamptic cases as in normal and non-convulsive toxaemic cases.

Urea Findings.

The plasma urea findings in these eclamptic patients were much less striking and less consistent than the uric acid findings. In individual cases changes in plasma urea did not coincide with changes in plasma uric acid, and a constant proportion between the values of these two nitrogen fractions was not found.

Findings Before Onset of Fits.

Table IV. shows the plasma urea findings before the onset of fits and labour. These are taken from the same series of cases as in Table III., though the number of patients in whom urea was estimated is rather less.

Table IV.

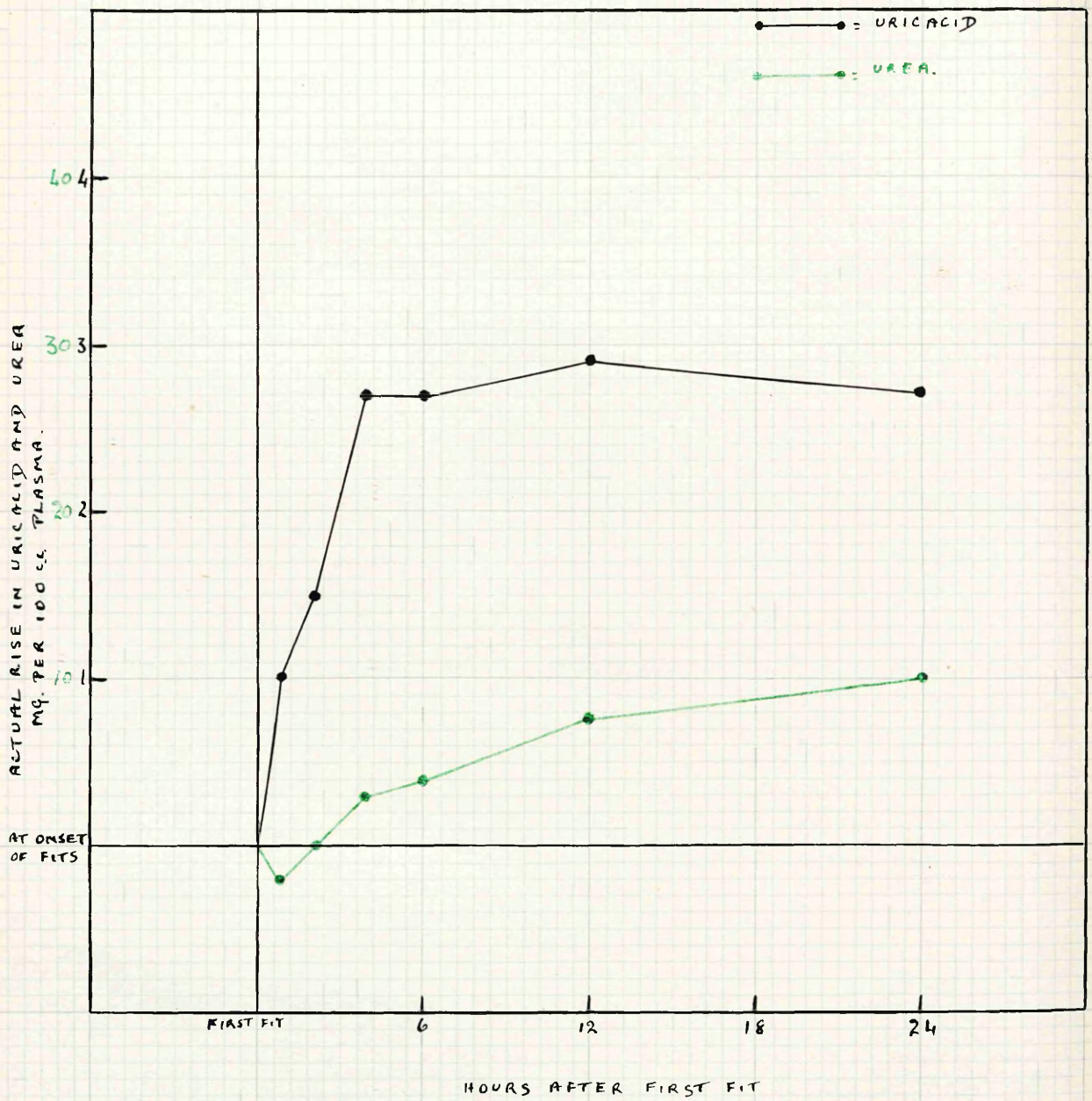
Plasma Urea Values in Normal and Non-Convulsive Toxaemic Patients at Term and in Eclamptic Patients before the Development of Fits.

	Number of Cases.	Plasma Urea. Mg. per 100 c.c.	
		Mean.	Range.
Normal Pregnant.	12	18	16 to 21
Mild Pre-eclamptic.	18	21	8 to 32
Severe Pre-eclamptic.	20	30	15 to 55
Eclamptic before Fits.	9	32	21 to 47

CHART IX.

Graph showing Changes in Plasma urea after the First Fit.

(Plasma uric acid changes from the same cases are shown for comparison.)



In contrast to the uric acid findings, the plasma urea value in these eclamptic cases, immediately before the onset of fits is, if anything, slightly higher than the mean plasma urea in the severe non-convulsive pre-eclamptic cases at the same stage in their pregnancy.

The Effect of Fits.

After the onset of fits, while the plasma uric acid was mounting rapidly, the plasma urea was found to change very little. In a few cases the urea value fell slightly for 1 to 2 hours after the onset of fits while in others it remained more or less constant. Several hours after the first fit it began to rise in most cases; this occurred slowly and continued for varying lengths of time. Neither the height of the rise nor its duration appeared to have any relation to the number of fits; a few patients with numerous fits did not show any increase in plasma urea value at all, while several with only one or two fits showed a definite increase lasting for 2 to 4 days.

The mean plasma urea changes in 8 of the cases examined before and after the onset of fits are shown in Chart IX.; the mean plasma uric acid findings from the same cases are included for comparison. The rise in urea lagged behind the rise in uric acid; in many cases the maximum blood urea value was reached about 24 hours after the last fit. Very high urea values with a much delayed return to normal occurred in the same cases in which the high uric acid values persisted for several days and were related to

a period of oliguria.

The Effect of Labour.

It has been found to be impossible to demonstrate clearly that labour causes changes in plasma urea similar to those already shown to occur in the plasma uric acid. The reasons for this are, that the changes of plasma urea following convulsions are irregular and tend to be prolonged, thus frequently obscuring the effect of labour; and also, that any rise in plasma urea which is caused by labour occurs later than the uric acid rise and is small if the labour is short. In a few of the cases of the present series, however, in which a week or more elapsed between the fits and labour, the findings indicate that changes of plasma urea, due to labour, occur as in normal cases.

The Effect of Convulsions on Plasma Uric Acid and Urea in Non-Pregnant Patients and in Animals.

The occurrence of the uric acid rise immediately after the first eclamptic fit, and the proportion between the height of the rise and the number of fits, suggests that the rise is a direct consequence of the fits. In order to investigate this point, the uric acid findings in a series of 10 patients (Cases 146 to 155) receiving "convulsion therapy" for mental disorders, were studied. Most of the patients were women between the ages of 20 and 40 years, who were suffering from schizophrenia or manic-depressive psychosis but were physically healthy. The convulsant drug used was Azoman, a proprietary preparation similar to Cardiazol. It is given intravenously; the latent period between the injection and the convulsion is very short (5 seconds to 10 minutes).

For the present study 4 blood samples were taken for examination; one immediately before the injection of Azoman and the others at half-an-hour, four hours, and twenty four hours after the fit. Plasma uric acid and urea were estimated in all samples. Two of the patients (Cases 154 and 155) did not convulse after the injection, due to insufficient dosage or acquired tolerance to the drug; these two serve as controls for the action of the drug itself. The results are shown in Table V.

CHART X.

Graph showing Changes in Plasma Uric Acid after Azomon Convulsions.

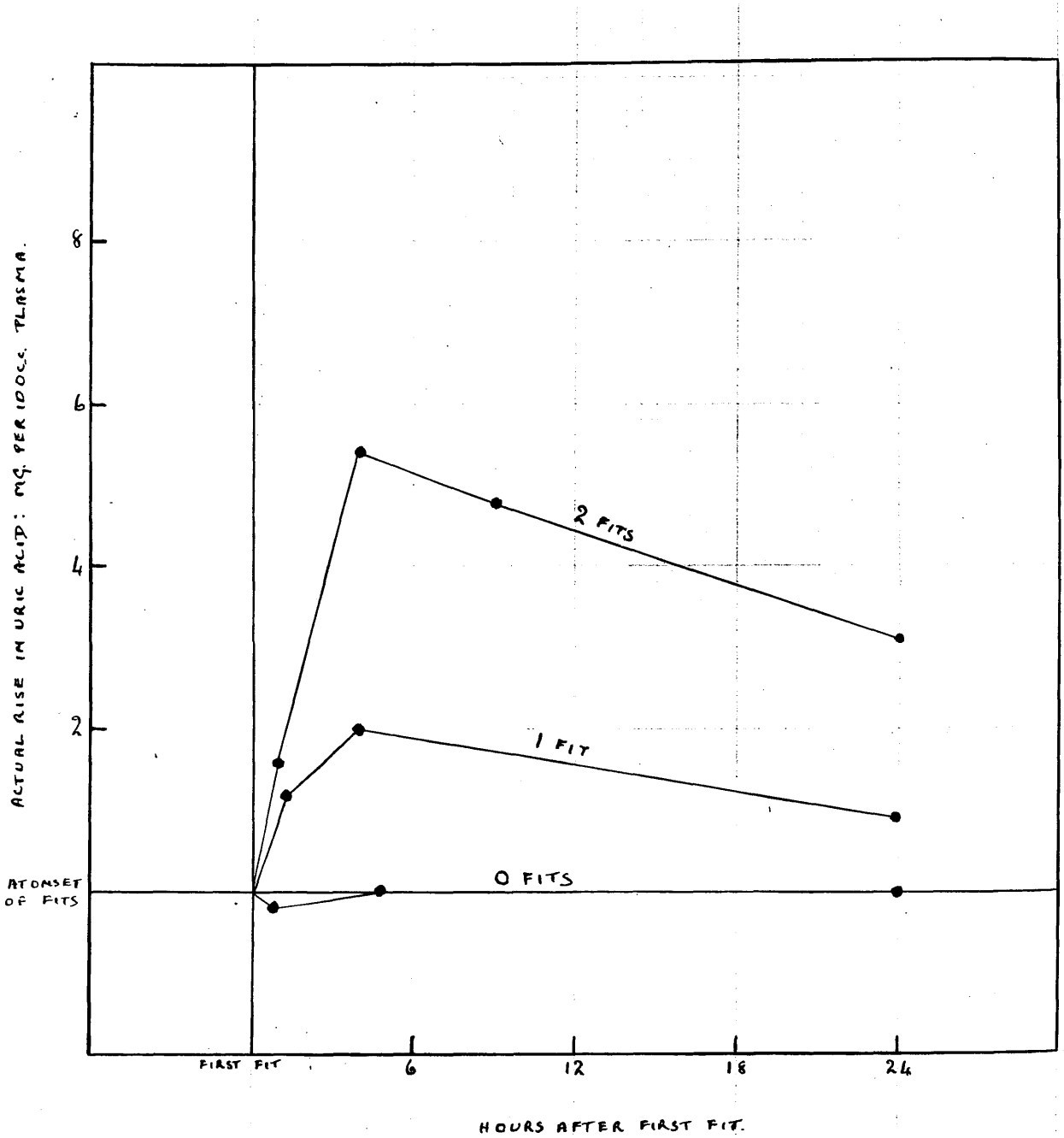


Table V.

Mean Uric Acid and Urea Changes after Azoman Convulsions in Non-Pregnant Patients.

Number of Fits.	Uric Acid. Mg. per 100 c.c.				Urea. Mg. per 100 c.c.			
	Before Fits.	After Fits. ½ hr.	4 hrs.	24 hrs	Before Fits.	After Fits. ½ hr.	4 hrs.	24 hrs.
0	3.8	3.5	3.8	-	28	24	25	-
1	3.2	4.3	5.3	4.1	22	22	23	27
2	3.3	4.9	8.7	6.4	25	26	25	37

In Chart X. the changes in the plasma uric acid following the fit are plotted as in Chart VI. In every case after the convulsion there was an immediate rise in plasma uric acid. The rise continued for 3 to 4 hours, after which time the values slowly returned to normal, but, at 24 hours after the fit they were still above the original value in all cases. In one case (Case 146) the patient had two severe convulsions; it will be seen from the table and Chart that the resultant rise in uric acid was much greater than in the cases with only one convulsion. This is in agreement with the findings in eclampsia; but it will be noticed that the rise in plasma uric acid in the Azoman cases is even greater than in the eclamptics. In the two cases without convulsions the plasma uric acid did not show any change, indicating that the rise in uric acid in the

other cases was due to the convulsion and not to the drug itself.

In all cases the plasma urea values remained practically constant for a long time after the fits, but, sometimes the values obtained 24 hours after the convulsion showed a slight rise. This finding is very similar to the changes in eclamptic patients.

The change in plasma uric acid after these artificially produced convulsions was also studied in 14 rabbits; an injection of the appropriate dose of Azoman was given intravenously and a typical convulsion with tonic and elonic phases occurred within a few seconds. In every case there was a rise in plasma uric acid soon after the convulsion. The mean results are shown in Table VI.

Table VI.

Rise in Plasma Uric Acid due to Azoman Convulsions in Rabbits.

	Plasma Uric Acid: Mg. per 100 c.c.
Before Injection.	0.5
½ hour after fit.	2.7
2 hours after fit.	3.1
4 hours after fit.	1.7

It was found that if the procedure was repeated on one rabbit on successive days, the uric acid rise following a convulsion became less. The patients receiving "convulsion therapy" were made to convulse every third day, but a

reduction in the uric acid rise was not noted in these patients, even after several weeks of this treatment.

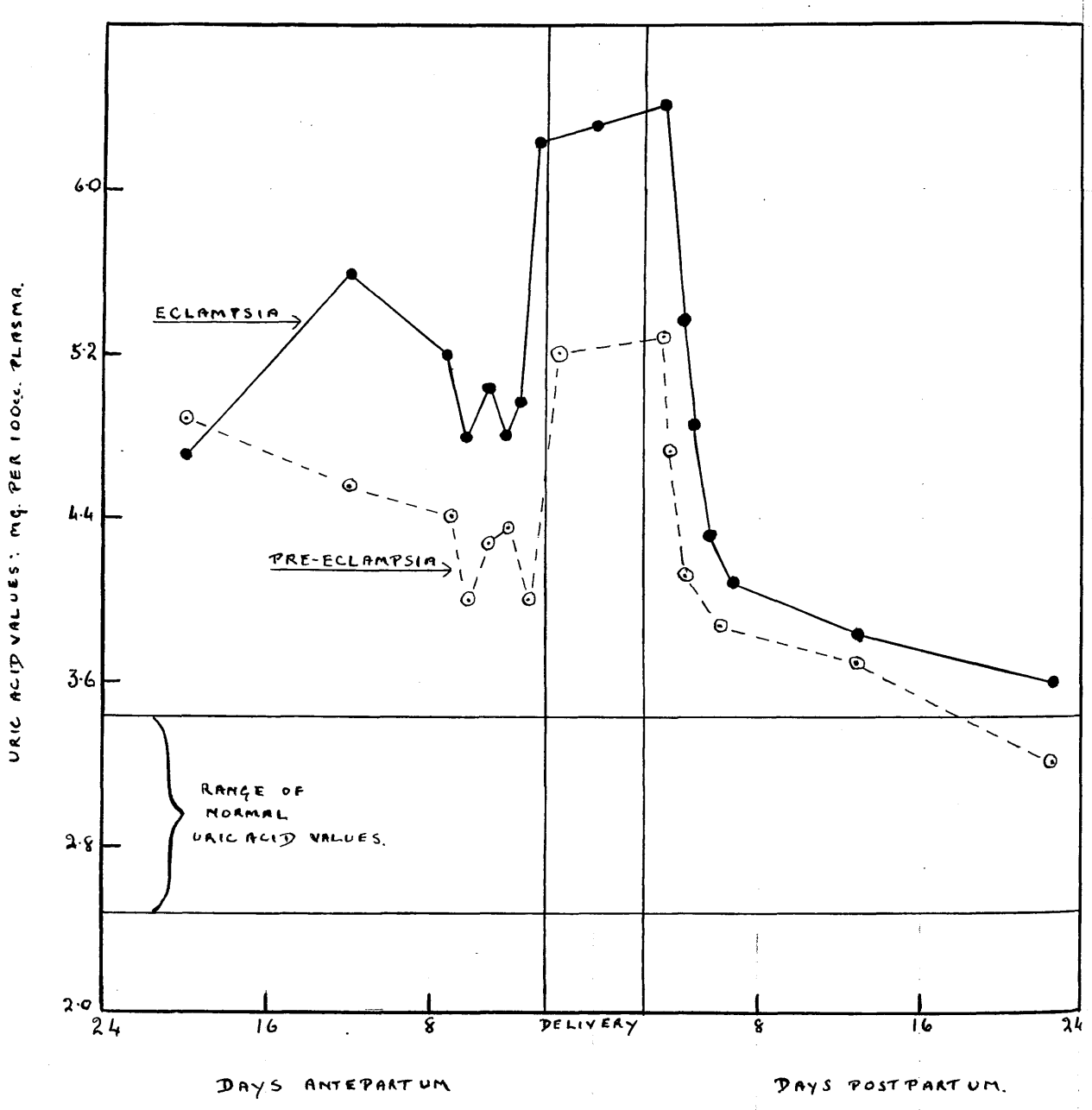
The results from this series of cases indicate that non-eclamptic convulsions cause a rapid and marked rise in plasma uric acid though they cause little immediate change in the plasma urea level. This gives further support to the view that the high uric acid values, found in eclamptic patients some hours after the onset of fits, have been caused in great part by the convulsions per se. Wuth (1926) studied the blood changes in epileptic cases after convulsions; his estimations were made once or twice on each patient but only at intervals of 24 hours or more. His findings show a rise in the non-protein nitrogen, uric acid and creatinine values following epileptic convulsions. This is presumably a similar effect to that in eclampsia or in Azoman convulsions.

Discussion.

The plasma uric acid in eclamptic patients is influenced by three factors - labour, preceding hypertensive toxaemia and convulsions. Labour causes the same changes in plasma uric acid in these cases as in normal cases, though the rise due to this cause in eclamptic cases, is often small because the labour is usually short. The uric acid value before the onset of fits and labour may show a slight relation to the clinical severity of the toxaemia, but it is often surprisingly low. By far the most important factor in causing a high uric acid value in eclamptic cases is the

CHART XI.

Average Uric Acid Values in 110 Eclamptic and 38 Pre-eclamptic Cases. (Stander and Cadden, 1934 b.)



very rapid increase which is caused by the actual convulsions; the extent of this increase is related to the number of convulsions.

High values for uric acid in eclampsia have been reported by various workers (Williams, 1921; Caldwell and Lye, 1921; Killian and Sherwin, 1921; Hellmuth, 1923; Heynemann, 1924; Plass, 1924; Stander, 1924; Stander, Duncan and Sisson, 1925; Bokelmann and Rother, 1925; Stander and Radelet, 1926; Cruickshank, Hewitt and Couper, 1927; Stander and Cadden, 1934). In most cases these can be sufficiently explained by the fact that the determinations were always made some time after the onset of fits.

Stander and Cadden (1934b), state that, the high uric acid values in eclamptic patients are out of all proportion to the slight changes in the other nitrogenous constituents of the blood and are present before the outbreak of convulsions; they give figures to support this statement, however, not from eclamptic patients before convulsions but from pre-eclamptic patients who did not develop convulsions. They show a chart of the "average uric acid values in 110 eclamptic and 38 pre-eclamptic cases" (see Chart XI.) and state that they are unable to explain the depression in the curves shortly before delivery, but that this may prove of significance when more is known about purine metabolism. It seems obvious from the present results that this depression is an antefact due to the inclusion of both ante-partum and intra-partum eclamptic cases; the uric acid values in ante-partum eclamptics, recovering from fits, fall

fall before the onset of labour and the values in intra-partum eclamptics before labour are low. The great increase in uric acid values seen during labour is due to the normal rise due to labour in both ante-partum and intra-partum cases, together with the rise due to fits in the intra-partum cases.

Cadden and Stander (1939), in a further investigation of the biochemical findings in eclampsia, give tables showing the finding "before, during and after convulsions." Their figures show highest uric acid values during convulsions but the estimations were not made frequently enough to show the definite relation between the uric acid values and the convulsions. They found no impairment of uric acid excretion in their cases and concluded that the high values were not due to renal insuffiency but were probably the result of diminished uric acid destruction in the liver. In the present series the great rapidity of the rise in plasma uric acid in all cases, without any concomitant rapid rise in the blood urea, suggests a similar conclusion, i.e. that the rise is due to some increase in uric acid production or diminished uric acid destruction rather than to a renal retention. Some eclamptic patients do show evidence of impaired renal function (oliguria, rising blood urea and persistence of high uric acid values) after the convulsive period (often for 24 to 48 hours after the last fit), and this evidence persists until a good urinary output is established. This renal impairment appears late in the disease and only in some of the cases. Stander and Cadden (1934b) and

Frey (1923), record similar findings. It is of interest to note that Dunn and Polson (1926) obtained a marked rise in blood urea in rabbits after the intravenous injection of very large amounts of uric acid. This was, however, due to a specific damage to the kidney; there is little evidence that the relatively low uric acid levels in eclampsia have any comparable effect on the human kidney.

The present work does not include any direct attempt to ascertain the cause of the uric acid rise after convulsions. It has been shown by various workers that, after convulsions, there is an increase in blood sugar, (Maclean, 1936; Titus, Willetts and Lightbody, 1930), blood lactic acid (Stander and Radelet, 1926), and inorganic phosphate (Stander, Duncan and Sisson, 1925); these blood fractions are all products of muscle metabolism and are produced, presumably, by the muscular effort of the convulsion. Burian and Schnur (1910) have shown that uric acid is formed by the breakdown of muscle hypoxanthine and it is presumably also produced by the actual muscular exertion during a convulsion. In a healthy subject, however, Folin, Berglund and Derrick (1924) have shown that any excess of uric acid is either excreted or rapidly destroyed in the body. The site of uric acid destruction in the dog has been shown to be the liver (Bollman, Mann and Magath, 1925; Gremels and Bodo, 1926), and it is possible that some uric acid may be destroyed normally by the human liver. It may, therefore, be assumed tentatively that the rise of plasma uric acid after eclamptic convulsions

is due to one or both of these factors: increased uric acid production by the muscular exertion, and diminished destruction of uric acid by the liver.

Summary.

The changes in plasma uric acid and urea occurring before, during and after convulsions in labour have been studied in a series of eclamptic patients. A rise of plasma uric acid was found to occur in every case immediately after the onset of fits, the height of the rise being dependent on the number of fits. Plasma urea values showed little immediate change after fits but a rise was often noted 24 or more hours later. Similar findings were obtained after artificially produced convulsions in non-pregnant patients and rabbits.

In eclamptic patients labour causes the same changes in plasma uric acid as in normal cases.

SECTION IV.

CHANGES IN BLOOD CONCENTRATION IN NORMAL AND TOXAEMIC PREGNANCY.

The occurrence of hydraemia during pregnancy has been known for many years, and has been shown by many workers using a variety of methods (Nasse, 1876; Meyer, 1887; Regnault, 1904; Zangemeister, 1904; Zunst, 1911; Miller, Keith and Rowntree, 1915; Mahnert, 1921; Plass and Bogert, 1924; Plass and Matthews, 1926; Schoenholz, 1929; Skajaa, 1929; Dieckmann and Wegner, 1934a; Oberst and Plass, 1935 and 1936; and Thomson, Hirsheimer, Gibson and Evans, 1938). More recently it has been shown that, in the hypertensive toxaemias of late pregnancy, the normal hydraemia is often absent and the blood may be abnormally concentrated. Rapid changes in the concentration of the blood have been shown to occur in these patients especially about the time of delivery. These rapid changes in concentration cannot be measured by direct estimations of blood volume, as these methods involve injections of dyes and cannot be repeated at short intervals of time in the same patient. Most authors are agreed that, over short periods of time, changes in the proportion of cells to plasma, as shown by rise and fall in blood-haemoglobin, red-blood cell count and haematocrit reading, may, in the

absence of haemorrhage, be taken as a fairly accurate index of variation in total blood or plasma volumes, produced by changes in the water content of the plasma. Of these methods, the haematocrit has been used most frequently in the study of these toxaemic patients.

Skajaa (1929), Schwartz and Dieckmann (1929), and Dieckmann (1933) investigated haemoglobin and haematocrit at various intervals before and after delivery in pre-eclamptic patients. They found raised values before delivery indicating a reduction of blood volume due to concentration of blood, and a great fall in values during the first few days of the puerperium, indicating an increase in blood volume due to dilution of blood at that time.

Skajaa (1929), and Schwartz and Dieckmann (1929) emphasized the relation between the blood concentration and the toxaemia and concluded that the marked post-partum dilution was due to the abatement of the toxaemia. In a more detailed study, Dieckmann (1933) found that the post-partum dilution of the blood occurred in pre-eclamptic cases but not in chronic toxaemic cases and suggested that this fact might be used in differentiating the two types of cases. Skajaa (1929) and Dieckmann (1933) were unable to find any correlation between post-partum blood dilution and the amount of oedema present. On the other hand, Plass and Bogert (1924) in an investigation of plasma protein variations in normal and toxaemic patients found plasma dilution after

delivery in both types of patients but more marked in the toxaemic ones. They noted that the dilution coincided with the disappearance of oedema, and concluded that the dilution was due to the oedema fluid entering the blood-stream. They suggested that the extent of the dilution varied with the extent of the oedema but they did not attempt to prove this.

A detailed study does not appear to have been made of the changes in blood concentration in normal cases about the time of delivery. Denecke, from a review of the literature in 1924, stated that a reduction of blood volume may be found during labour in normal cases. Payer (1904), Carton (1924), and Horvath (1924) have shown that in capillary blood an increase of a half to one million in the red-blood cell count may occur during labour with a return to normal in the puerperium; Eckelt (1919) noted similar changes in the plasma proteins in normal cases. This increase was thought to be due to the muscular effort and increased abdominal pressure during labour causing more blood to be driven into the peripheral circulation. The changes after delivery were attributed to blood loss and the filling up of the splanchnic circulation. Dieckmann and Wegnel (1934a) studied changes in blood and plasma volumes in a large series of normal patients, but only four patients were examined frequently during labour and the early puerperium. In these four patients, changes in haematocrit values were found indicating concentration of the blood during labour and dilution afterwards. Oberst and Plass (1936) found increased

concentration of plasma and cells early in labour and a further increase in concentration at delivery. Skajaa (1929), however, found little or no change in haematocrit values in his normal cases; they were, however, not examined at frequent regular intervals.

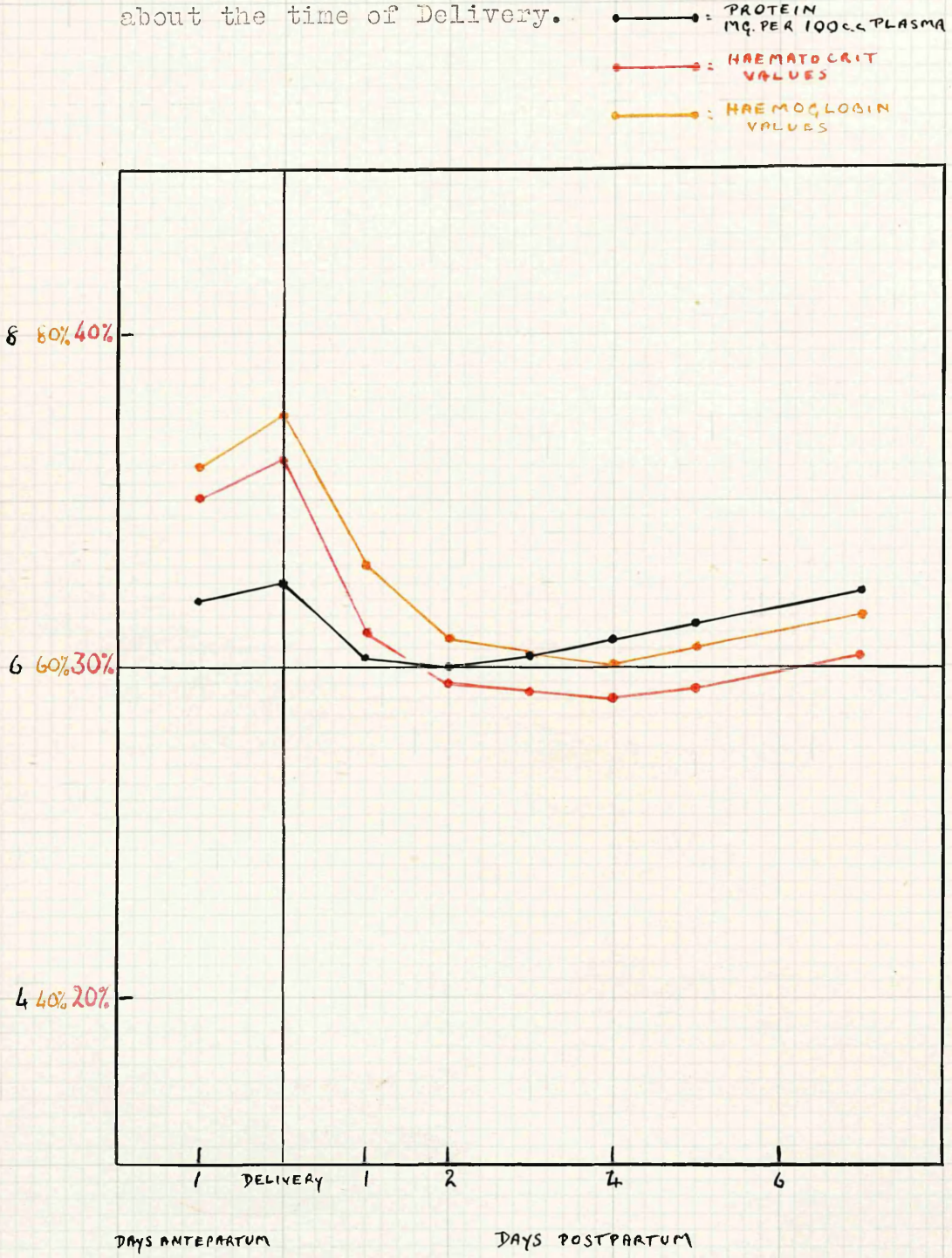
The present paper is a detailed study of the changes which occur in the red-cell volume, estimated by the haematocrit, in normal and toxaemic patients about the time of delivery and in the puerperium. Rapid variations in haematocrit may, theoretically, be due to alterations in the total number of red cells in the body, as after haemorrhage or transfusion; or to changes in size of the individual red cells, due to osmotic changes in blood; or to changes in the number of red cells per cubic centimetre of blood, due to an increase or decrease in the absolute amount of water in the plasma.

In a small preliminary series, accurate haemoglobin estimations were made along with the haematocrit determinations and it was found that changes were absolutely parallel in individual patients, i.e. the relation between blood-haemoglobin and the total cell volume was constant except for insignificant variations within the range of experimental error. This constancy of the volume index indicates that the changes in haematocrit values are not due to swelling or shrinkage of red cells. The actual size of red cells in pregnancy has been shown by Dieckmann and Wegner (1934b) to be slightly larger than normal, but this does not affect the point at issue.

Changes in the haematocrit values due to changes in

CHART XII.

Changes in Haematocrit, Haemoglobin and Plasma Proteins about the time of Delivery.



the total number of red cells in the body do occur in certain cases, but these cases can be easily recognised because they are due to easily ascertained factors. A loss of red cells may be due to haemorrhage which is noted clinically or to haemolysis which is shown by bilirubin estimations. A rapid increase in red cells may be due to blood transfusion, and the volume of this is known. Regeneration of red cells is a slower process and only plays a part in gradual changes in haematocrit. Patients in whom the total number of red cells in the body is altered suddenly can, therefore, be considered separately from the others.

The cause of the changes in the haematocrit is the dilution or concentration of the blood due to the passage of water from the tissues to plasma or vice-versa. This water naturally carries with it diffusable electrolytes and a certain amount of protein. In the present series plasma protein estimations were made daily for several days before and after delivery in 10 patients (Cases 24, 25, 47, 51, 79, 81, 93, 94, 95 and 115). The mean results for plasma protein, haemoglobin, and haematocrit from these patients are plotted in Chart XII. to show the changes about the time of delivery. The haemoglobin and haematocrit curves run absolutely parallel as has been noted earlier. The plasma protein changes are similar to the haematocrit changes, but the rise and fall of the curve is less in the case of the proteins because the fluid entering and leaving the blood-stream carries a certain amount of

protein with it. The conclusion that the changes of the haematocrit are due to the transfer of fluid from the blood to the tissues and vice-versa is in agreement with the findings of Oberst and Plass (1935 and 1936) who estimated changes in cell volume, specific gravity, and water content of the blood, haemoglobin and plasma proteins in pregnant women at term, during delivery, and in the puerperium, and found corresponding changes in all fractions.

Present Technique.

In the present investigation venous blood was drawn from the arm without any obstruction to the blood flow. Haematocrit readings were made using a standard technique, and care was taken that all details were the same in every case. The haematocrit values are recorded as the percentage of the volume of the whole blood which consists of cells.

The normal haematocrit findings with this technique were as follows:

Normal non-pregnant female, 42; corresponding to haemoglobin of 84 per cent.

Normal pregnant female at 39 weeks, 33; corresponding to haemoglobin of 68 per cent.

The series studied throughout delivery consisted of 96 cases which have been classified as follows:

20 normal cases; 56 pre-eclamptic cases;

20 chronic toxaemic cases.

Patients with any convulsions, eclamptic, epileptic or hysterical have been excluded from the present series.

Haematocrit readings were made about 7 days ante-

partum, near the onset of labour and late in labour, and daily during the first 3 to 5 days of the puerperium, after which readings were made at intervals of 2 to 3 days till the patient's dismissal. In many of the toxaemic cases readings were made at 1-day or 2-day intervals during the last few weeks ante-partum, and in many of the normal and toxaemic cases the blood was examined at intervals of a few hours during labour and the first day after delivery. Only the mean figures are given in this section; the individual cases showed the usual scatter, but the variations in each individual case follow quite consistently the mean figures for the group.

Ante-Partum Findings.

The mean haematocrit values for all cases in each group are shown in the following table:

Table VII.

	Normal.		Pre-eclamptic.		Chronic toxaemic.	
	Range.	Mean.	Range.	Mean.	Range.	Mean.
7 days ante-partum.	29 to 37.	33	24 to 42.	35	20 to 44.	33
At onset of labour.	29 to 39.	36	25 to 43.	36	22 to 45.	34

Seven days ante-partum the mean value for pre-eclamptic cases is slightly higher than that of the normal, while the mean

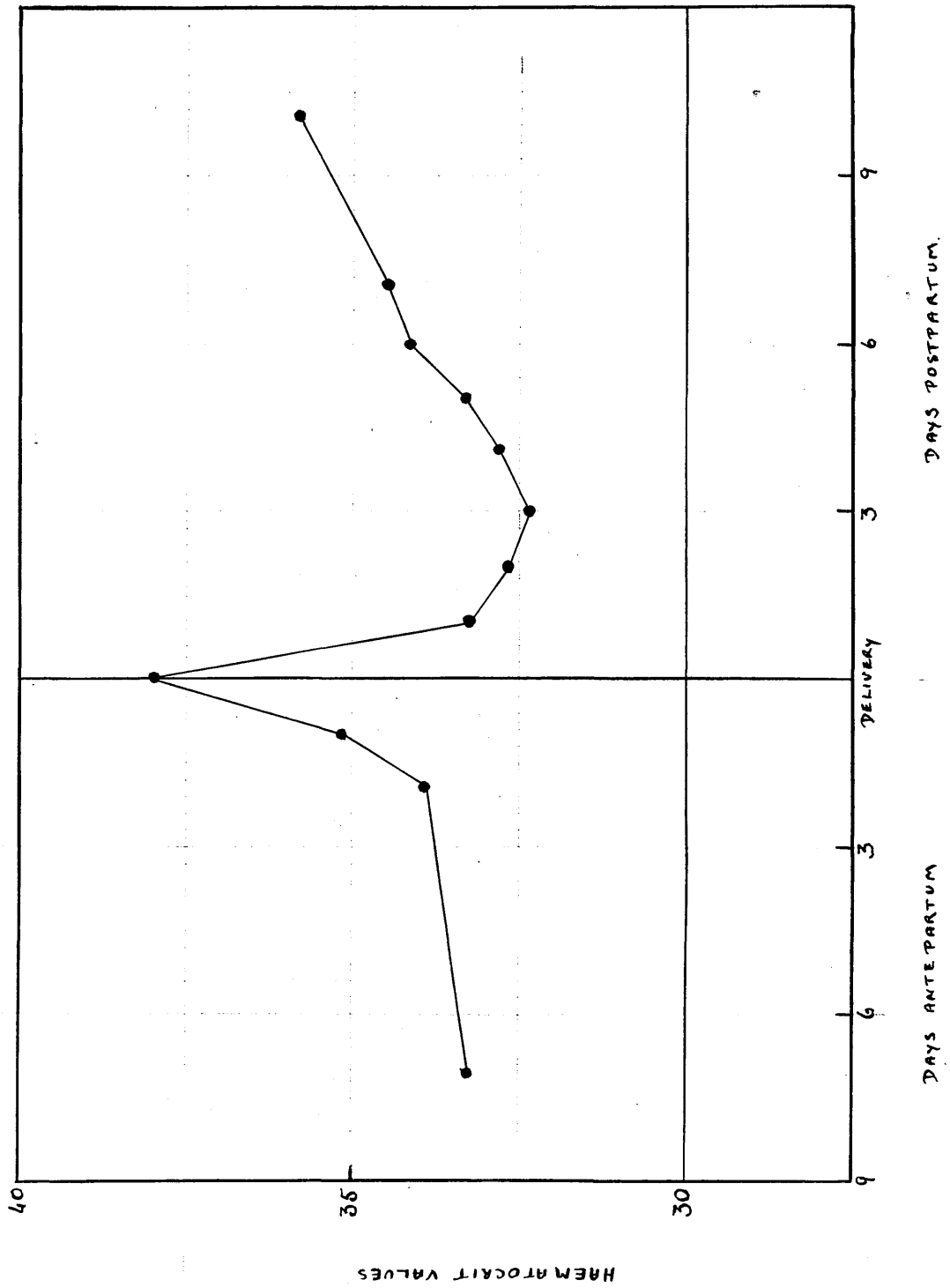
value for the chronic toxaemic cases is about normal. The range of the values in both the pre-eclamptic and chronic toxaemic groups is considerably greater than in the normal group. The significance of these findings will be discussed later with the findings during labour and post-partum. It will be noticed that the mean values for each group show that the blood becomes rather more concentrated during the week before the onset of labour. In the pre-eclamptic cases studied during the last few weeks ante-partum it was found that the haematocrit values varied from day to day. With very few exceptions a rise in haematocrit was found to accompany increased severity of the toxaemic signs while a fall in haematocrit accompanied clinical improvement. (This is in agreement with the work of Skajaa (1929), Dieckmann (1933), and Schwartz and Dieckmann (1929). Similar examinations were made in only a few normal cases but in these the value was more constant, showing only a gradual rise before the onset of labour.

Changes during Labour and the Puerperium.

In all cases, normal and toxaemic, the haematocrit figures showed a rise during labour followed by a fall during the first 3 days of the puerperium. The rise and fall varied in extent from case to case but were invariably present. In most patients the values increased steadily during labour, reaching a maximum at delivery or a few hours post-partum, after which they began to fall. In a few cases the values

CHART XIII.

Mean Haematocrit Changes before and during Labour, and throughout the Puerperium, in normal cases.



remained high or even increased 6 to 12 hours after delivery, and then fell, while in other cases the haematocrit value reached a maximum early in labour, and showed a slight decrease before delivery, after which the definite post-partum fall began. It will be shown later, in Section VII., that a delayed fall in haematocrit values after delivery often accompanies signs of slight post-partum collapse. In particular the extent of the rise during labour was found to be unrelated to the length or severity of the labour. In the following charts, the maximal value obtained on the day of delivery has been used as the figure for delivery. The mean haematocrit values of 10 normal cases (patients who were delivered by forceps or who had had haemorrhage or were infected being excluded) from 7 days ante-partum till 10 days post-partum are shown in Chart XIII. The curve shows a rise in values from 7 days ante-partum and reaches a maximum at delivery. The haematocrit values decrease rapidly during the 1st day post-partum and the fall continues more slowly till the 3rd day post-partum when the values are definitely below the original ante-partum level. After the 3rd day post-partum, the curve begins to rise steadily, approaching normal non-pregnant levels by the 10th day post-partum. The post-partum dilution is definitely not due to blood loss; it appears to be an over-compensatiob for the increase in blood concentration which occurred before delivery.

Several factors were found to influence the extent

of the post-partum dilution, and these will be dealt with before a comparison is made between the different types of cases.

Factors Influencing Post-Partum Dilution.

A greater post-partum fall in the haematocrit values was found to be produced by three factors: haemorrhage, delivery by forceps, and infection. Normal and toxaemic cases were affected alike, and in order to demonstrate these changes all the cases from the series have been divided into four groups.

(a) 57 patients delivered spontaneously without haemorrhage or with only insignificant bleeding and without any subsequent infection.

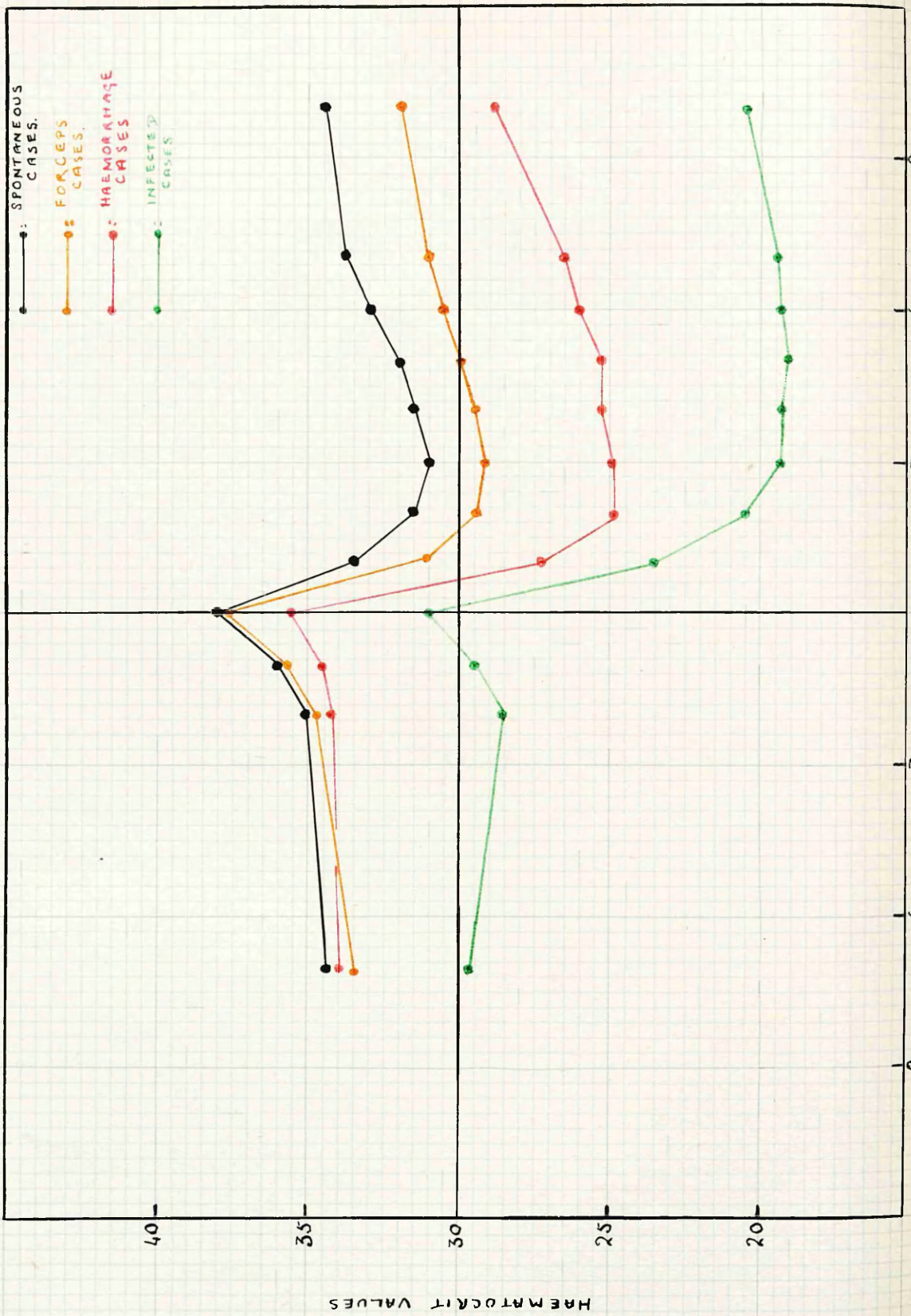
(b) 7 patients with moderate or severe haemorrhage at delivery. No exact measurements of haemorrhage were made; the amount in every case was estimated clinically as none, slight (about 200 c.c.), moderate, or severe. Haemorrhage less than 200 c.c. was considered insignificant.

(c) 21 patients delivered by forceps without haemorrhage or infection.

(d) All the patients, 11 in number, in whom there was any elevation of temperature during the puerperium, excluding slight reactionary temperatures on the first day post-partum.

CHART XIV.

Mean Haematocrit Values in Spontaneous, Forceps, Haemorrhage, and Infected Cases.



The mean haematocrit values for these four groups have been plotted in Chart XIV. Patients delivered spontaneously, without haemorrhage or infection, show the smallest post-partum fall in haematocrit values. It is obvious that much haemorrhage at delivery will cause a marked post-partum fall as a result of actual loss of cells and the compensatory blood dilution which follows any loss of blood. The graph shows a much steeper post-partum fall in these haemorrhagic cases, chiefly within the first 24 hours; the minimal value being reached on the 3rd day post-partum. A definite rise in the haematocrit values begins after the 5th day, but the mean value is still well below normal at 10 days post-partum. The average loss in these cases is between 500 c.c. and 600 c.c. In theory, taking the blood volume as 5 litres and assuming that the volume is maintained by dilution after a haemorrhage, the loss of 550 c.c. would reduce the haematocrit by one-ninth, i.e. from 36 to 32. This is superimposed on the fall of 7 which occurs in spontaneous deliveries without haemorrhage. As a result of this combination of 4 + 7, the expected fall in these cases would be from 36 to 25, which is the actual drop seen in Chart XIV.

The curve from patients who were delivered by forceps shows a post-partum fall resembling that of the haemorrhage cases except that it is of lesser extent. In delivery by forceps, slight haemorrhage, as from an episiotomy, is often regarded as normal and left unnoted in the clinical records. Though every patient in whom even moderate haemorrhage was noted has

been excluded, it seems probable that the slight and unnoted haemorrhage occurring in patients delivered by forceps accounts for the greater fall in haematocrit figures in these cases, as compared with spontaneous deliveries. In order to exclude the possibility that the greater fall was due to the anaesthetic at delivery, a study was made of a series of patients in which an anaesthetic was given for various purposes 2 or 3 weeks before delivery. The only change found was a rise of about 1 in the haematocrit values for 24 to 48 hours after the anaesthetic. The anaesthetic at delivery thus played no part in the greater fall of haematocrit readings in the patients delivered by forceps.

The findings in the group of cases labelled infection are very striking. Of the chronic toxaemic cases, none of the patients became infected; all the infected cases are from the pre-eclamptic and normal groups. In 10 of the 11 cases the infection of the patient was mild, shown by slight irregular fever and offensive lochia; cervical swabs were negative for haemolytic streptococci and there was not any evidence of haemolysis as shown by the van den Bergh reaction on the plasma. In the remaining case (Case 53) the infection was due to haemolytic streptococci and a positive van den Bergh reaction in the plasma afforded definite evidence that haemolysis was occurring. The most important finding in these cases was the very low ante-partum haematocrit readings; indeed, three-quarters of the patients from the normal and pre-eclamptic groups, who had ante-partum values below 30, fell into this

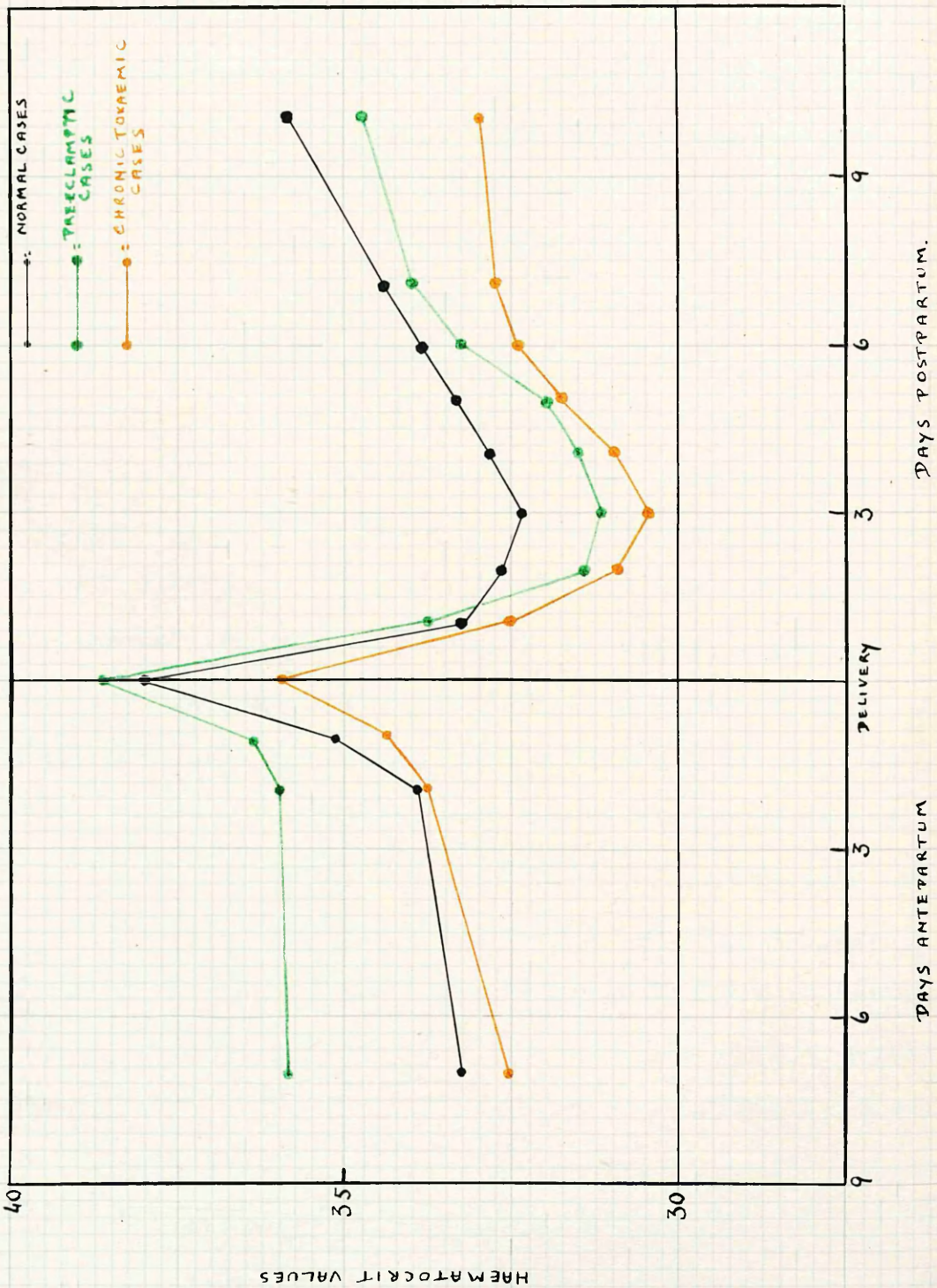
group, i.e. became infected in the puerperium. The graph for these cases shows a very marked post-partum fall and the fall continues longer than that of the haemorrhagic cases; the haematocrit readings do not reach their minimum until the 5th day post-partum. This association between ante-partum anaemia and post-partum infection appears to be of importance. Detailed bacteriological examination, except for the exclusion of streptococcus haemolyticus, was not made in these cases; the infection may be due to organisms which are normally held in check, but which become pathogenic to the patient after delivery because her resistance is lowered as a result of the previous anaemia. It is of interest to note that the only case of the group that had a normal ante-partum haematocrit value was the patient who was infected by haemolytic streptococci, presumably an exogenous infection. Although the infection in most cases was mild, it was usually prolonged, and it can be seen from the graph that the curve shows very little rise compared with that in the other groups of cases. Transfusion was resorted to in several of the patients, but, despite this, recovery was slow. Further investigation is necessary to ascertain whether a blood transfusion given ante-partum to these anaemic patients would reduce the liability to post-partum infection.

Changes in Normal and Toxaemic Cases.

In view of the effect of delivery by forceps,

CHART XV.

Mean Haematocrit Values in Normal, Pre-eclamptic and Chronic Toxaemic Cases.



haemorrhage and infection on the post-partum blood dilution, all patients with these complications have been excluded from the subsequent discussion. Only patients delivered spontaneously, without haemorrhage, or any puerperal infection are considered. In Chart XV. the mean haematocrit values for all these uncomplicated cases have been subdivided into normal, pre-eclamptic, and chronic toxaemic groups to show the changes occurring from 7 days ante-partum till 10 days post-partum. The normal curve has already been described. The mean graph for the pre-eclamptic cases, 31 in number, shows values 7 days ante-partum considerably higher than the normal cases, but the post-partum fall is rather greater, so that the curve falls well below the normal and remains low. The mean curve for the chronic toxaemic cases, 18 in number, shows a rather lesser rise and fall than the normal. This chart has been included only for comparison with the results of other workers. The present findings for the toxaemic cases are essentially similar to those results - pre-eclamptic patients having a concentrated blood for several days before delivery with a further increase in concentration on the day before delivery and a definite post-partum dilution; chronic toxaemic cases showing less marked changes. The extent of the changes in concentration in the normal cases is obviously of great significance in this connexion but its importance does not appear to have been sufficiently appreciated.

When the individual cases are analysed, the

CHART XVI.

Mean Haematocrit Values in Severe Pre-eclamptic Cases, Oedematous and Non-oedematous.

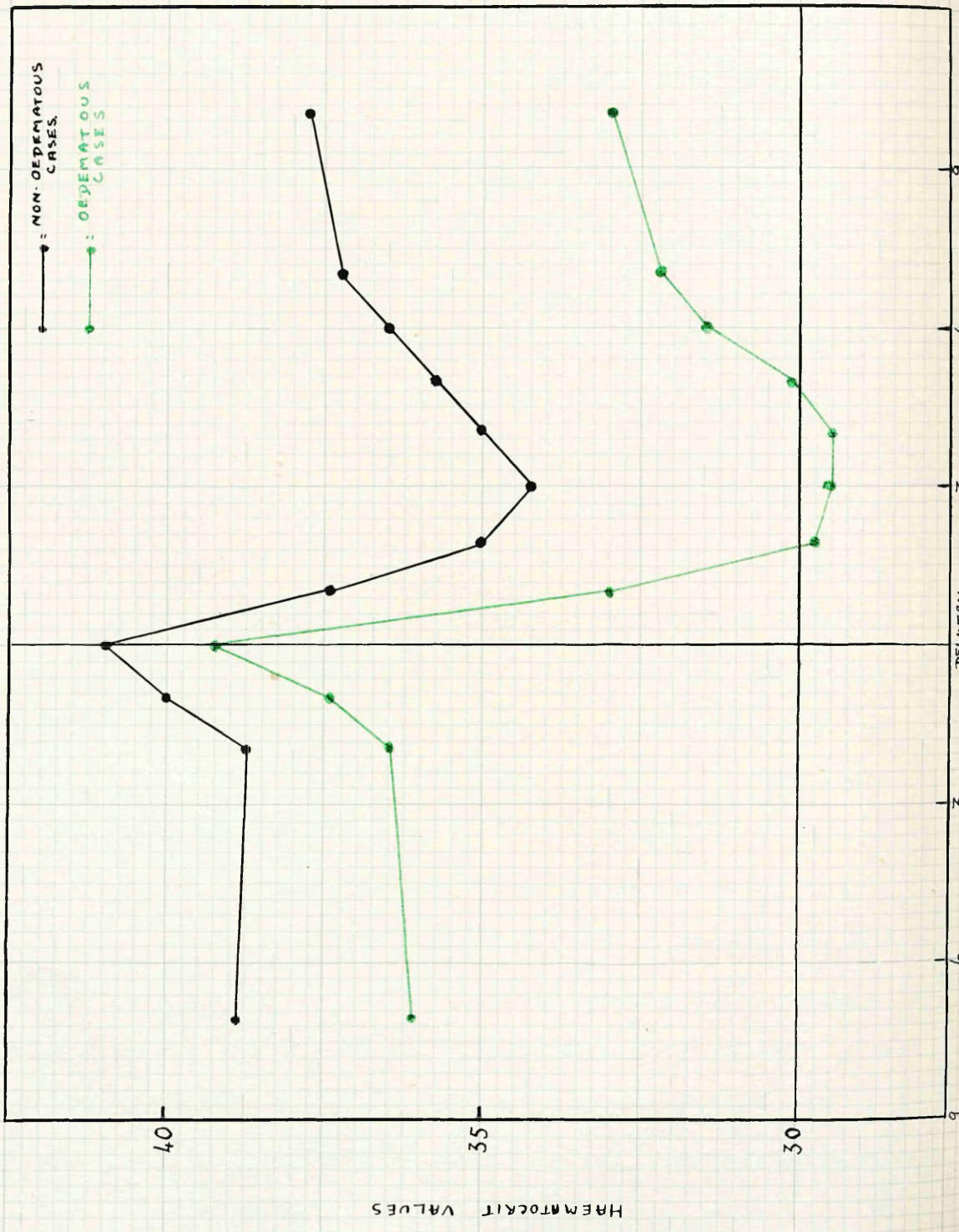
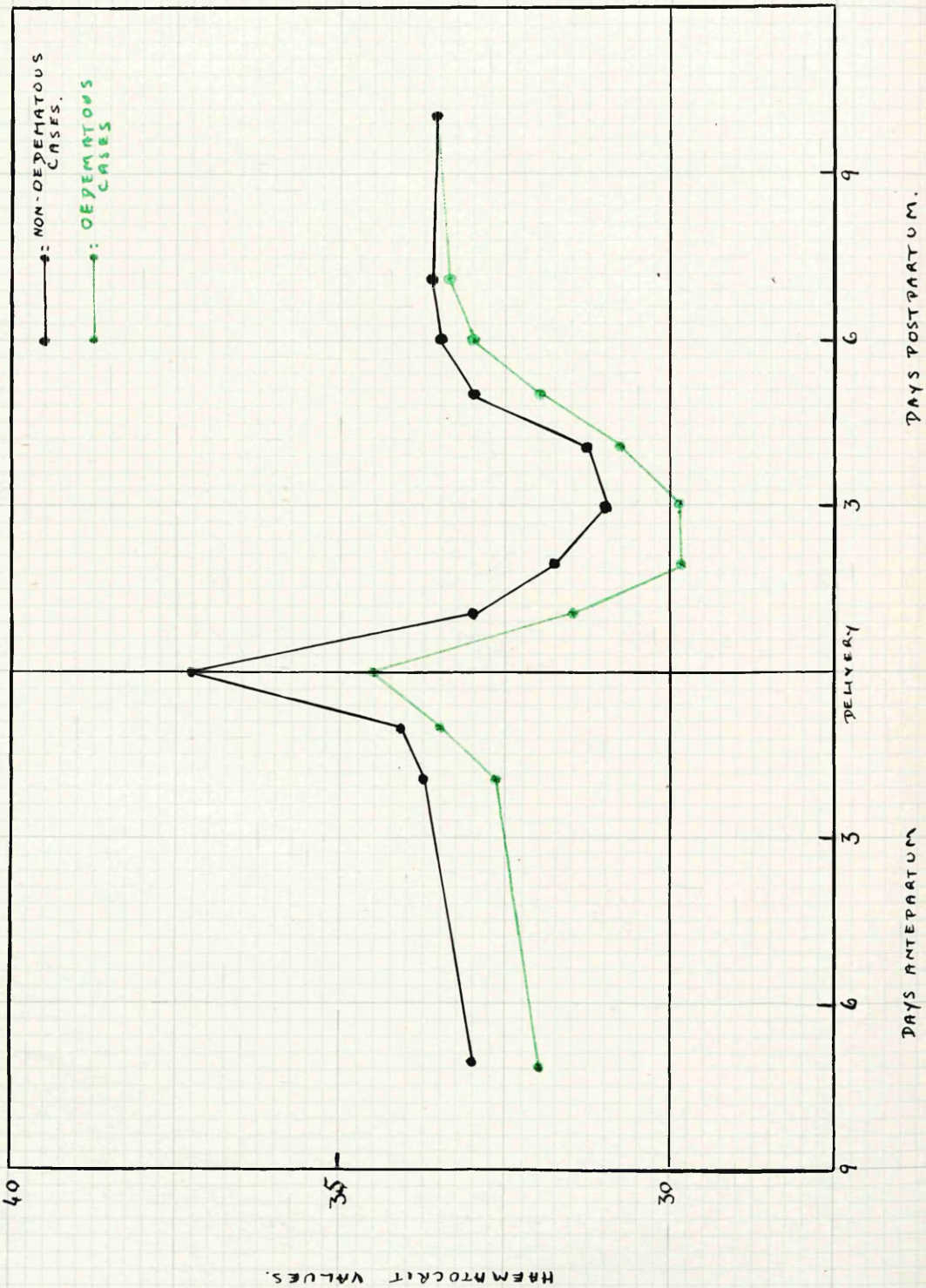


CHART XVII.

Mean Haematocrit Changes in Mild Pre-eclamptics



characteristics of the pre-eclamptic curve are found to depend on two factors; the severity of the toxaemia, and the presence or absence of oedema. These pre-eclamptic cases have been sub-divided from a clinical point of view into 20 severe and 11 mild cases. In the mild cases the patients had only slight hypertension, albuminuria or oedema and correspond to the low reserve kidney cases of American writers; the severe class includes all the patients with more serious symptoms. The results from the severe and mild pre-eclamptic cases are plotted in Charts XVI. and XVII. respectively.

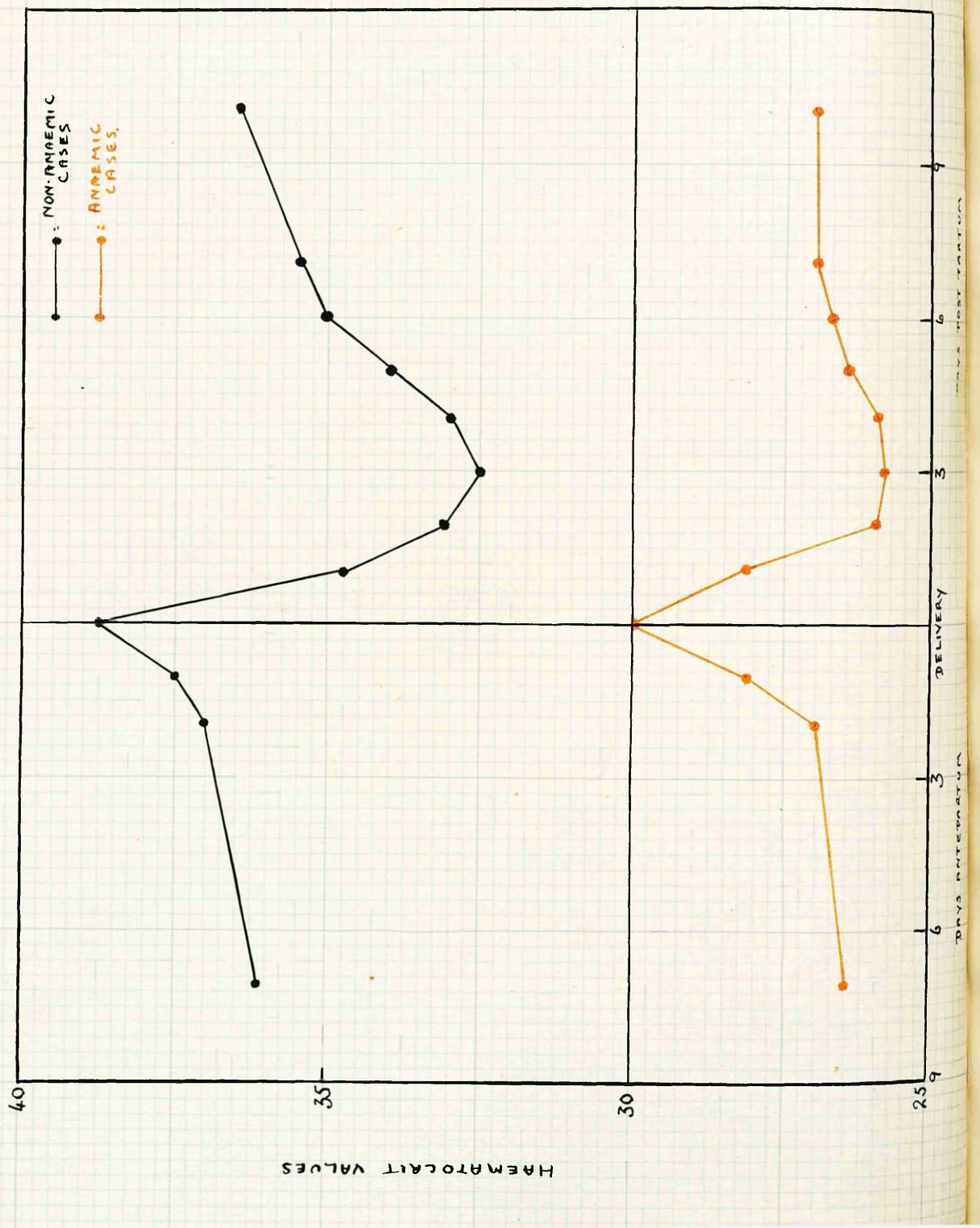
In each graph, the patients with clinically recognizable oedema have been separated from the others; account has not been taken of occult oedema. Oedema was present in 12 patients in the severe group and in 6 patients in the mild group. It will be noticed from each graph that the mean ante-partum values of the oedematous cases are lower than those of the non-oedematous cases; the less marked difference among the mild cases is probably because these patients had only slight oedema. A rise during labour occurs in all cases. The post-partum dilution is greater in the oedematous patients, especially in the severe pre-eclamptic, and these latter do not return to normal haematocrit levels by the end of the puerperium. It would appear that in those patients with oedema there is a relative hydraemia or an associated anaemia. This becomes more apparent after delivery; the very great post-partum fall is probably due to dilution of the blood by oedema fluid.

To exclude the influence of this factor, consideration may be limited to those patients without oedema. The mean value for the mild cases is almost the same as that for the normal. Those patients with severe pre-eclamptic symptoms have very high values 7 days ante-partum, but the rise in value before delivery is less than in normal cases and begins only 48 hours ante-partum. The post-partum fall in the severe cases is almost the same as that in normal cases although all values are at a higher level. This analysis shows, therefore, that, apart from the effect produced by oedema, the pre-eclamptic findings differ from those of the normal cases only in showing higher haematocrit values during the week or more before delivery. The increased post-partum dilution of the blood in the entire group of pre-eclamptic cases, which was shown in Chart XV., is therefore really due to the inclusion of oedematous cases. The wide ante-partum range of values in the pre-eclamptic cases, shown in page 51, is caused by the subnormal values of the oedematous mild cases and the high values of the non-oedematous severe cases.

When an analysis of the chronic toxaemic cases is made, the same influence of oedema is found, but there is another factor of importance. From a study of the ante-partum haematocrit values, the chronic toxaemia cases are found to fall into two well defined groups - 5 anaemic patients with haematocrit values between 20 and 30, and 11

CHART XVIII.

Mean Haematocrit Values in Chronic Toxaemic Cases, Anaemic and Non-anaemic.



non-anaemic patients with haematocrit values between 34 and 44. The terms "anaemic" and "non-anaemic" are used for convenience; the term "anaemic" in the present context is not intended to signify a reduction in the total haemoglobin in the body but merely a reduction in the amount per c.c. of blood. It is highly probable that the low haematocrit values in this "anaemic" group were actually the result of hydraemia. All the anaemic patients had marked oedema but only 3 of the non-anaemic patients had oedema.

The results from these two groups are shown in Chart XVIII. The curve from the non-anaemic cases closely resembles that of the non-oedematous severe pre-eclamptic cases, showing high ante-partum values, with slight pre-delivery rise and a normal post-partum fall. The findings in the anaemic cases are different from all the others. Concentration and dilution occur as in other groups but are of equal extent and there is no post-partum rise towards normal values; apart from the rise and fall in values during the 2 days before and after delivery, the haematocrit appears to remain unchanged in these cases.

Discussion.

The present results are in accordance with the findings of early workers who showed that during labour in normal cases, there was a rise in blood-haemoglobin and red blood-corpuscles followed by a fall in values in the

puerperium. In pre-eclamptic toxaemias, Skajaa (1929), Schwartz and Dieckmann (1929), and Dieckmann (1933) noted the high ante-partum haematocrit values, the rise during labour and the post-partum fall. The present series of results corroborates these findings but shows that the same changes, before, during and after labour, occur in all patients, toxaemic or otherwise. When the effect of various interfering factors such as haemorrhage, sepsis or oedema is excluded, the post-partum dilution of the blood is found to be approximately equal in all cases. Dieckmann's conclusion that chronic toxaemic cases do not show the pre-parturitional and post-parturitional changes is not in accordance with the present findings. The present results show, in contrast to the findings of certain other workers, that the presence of oedema does increase the post-partum dilution.

If the total plasma volume is calculated from the haematocrit results it is found that the changes described involve the transference of considerable volumes of fluid from the tissues to the blood stream and vice versa. During the last 7 days ante-partum, the mean haematocrit value for normal cases rises from 33.5 to 38.2; if the total blood volume in an individual case were 5 litres, of which 3¼ litres were plasma, this rise would be equivalent to the removal of 590 c.c. of fluid from the plasma. The post-partum fall in the haematocrit value in these cases would denote an increase of plasma fluid of 745 c.c. In the non-oedematous toxaemic

cases the post-partum increase in plasma volume is approximately the same: a mean of 782 c.c. in severe pre-eclamptic cases and of 747 c.c. in chronic toxaemic cases. In the chronic anaemic cases although the haematocrit changes appear slight, they signify the transference of almost the same quantities of fluid as in normal cases, plasma decreasing by 583 c.c. ante-partum, and increasing by 678 c.c. post-partum. In the oedematous pre-eclamptic cases, however, the changes are considerable; the mean plasma volume is increased by 1487 c.c. in the first 2 days post-partum.

The probable cause of the changes in blood concentration in normal cases is uncertain. The effect of muscular work, increased abdominal pressure and loss of water by perspiration during labour (as suggested by Denecke (1924), Payer (1904) and others) may undoubtedly cause an increase in blood concentration during labour but they do not satisfactorily explain the total increase in concentration before delivery; (a) more than half of the rise in haematocrit values occurs before the onset of labour, and (b) in several cases the haematocrit values begin to fall during the last few hours of labour when these factors are most marked. The post-partum dilution of the blood is much greater than the increase in concentration which occurs during labour, but it is almost equal to the total increase in concentration during the last 7 days ante-partum.

When the effect of all interfering factors is

excluded it is clear that the results from the pre-eclamptic and chronic non-anaemic toxaemic cases are very similar; they differ from the normal only in having higher haematocrit values at 7 days ante-partum and a smaller rise in values before delivery. This variation from the normal appears to be the direct result of the toxaemia since the blood in these patients becomes more concentrated than normal with the onset of the toxaemia. The post-partum dilution of the blood in both types of case is equal to the post-partum dilution in normal cases, and is presumably equal to the total increase in concentration which occurred ante-partum. It seems likely that the same ante-partum concentration occurs in these cases as in normal ones, but that it occurs earlier. So far as these findings are concerned, there does not appear to be any differance between the toxaemia of the pre-eclamptic and that of the chronic non-anaemic toxaemic patients.

The chronic anaemic toxaemic patients, however, appear to be quite unrelated to the other toxaemic patients both clinically and in their haematocrit findings. They are debilitated, pale and very oedematous, and do not show any improvement after delivery apart from the disappearance of oedema. Without direct estimations of blood volume it is impossible to decide whether the low haematocrit values in these patients are due to hydraemia or true anaemia. The same lack of improvement in severe anaemia is seen in multi-:parous patients with accidental haemorrhage though none of

present series had accidental haemorrhage.

Summary.

Definite increase in blood concentration, as indicated by changes in the haematocrit values, occurs in normal patients during the last 7 days, and particularly during the last 2 days, ante-partum; this is followed by a rapid blood dilution during the first 3 days post-partum. After this, the blood gradually returns to normal. The changes involve the transfer of 20 to 25 per cent of the fluid of the plasma from the blood into the tissues and back again.

Similar changes occur in patients suffering from pre-eclamptic toxaemia, but these patients have in addition a definite increase in blood concentration for one or more weeks before delivery. Patients with symptoms of chronic toxaemia fall into two groups, a series with anaemia (or hydraemia) which show the normal haematocrit changes about the time of delivery but do not show any rise in values during the puerperium, and a series without anaemia which closely resembles the pre-eclamptic cases in their haematocrit changes.

The post-partum dilution of the blood is increased in patients in whom there is oedema before delivery, haemorrhage at delivery or infection in the puerperium.

S E C T I O N V.

VOMITING IN PREGNANCY.

In many different conditions in pregnancy severe vomiting is the main feature; in some the vomiting appears to be the primary factor in causing the abnormalities characteristic of the disease, while in others, the vomiting is secondary to some other factor. At any time during pregnancy severe vomiting may be caused by an illness unrelated to the pregnancy, e.g. appendicitis, brain tumour etc., but the present section deals only with cases in which the vomiting was related to the pregnancy. The cases are dealt with under the following headings:

A) Vomiting of early pregnancy, or Hyperemesis Gravidarum.

B) Vomiting of late pregnancy.

Hyperemesis Gravidarum.

This term, hyperemesis gravidarum, is applied to the condition in which patients in the early months of pregnancy suffer from persistent and excessive vomiting. The vomiting is, in some way, caused by the pregnancy itself but the actual etiology is, at present, obscure.

Biochemical investigations of the blood in cases of hyperemesis have revealed certain abnormalities to be

constantly present in severe cases. Anhydraemia, resulting from the loss of water and salts, has been shown by Dieckmann and Crossen (1927) to be an important feature of the disease and these workers have emphasized the great value of intravenous saline and glucose transfusions in treatment. Raised blood non-protein nitrogen, urea and uric acid values are found by many workers (Williams, 1921; Killian and Sherwin, 1921; Stander, 1924; Dieckmann and Crossen, 1927; Peckham, 1929; Glassman, 1938; Herold, 1939). Jaundice is usually mentioned as a possible symptom in extremely severe cases and increased plasma bilirubin has been frequently reported (Heynemann, 1928; Herold, 1939; Fikentscher, 1939). Blood chlorides are usually decreased (Dieckmann and Crossen, 1927; Peckham, 1929) and there may be an accumulation of amino and lactic acids and acetone bodies in the blood. Most recent investigators are agreed that all these changes are due to the dehydration and undernourishment produced by the vomiting.

Biochemical studies have been made on several hyperemesis patients in the present investigation with regard to anhydraemia, non-protein nitrogen retention and hyperbilirubinaemia and the findings are in agreement with those of previous workers. The cases have been subdivided clinically according to the severity of their condition as, very severe, severe and moderately severe. No mild cases are included in this study. The results from 12 cases

(156 to 167) are shown in Table VIII.

Table VIII.

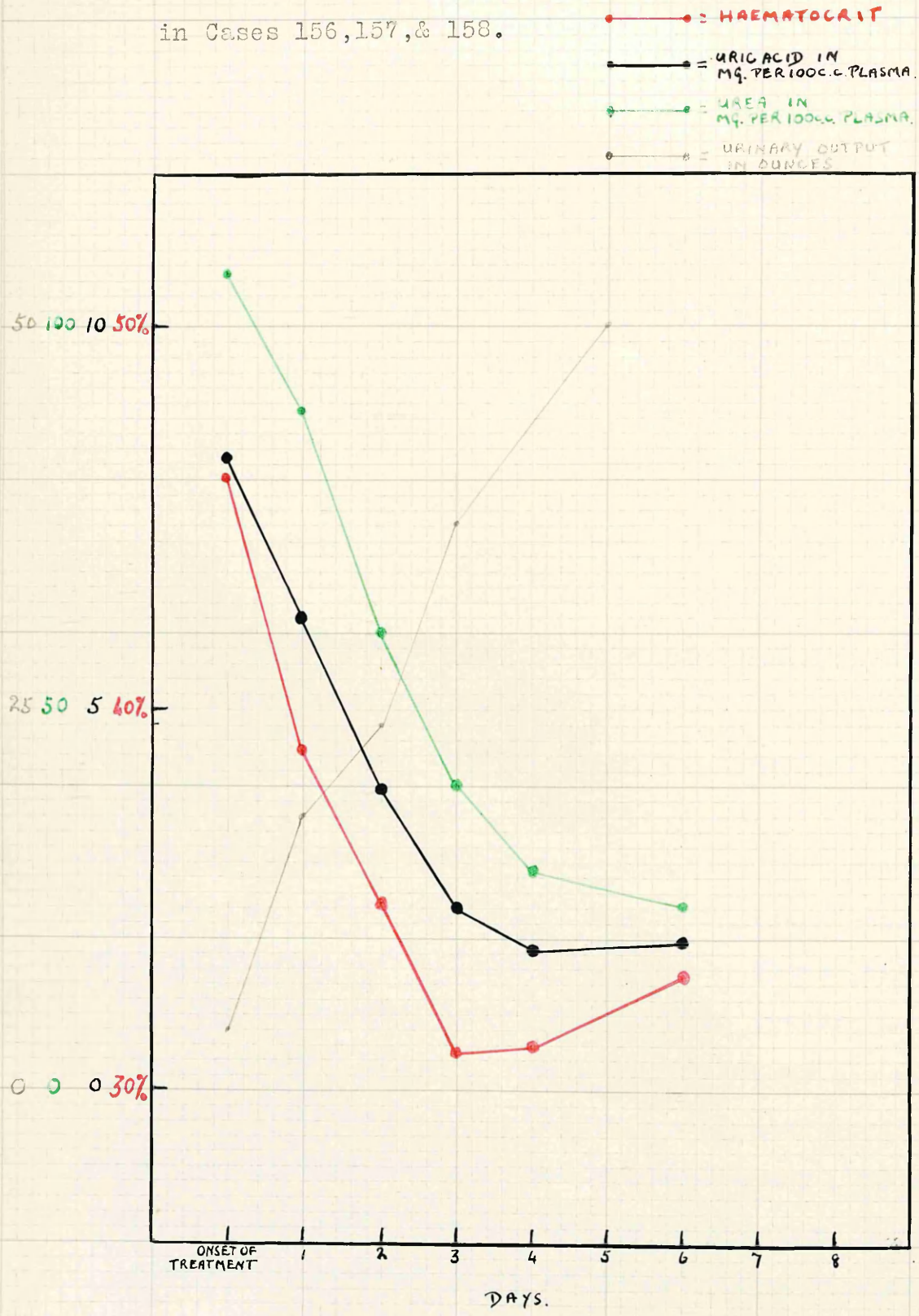
Mean Results from 12 Hyperemesis Cases.

	Haematocrit %		Uric Acid mg. per 100 c.c.		Urea, mg. per 100 c.c.		Bilirubin units.	
	Mean.	Range.	Mean	Range.	Mean	Range.	Mean.	Range.
Very Severe.	48.5%	45% to 52%	13.6	10.9 to 16.4	164	136 to 191.	3	1 to 6
Severe.	43%	37% to 50%	6.4	4.0 to 8.9	55	12 to 76	2	1 to 4
Moderately Severe.	40%	38% to 44%	3.1	2.2 to 4.3	22	16 to 27	1	-

Blood concentration, as measured by the haematocrit, was at the upper limit of normal in the moderately severe cases and greatly increased in the very severe cases. Nitrogen retention appeared to vary directly with the blood concentration in individual patients but there were variations between the haematocrit values and the degree of nitrogen retention from patient to patient. In one patient, case 164, the haematocrit value was 37% while the plasma uric acid was 8.9 mg. per 100 c.c. and the blood urea 40 mg. per 100 c.c.; in another patient, case 159, the haematocrit was 44%, uric acid 4.0 mg. per 100 c.c. and urea 12 mg. per 100 c.c. These findings are presumably partly due to the differences in the capacity of the kidneys to deal with the abnormal situation in each

CHART XIX.

Composite Graph of Changes during Treatment in Cases 156, 157, & 158.



case; anaemia may account for the low haematocrit values obtained from some patients with a nitrogen retention and in such cases the plasma protein value would be a better guide to the degree of blood concentration than the haematocrit value. In cases with nitrogen retention the uric acid and urea were usually retained roughly to the same degree and the proportion between their values in mg. per 100 c.c. was approximately 1 to 10.

One very severe (156) and 2 severe (157 and 158) cases of hyperemesis were followed during the first few days of treatment and serve to show the rapid improvement, in all the biochemical findings, which usually accompanies blood dilution. Two of the cases, 156 and 158, were given continuous intravenous glucose saline during their first 48 hours in hospital; the third case, 157, had no intravenous fluid. A composite graph of the changes in the haematocrit, plasma uric acid and blood urea values in these 3 cases is given in Chart XIX.; the daily urinary output in ounces is also charted. It will be noticed that the haematocrit, uric acid and urea curves are practically parallel and that there is an inverse proportion between the values of the blood non-protein nitrogen fractions and the volume of urine excreted indicating that the nitrogen retention was the direct result of the anhydraemia. In 4 of the 12 cases (cases 157, 158, 159 and 165) in the present series the plasma gave a positive delayed direct reaction to the

van den Bergh test and the plasma bilirubin ranged from 2.5 to 6 units per 100 c.c. plasma. The hyperbilirubinaemia was apparently unrelated to the anhydraemia and azotaemia although it was present in most of the cases in which the vomiting had lasted for many weeks. Several previous workers (Herold, 1939; Fikentscher, 1939) have concluded that the serum bilirubin may be taken as an index of the severity of the metabolic upset in each case; the present findings, however, do not support this view - cases with hyperbilirubin-:aemia were always of the severe type but several of the most severe cases had normal plasma bilirubin values.

There is a factor in these hyperemesis cases which tends to cause discrepency between the severity of the clinical condition and the biochemical findings. This is, the development of Wernicke's encephalopathy. Campbell and Biggart (1939) first drew attention to this syndrome in cases dying after a period of severe vomiting e.g. in cases of gastric carcinoma, and they found changes in the capillary endothelium together with small haemorrhages, at certain sites in the brain. Sheehan (1939b) found similar lesions in the brains of hyperemesis patients who developed the terminal cerebral syndrome. The features of this syndrome are apathy, drowsiness and paralysis of various parts e.g. soft palate, eye muscles etc; squint, diplopia, and partial or complete blindness frequently occur and before death there may be a phase of great loquacy or mental confusion

ending in coma. The patient usually lives only a few days after this syndrome is fully developed but early signs may be found in many cases of severe hyperemesis. These early signs are nystagmus, apathy, tenderness of leg muscles and a rapid pulse. Before the pathological lesions in the brain responsible for this cerebral syndrome were recognised and fully described, all these mental and nervous symptoms and signs used to be considered to be the result of some severe intoxication. Dieckmann and Crossen (1927), Fitzgerald and Webater (1938), Herold (1939), and others, describe cases with typical signs and symptoms of Wernicke's encephalopathy but they did not recognise the pathological basis of the condition. It is now suggested that the cerebral lesions are the result of a vitamen deficiency - particularly a deficiency of the vitamen B complex. A most important fact, pointed out by Sheehan (1939b) is, that this syndrome does not necessarily develop in the cases with the most severe vomiting and is liable to occur in patients after the vomiting has been controlled by treatment. When it develops in such cases the severity of the clinical condition becomes much greater than the accompanying anhydraemia and azotaemia would suggest.

In hyperemesis, therefore, there are two quite distinct processes which may be present in any case and either of them may be the cause of death. These are

I) Dehydration and anhydraemia with resulting azotaemia, in

some cases associated with hyperbilirubinaemia or acidosis.
II) Wernicke's encephalopathy.

These factors are of course closely related, in that they are both the result of the vomiting. In the great majority of severe cases both factors are present but their relative importance varies from case to case. Death, in hyperemesis, is sometimes due to other factors - namely - shock, resulting from operative interference, or delayed chloroform poisoning following operation. The patients are greatly predisposed to these conditions by the previous effects of the vomiting.

The findings in four fatal cases illustrate the relative significance of these factors.

Case 168. This patient had vomited for 12 weeks and then developed a "peculiar mental state". She refused food and drink and became very lethargic. Urinary output was good and there was no vomiting. Later blindness developed and hysterotomy was performed. The temperature rose to 102° and the pulse rate was 160. She remained unconscious after the operation and had twitchings of her face and arms. Death occurred about 24 hours after the operation. The blood urea at death was 57 mg. per 100 c.c. At the autopsy petechial haemorrhages were found in the corpora mammillaria, mid brain and floor of the fourth ventricle. There was no evidence that the death was due to shock. In this case there was a moderate nitrogen retention but Wernicke's encephalopathy was apparently the more important factor in causing death.

Case 169. This patient had vomited for 7 weeks and had evidence of severe acidosis. She improved greatly and was able to take light diet but the pulse rate began to rise. Slight vomiting continued and the urinary output was good. Her voice became very husky. Temperature 101°. Pulse 145. She became deeply comatose after only 1/3 gr. omnopon had been given. Hysterotomy was performed and practically no anaesthetic was necessary because the coma was so deep. She died 4 hours after the operation. Blood urea at death was 69 mg. per 100 c.c. Plasma bilirubin 2.5 units. At the

autopsy there were few signs of Wernicke's encephalopathy; only a few petechial haemorrhages were found in the floor of the fourth ventricle and all other parts of the brain appeared to be normal. There were several moderately large subendocardial haemorrhages on the left side of the interventricular septum; these have been shown to be a frequent post-mortem finding in cases of shock (Sheehan, 1939a), and it may be assumed in this case, where death occurred a few hours after operation, that the ultimate cause of death was shock.

Case 170. This case occurred before the pathological basis for Wernicke's encephalopathy was recognised but the history during the last few days of life is very typical. The patient had been vomiting for 5 weeks and then became very drowsy and the pulse rate rose. Intravenous glucose saline was given and the vomiting became less; the drowsiness increased and paralysis of the soft palate was noted. Hysterotomy was performed. The patient recovered consciousness but later became comatose and died 14 hours after the operation. At death plasma uric acid was 2.4 mg. per 100 c.c. and urea was 42 mg. There was, therefore, only slight azotaemia in this case and it may be presumed that Wernicke's encephalopathy was the more important factor in causing death.

Case 171. This case also occurred before the pathological basis for Wernicke's encephalopathy was recognised. In this case, however, there were no signs of cerebral disturbance until the day before death when the patient became extremely noisy and restless. The temperature rose gradually to reach 107° at death and the pulse rate was 170 to 180. Examination of the blood at death revealed gross non-protein nitrogen retention; the plasma uric acid was 20.9 mg. per 100 c.c. and the blood urea was 220 gms. It may be presumed that azotaemia and anhydraemia were more important factors in causing death in this case than Wernicke's encephalopathy.

The biochemical findings in severe hyperemesis cases may be summarised as follows.

I) High haematocrit values.

II) High plasma uric acid and urea.

III) A positive delayed direct reaction to the van den Bergh test and slightly increased plasma bilirubin in some cases.

These changes are the direct result of the

dehydration produced by the vomiting. A cerebral syndrome, Wernicke's encephalopathy, develops in some cases, probably the result of vitamin deficiency; this causes the severity of the clinical signs to be increased out of proportion to the biochemical findings and is frequently the cause of death.

Vomiting of Late Pregnancy.

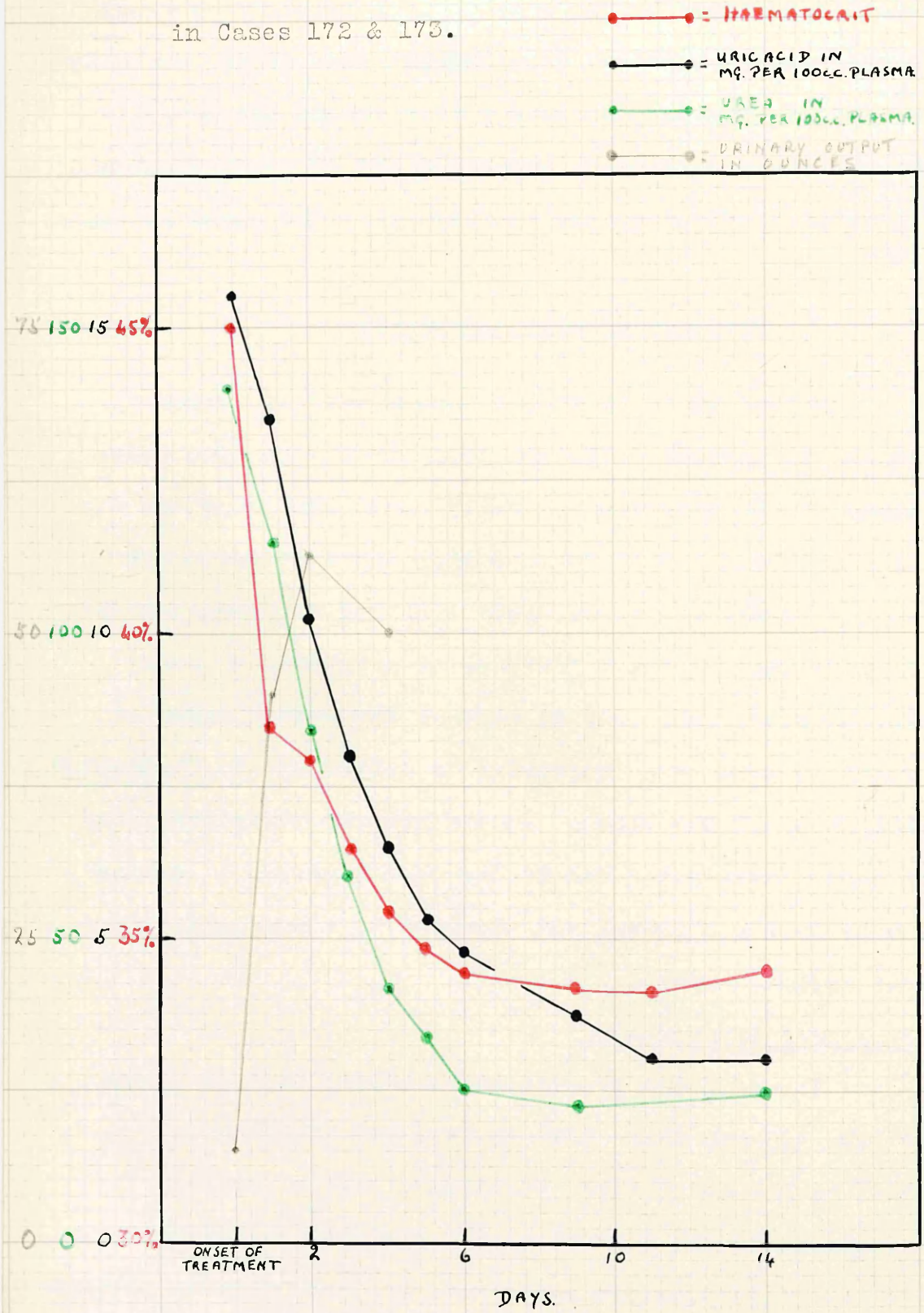
Severe vomiting in the second half of pregnancy may be due to several different conditions - in some cases the cause of the vomiting is easily found but in others the vomiting may be the only obvious sign and such cases are frequently labelled - "pernicious vomiting of pregnancy" or "toxic vomiting" or "late vomiting of pregnancy". Such labels, however, only tend to obscure the actual cause of the vomiting which can frequently be determined by biochemical investigation of the blood. In the present study many such cases were investigated and it has been possible to divide them both from a clinical and biochemical standpoint into several definite groups.

1. Prolonged Hyperemesis.

Three of the present cases belong to this group, cases 172, 173 and 174. Each patient was admitted to hospital as a case of "late vomiting", but on careful interrogation it was found that the vomiting, although more severe immediately before admission, had been present throughout the early months. Cases 172 and 173 were both extremely ill

CHART XX.

Composite Graph of Changes during Treatment in Cases 172 & 173.



on admission and the blood investigation revealed gross changes; in case 174, the vomiting was not so severe and the biochemical changes were less. The findings in the three cases are given in the appendix and a composite graph of the changes occurring during treatment in cases 172 and 173 is given opposite - Chart XX. The blood was very concentrated (haematocrit 46% and 44% respectively) and there was very marked azotaemia in each case; the plasma uric acid and urea values were increased proportionally - the ratio between their values in mg. per 100 c.c. being 1 to 10 approximately - as in the hyperemesis casespreviously described. Cases 172 and 174 were slightly jaundiced; the plasma gave a positive delayed direct reaction to the van den Bergh test and the plasma bilirubin was 12.2 and 5.6 units respectively: in case 173 the van den Bergh test was negative. The urinary output in each case was very small before treatment was given but, as can be seen from Chart XX., immediately the anhydraemia was relieved by intravenous glucose saline, the urinary output increased and the nitrogen retention in the blood diminished. The uric acid and urea values decreased more or less with the haematocrit values and maintained their mutual proportion. These blood changes are all similar to those found in the very severe hyperemesis cases and it seems likely that they are the direct result of the unusually prolonged vomiting. In cases 172 and 173 there were early signs of Wernicke's

encephalopathy. Case 172 was very drowsy and had nystagmus, muscular tenderness and pain over the sciatic nerves. Case 173 had nystagmus and tenderness over several nerves. All these signs cleared up rapidly with the intravenous glucose therapy and large doses of Vitamen B_1 intramuscularly.

In this group of cases the characteristic findings which differentiate them from the cases of the other groups are

a) The history of the prolonged vomiting.

b) Negative clinical findings apart from the vomiting.

c) Blood changes - all secondary to the vomiting as in the early hyperemesis group.

d) Steady improvement as the anhydraemia is relieved.

II. Pyelitis of Pregnancy.

Pyelitis is a frequent cause of very severe and prolonged vomiting. About the fifth month of pregnancy is the commonest time for symptoms of pyelitis to occur but they may begin or become very severe at any time in the second half of pregnancy. In most cases the diagnosis is easily made from the history of lumbar pain, rigors etc. and from the urinary findings, but quite frequently cases present themselves with vomiting as the only symptom and sign, and it is not until several days have elapsed that the vomiting is found to be due to the urinary tract infection. In such a case the vomiting is presumably due to complete blockage of the ureter resulting in a temporary pyonephrosis on the affected side; if the urinary tract on the other side is uninfected

the urinary findings are normal.

In the great majority of cases of pyelitis of pregnancy no abnormality of the blood can be found. In a few severe cases, however, nitrogen retention develops and there is sometimes slight jaundice. Six cases of severe pyelitis, cases 175 to 180, were investigated in the present study. The biochemical findings during the course of the disease, in each case, are given in the appendix. The haematocrit values were low in each case; the mean haematocrit value from the 6 cases was 29.5% with a range of 27.5% to 31%. The values showed very little change throughout the course of the disease. The absence of blood concentration in these cases is rather surprising since there had been severe and prolonged vomiting in each case. The low haematocrit value appears to be a constant finding in cases of pyelitis of pregnancy and it may be of help in the differential diagnosis of these cases of severe vomiting without obvious cause. For example, case 178 was admitted to hospital as a case of "late vomiting" near term; blood pressure was normal, there was no pyuria but the urinary output was small. Vomiting was very severe but the haematocrit value was only 27.5%. The patient went into labour soon after admission and was delivered the same day; the maximum haematocrit value during labour was 32%. On the next day she passed urine heavily laden with pus and her general condition improved greatly; plasma uric acid and urea decreased rapidly to normal. The diagnosis of

pyelitis was suggested in this case on admission from the history of severe vomiting, diminished urinary output, and because the haematocrit value was low in spite of the vomiting. The sudden excretion of large quantities of pus after delivery confirmed the diagnosis. Anaemia resulting from the infection is a possible cause of the small cell volume.

The original uric acid and urea values in 5 of the cases were practically within normal limits although the patients were sharply ill at the time. The uric acid and urea values rose very late in the disease - in the two fatal cases, 176 and 177, the rise began only a few days before death. In case 179 the uric acid and urea values were always within normal limits although this patient appeared to be acutely ill with vomiting and pyuria. Case 180 was only admitted to hospital late in the disease and therefore the original urea value obtained was high.

Labour appeared to aggravate the condition of these patients greatly - presumably due to further obstruction of the ureters causing increased spread of the infection into the kidney tissue. It will be noticed that in cases 175 and 176 the nitrogen retention mounted rapidly before and after delivery. In cases 175, 176 and 177 where a rapid increase in the nitrogen retention occurred, the blood urea rise was greater than the corresponding rise in plasma uric acid.

In 4 of the 6 cases, 175, 177, 178 and 180, studied in detail the plasma gave a positive immediate direct reaction

to the van den Bergh test and the plasma bilirubin was raised: the mean value for the four cases was 4.5 units with a range of 3 to 7 units.

In the three fatal cases, 176, 177 and 180, post-mortem examination revealed gross pyelonephritis.

In case 177 there was great destruction of the kidney tissue on both sides. The right kidney weighed 180 gms. and the left 90 gms. The left kidney was small and misshapen and had gross hydronephrosis; only one or two small areas of kidney tissue remained. The surface of the right kidney was irregular and showed large deeply congested areas; again there was gross distension of the calyces and the kidney tissue showed diffuse and severe pyelonephritis with small abscesses in the outer cortex. In spite of this gross renal damage which must have been present for several weeks before death, there was no retention of plasma uric acid and urea in the blood until the day before death.

In cases 176 and 180 there was less destruction of the kidneys but pyelonephritis was diffuse and severe; there were numerous small cortical abscesses with purulent tracks leading through the cortex and medulla to the pelvis. In these cases a nitrogen retention was present for several days before death.

The characteristic biochemical findings in the blood in cases of pyelitis of pregnancy are:

a) A low haematocrit value.

b) A retention of non-protein nitrogen fractions, the rise in blood urea being relatively greater than the rise in plasma uric acid. In some cases with gross renal damage, however, there may be no retention of these fractions until very late in the disease.

c) A positive immediate direct reaction to the van den Bergh test and a slight increase in plasma bilirubin in some cases.

III. "Erythroblastosis Foetalis".

In four of the cases (181, 182, 183 and 184) of

severe vomiting in late pregnancy in the present series an abnormality of the ovum was found to be present.

The vomiting in these cases commenced about the 28th to the 32nd week of pregnancy and was exceedingly severe. The patients rapidly became acutely ill and were delivered prematurely of still-born children. In each case the uterine contents were noted to be large and oedematous: in case 181, the 7 month foetus was hydrocephalic, weighed 7 lbs. and the placenta weighed 5 lbs. 6 oz.; in case 182, the foetus at $7\frac{1}{2}$ months was very oedematous and weighed 7 lbs., the placenta was noted to be large; in case 183, the 6 month foetus had generalised oedema but weighed only $4\frac{1}{2}$ lbs. while the placenta weighed $1\frac{3}{4}$ lbs.; and in case 184, the foetus at $7\frac{1}{2}$ months weighed $8\frac{1}{2}$ lbs. In 3 of the cases the liquor amnii was noted to be a very deep yellow colour. These children and placentae were not examined further but they presumably belong to the erythroblastosis group of abnormalities.

During the acute stage of the illness each of these patients was very restless and exhibited slight mental confusion. There was slight icterus in three of the cases, 181, 183, and 184, while case 182 became deeply jaundiced after delivery. The plasma gave a positive delayed direct reaction to the van den Bergh test in each case on admission. The blood pressure was raised in one case but was normal in the other three. The urine contained some albumen and a trace of bile in each case.

Examination of the blood showed slightly increased haematocrit values in all cases, but in cases after delivery or after intravenous glucose had been given the increase was not an absolute one. Plasma uric acid, urea and bilirubin were raised in each case. In three of the cases, 181, 183 and 184, the uric acid value was relatively much higher than the urea value and in one case the blood urea was normal. The abnormalities cleared up gradually after delivery. In three of the cases, 182, 183 and 184, continuous intravenous glucose was given and appeared to aid recovery. The plasma bilirubin ranged from 2.9 units in case 181 to 22 units in case 182. (Individual findings given in appendix).

In these patients the blood changes were not merely the result of severe vomiting; there was no obvious relation between anhydraemia and azotaemia. There is presumably a relationship between the mother's illness and the abnormality of the products of conception but it is impossible to say which is primary. Although the term "erythroblastosis foetalis" has been used to describe this group of patients, it is not suggested that all patients delivered of children with erythroblastosis, suffer from this condition. The condition appears to be unrelated to hypertensive toxaemia - the patients were all extremely ill without any marked rise in blood pressure or sign of impending eclampsia and the blood urea values were high in contrast to those in the pre-eclamptic cases. The early appearance of jaundice, positive delayed

direct reaction to the van den Bergh test, and the raised plasma bilirubin in each case suggest that liver damage may be a primary factor but the etiology of the condition is obscure. The main characteristics of this severe vomiting group are:

a) The foetal and placental abnormalities.

b) Slight jaundice and raised plasma bilirubin with a positive delayed direct reaction to the van den Bergh test.

c) Very high plasma uric acid and moderately high blood urea values.

IV. Miscellaneous Group.

This group includes cases of vomiting due to hypertensive toxaemia, acute yellow atrophy and idiopathic vomiting of late pregnancy.

Hypertensive toxaemia must be included as a cause of vomiting in late pregnancy but it is seldom the cause of prolonged and severe vomiting. Severe pre-eclamptic and eclamptic cases often have a moderate amount of sickness but vomiting is rarely the main feature of the disease. Such cases can, therefore, be easily differentiated from the other groups described here, by the presence of the characteristic signs of hypertensive toxaemia. In the series of 56 pre-eclamptic and 42 eclamptic cases studied in sections II. and III., no patient had sufficient vomiting to warrant her inclusion in a group of late vomiting cases.

Acute yellow atrophy is certainly a cause of very

severe vomiting and this condition will be discussed in the next section on jaundice in pregnancy.

With regard to idiopathic vomiting of late pregnancy it seems very unlikely that such a condition exists. Detailed examination of each case usually reveals the cause or nature of the vomiting but, at present, this term may be given to any case, in the second half of pregnancy, with severe and prolonged vomiting which cannot be included in any of the groups described here. It will be noted that the terms "toxic vomiting", and "pernicious vomiting" of pregnancy are not used in the present work. These terms have been applied to almost any case of vomiting with signs of a general metabolic disturbance - for example, cases of severe pyelonephritis with raised temperature and azotaemia, cases of severe hyperemesis with azotaemia, acidosis or Wernicke's encephalopathy, and cases of hypertensive toxaemia with some vomiting. In some of these cases the metabolic upset is the result of the vomiting while in others it results from the cause of the vomiting; the use of terms such as "toxic vomiting" or "pernicious vomiting" of pregnancy only tend to obscure the nature of the illness present.

Summary.

Cases of severe vomiting, related to, and occurring during pregnancy, are studied. They are classified as follows:

A. Vomiting of Early Pregnancy. - Hyperemesis Gravidarum.

B. Vomiting of Late Pregnancy.

I. Prolonged Hyperemesis.

II. Pyelitis of Pregnancy.

III. "Erythroblastosis Foetalis."

IV. Miscellaneous Group - including hypertensive toxaemic cases, acute yellow atrophy cases and idiopathic cases.

The biochemical findings from cases in each group are given and the points of differentiation are discussed.

S E C T I O N VI.

JAUNDICE IN PREGNANCY.

In the preceding sections many patients suffering from various conditions have been noted to have slight jaundice with raised plasma bilirubin values and to give positive reactions to the van den Bergh test. In some cases the pathological basis for these findings can be traced, but in others the actual cause is obscure.

In Eclampsia, haemolysis may give rise to a positive indirect reaction to the van den Bergh test and increased plasma bilirubin - e.g. in case 121 described in Section III., or the liver haemorrhages may cause a positive delayed direct reaction to the van den Bergh test and increased plasma bilirubin.

In Hyperemesis, however, the cause of the raised plasma bilirubin and positive van den Bergh test in some patients, is not clear. Herold (1939), obtained no evidence of increased destruction of red blood cells in hyperemesis patients and he concluded that the raised serum bilirubin and increased porphyrin excretion, which he found in these cases, were due to depressed liver function. Pathological findings in the livers of fatal cases do not give much indication as to the cause of the jaundice; it can not be ascribed in any

way to the central fatty change found in some fatal cases.

Slight jaundice in cases of Pyelonephritis may possibly be due to an early infective cholangitis. In fatal cases of pyelonephritis with jaundice, polymorph infiltration is found around the portal tracts; and, in four of the pyelitis cases of the present study the plasma gave a positive immediate direct reaction to the van den Bergh test, suggesting that the jaundice was of the obstructive type. Slight jaundice, raised plasma bilirubin and a positive delayed direct reaction to the van den Bergh test are also features of the cases of late vomiting with foetal abnormalities. The possible liver lesions in these cases are discussed later. In all these conditions just mentioned, with the exception of the last, the jaundice is very much a secondary feature of the disease - it appears late in the disease and is slight.

The present section deals with cases in which jaundice is one of the main features. Cases of obstructive jaundice due to gall stones etc. are not included. Three different conditions have been found to cause such jaundice; these are,

1. Obstetric acute yellow atrophy.
2. True acute yellow atrophy, and
3. Delayed chloroform poisoning.

Sheehan (1940), has shown that the pathology of the condition frequently described as acute yellow atrophy of pregnancy is quite different from that of the acute yellow atrophy met with

in non-pregnant patients. He refers to the former as obstetric acute yellow atrophy and the latter as true acute yellow atrophy and this terminology will be used here. In each condition jaundice is the main feature of the disease and clinically the two conditions are very alike although their pathology is quite distinct. One case of each type of liver damage has been studied in the present series and it will be shown that the biochemical changes differ widely in the two conditions.

Obstetric Acute Yellow Atrophy.

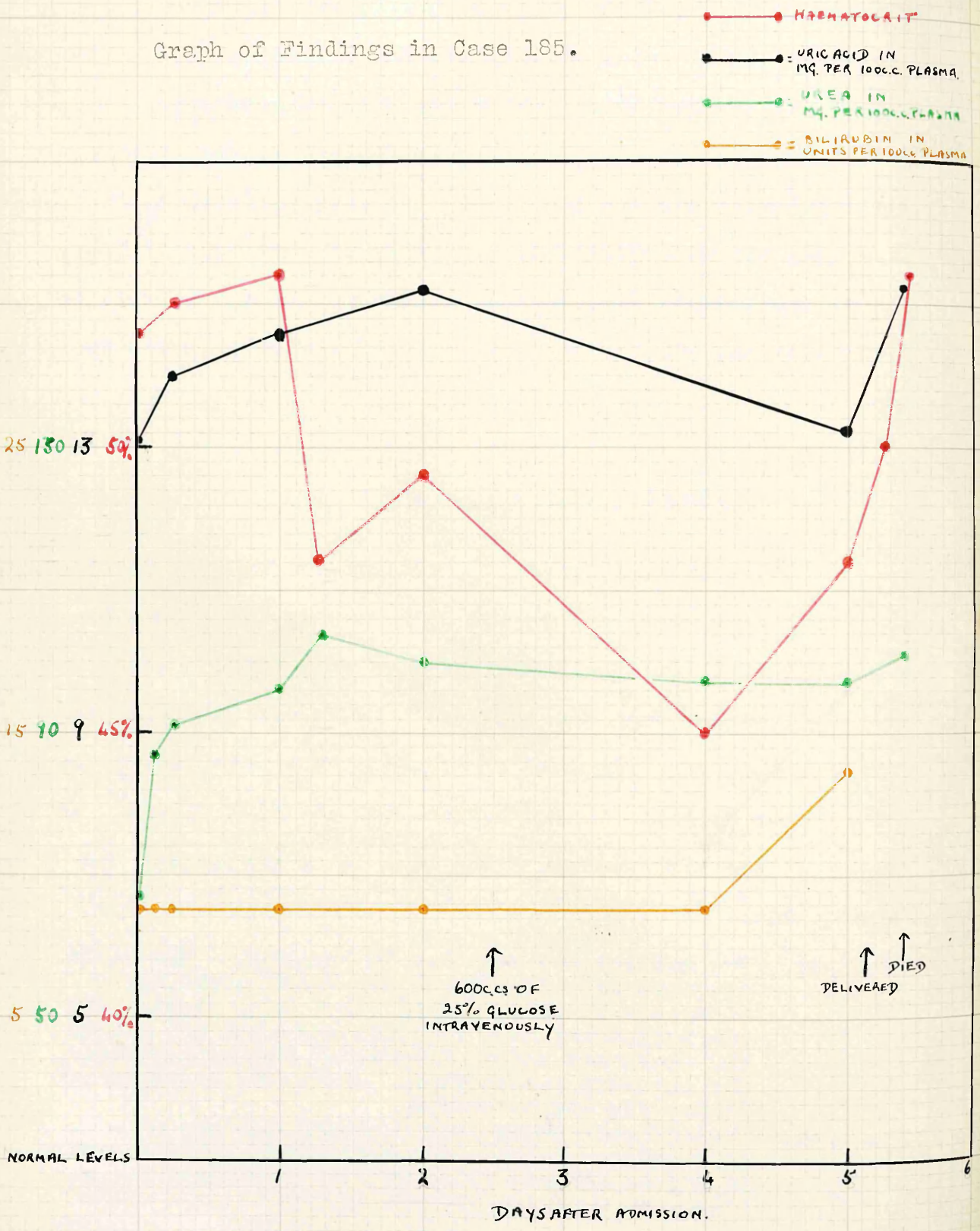
The patient, case 185, suffering from this condition was admitted to hospital in the 32nd week of her pregnancy as a case of late vomiting. She had been well until 2 weeks before admission when she began to have severe vomiting and complete anorexia. Attacks of vomiting were preceded by some pain in the epigastrium and between the shoulder blades. At this time the blood pressure was said to be normal and the urine clear.

26. 3.40. The patient was admitted to hospital. The blood pressure was 176/120 and the urine contained albumen - .5 parts Esbach - but no acetone. She was well-nourished; her lips were red and glazed and there was slight icterus of the conjunctivae. She was very constipated. There was no abdominal tenderness and no oedema.

27. 3.40. She had only occasional vomiting and was able to take ordinary diet. Blood pressure 140/110, urine contained albumen .5 parts E., no acetone and no bile. T. 97.6; P. 100; R. 20.

CHART XXI.

Graph of Findings in Case 185.



28. 3.40. Slight general icterus was noted but there was no bile in the urine. Stools were normal and the urinary output was good. She complained of backache and epigastric pain and had 22 oz. sickness. T. 97; P. 120; R. 20. The jaundice deepened during the next few days and she was rather drowsy - vomiting occasionally. The urinary output was good (42 to 62 oz. per day) and the urine contained .5 parts E., no acetone and no bile.

30. 3.40. 600 c.c. 25% glucose saline was given intravenously and this was followed by a definite improvement in her general condition. No further intravenous fluid was given.

1. 4.40. The patient had no vomiting and the jaundice was unchanged.

2. 4.40. Labour pains began at 8 a.m. The jaundice appeared to be deeper and mucosae were rather cyanosed. The patient was extremely drowsy but her mental condition was otherwise normal.
10 a.m. T. 99; P. 112; R. 20. Blood pressure 170/115
3 p.m. She was delivered spontaneously of a living, premature, female child weighing 4¾ lbs.; the placenta was not weighed. She became very sick and had severe retching soon after delivery. Her skin felt extremely hot. Blood pressure 140/125.
4.30 p.m. The patient became very collapsed - blood pressure 140/130. She was very apathetic and the vomiting became severe. Continuous intravenous glucose saline was given but the patient did not improve.
9 p.m. She became very restless and later comatose. Death occurred at 11.10 p.m.

The biochemical findings are given below and are shown opposite in graph form - Chart XXI. - plasma sodium and whole blood chloride are not included in this graph.

Biochemical findings in Case 185.

	28. 3. 40.			29. 3. 40.		30.3.40.	1.4.40.	2. 4. 40.		4.30 p.m.	P.M. Died 11.10 p.m
	10 a.m.	12 m.d.	4 p.m.	10 a.m.	3 p.m.			10 a.m.	3.20p.m.		
Haemt	52%	54%	52.5%	53%	48%	49.5%	45%	48%	50%	53%	
Sodium.	335	352	333	359	348	355	359	355	337	369	
W.B. Cl.	340	465	393	423	430	463	428	498	410	628	
Uric Acid.	13		14	14.6		15.2		13.2			15.2
Urea.	69	87	91	96	104	100	97	97			101
Bilirubin.	8.8	8.8	8.8			8.8	8.8	13.6			
Urinary Output.			37 oz.		42 oz.	62 oz.	72 oz.	52 oz.			

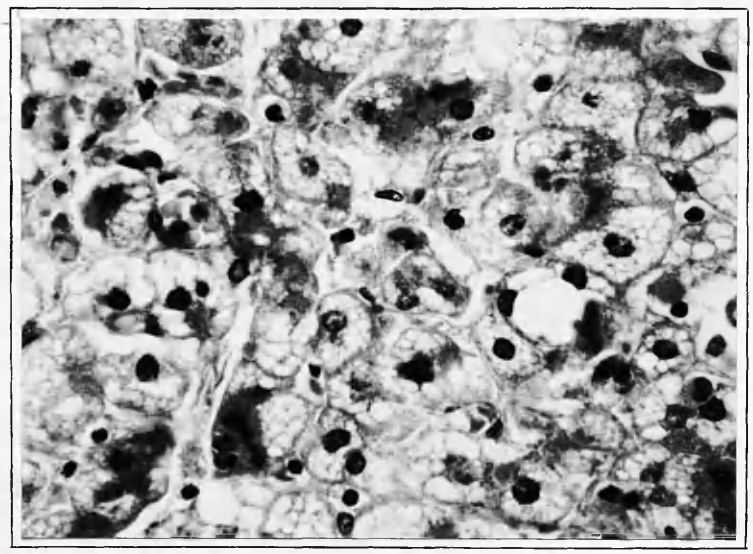
Delivered 3.10 p.m.

CASE 185.

The blood was extremely concentrated throughout the illness. The plasma urea values rose rapidly during the first day of the investigation and then remained almost unchanged; the plasma uric acid values were greatly increased and were relatively much higher than the urea values. The plasma gave a positive delayed direct reaction to the van den Bergh test and the plasma bilirubin was high. Blood dilution, accompanied by a fall in plasma uric acid and urea, followed intravenous glucose transfusion and the improvement lasted for more than 24 hours. Whole blood chlorides were abnormally low until a few hours before death when they rose to 628 mg. per 100 c.c.; plasma sodium values ranged around the upper limit of normal until a few hours before death when they rose to 369 mg. per 100 c.c. Some of the urinary findings were rather unexpected. During the period of observation the patient's urinary output was very good - 37 to 79 oz. per day, in spite of the fact that her blood was very concentrated. Unfortunately no renal function tests were performed but it may be presumed that the concentrating power of the kidney was poor. Bile was never present in the urine although the plasma bilirubin was raised and there was obvious jaundice. Urinary albumen remained constant at .5 parts Esbach throughout the period of examination.

At the autopsy, performed three hours after death, the liver weighed 1,100 gms. and was very soft. It was light yellow in colour with a fine mottling of pin-point red dots.

PLATE IV.



Liver in Obstetric Acute Yellow Atrophy.

High power view showing cytoplasmic change in centrolobular cells and absence of necrosis.

(Case 135)

On histological examination the centrolobular cells were found to be swollen and filled with a fine foam of small fatty globules. The cells of the outer halves of the lobules were unaffected. There was no trace of necrosis of liver cells. A microphotograph of the centrolobular cells is shown in Plate IV. The changes are typical of obstetric acute yellow atrophy, as described by Sheehan (1940), although the zone of fatty change is less extensive in the present case than in those originally described. The kidneys were small and very firm. The cortex was very white, the normal striation was distorted and the capsule was adherent. Microscopically there was a very fine early interstitial fibrosis. The glomeruli appeared to be normal but there was evidence of tubular damage - the epithelium of the convoluted tubules was very low; some of the cells were necrosed and in parts there was regeneration of tubular cells. A soft, lobulated tumour mass, about 4" x 3" x 2" was found lying on the posterior abdominal wall behind the pancreas on the left side, with no attachment to any organ. On section, the tumour consisted of yellowish tissue, obviously fasciculated, with several haemorrhages into its substance and some cystic change. From microscopic examination a diagnosis of benign chromaffin cell tumour (paraganglioma) was made. The remaining post-mortem findings revealed no abnormalities except for two small pedunculated subperitoneal uterine fibroids, one of which showed marked red degeneration and contained quite a

large patch of necrosis.

In this case (185) the degenerative changes in the liver were typical of obstetric acute yellow atrophy; they were confined to the centre halves of the lobules, however, and they do not seem a sufficient cause of death. The part played by the adrenalin-secreting paraganglioma which was found at the post-mortem examination in this case must be estimated. A history was not obtained of paroxysmal hypertension or other clinical manifestations which have been reported in the literature as associated with these benign chromaffin cell tumours (Eisenberg and Wallerstein, 1932; Belt and Powell, 1934; Wells and Boman, 1937; Howard and Barker, 1937; Hegglin and Nabholz, 1938; and Holst, 1938). Detailed blood pressure readings were not available in the present case, but after admission to hospital, these varied between 176/126 and 140/110. A greatly increased susceptibility to shock, however, is one of the outstanding features of the benign chromaffin cell tumour syndrome and Oberling and Jung, (1927), report an example of this syndrome in a pregnant woman who died of shock six hours after a normal delivery. It seems very likely that, in the present case, the ultimate cause of death was shock. This conclusion is supported by the clinical findings - the sudden deterioration in the patient's condition during labour, the greatly diminished pulse pressure after delivery and final collapse. This case may, therefore, be regarded as one of mild obstetric acute

yellow atrophy in which death occurred from an incidental cause.

Stander and Cadden (1934a) describe a case of "acute yellow atrophy of the liver" in pregnancy with findings very similar to those of the case just described. The disease appears to have been more acute in their case - death occurring 6 days after the onset of symptoms. They found raised blood non-protein nitrogen, uric acid, amino-acids and creatinine with low blood chlorides and diminished CO_2 volumes per cent; the only difference between their findings and those of the present case is in the blood urea value which was within normal limits in their case. The pathological liver changes in the two cases appear to be identical. Sheehan (1940) describes six cases with similar pathological findings and has pointed out that this type of liver lesion is not, as suggested by Stander and Cadden (1934b), a stage preceding the necrosis of true acute yellow atrophy but is a definite entity.

There are several points of resemblance between the biochemical findings in case 185 and those in cases 181 to 184, the third group of late vomiting cases associated with erythroblastosis foetalis. Severe vomiting and varying degrees of jaundice are the main features of each case and the time of onset of symptoms is approximately the same in all cases. Increased blood concentration, very high plasma uric acid, raised blood urea, positive delayed direct reaction

to the van den Bergh test and raised plasma bilirubin are found in both types of case. The question arises whether these late vomiting cases are non-fatal examples of obstetric acute yellow atrophy. Duncan and MacLachlan (1933), reported a case of "yellow atrophy of the Liver in Pregnancy" in which the biochemical findings resembled those of the present series. The blood urea reached 168 mg. per 100 c.c. but uric acid was not estimated. Their patient recovered after the delivery of a macerated foetus. Sheehan (1940), pointed out that the appearance of the liver lesions in this condition suggested that they were of a reversible nature and that they were possibly only the result of a particular type of metabolic disturbance; he mentions that in his cases all the children were still-born. In case 185, the child was born alive but, as has been mentioned, the liver lesions were not very far advanced and this case was presumably only a mild example of obstetric acute yellow atrophy; there was the additional factor of the adrenaline secreting tumour. One can only speculate with regard to the relation of the liver changes and this tumour but hyperadrenalinism resulting in a depletion of liver glycogen may have been a factor in causing the liver damage. Different factors causing glycogen depletion of the liver may be operative in other cases of obstetric acute yellow atrophy.

From the biochemical findings in this case and the few previously described in the literature the characteristic

changes in the blood in obstetric acute yellow atrophy may be summarised as follows.

I) Increased blood concentration.

II) Very high plasma uric acid and moderately raised blood urea values.

III) Low whole blood chlorides.

IV) Positive delayed direct van den Bergh test and raised plasma bilirubin.

These changes are, of course, by no means diagnostic of obstetric acute yellow atrophy but they may be helpful when taken in conjunction with other findings.

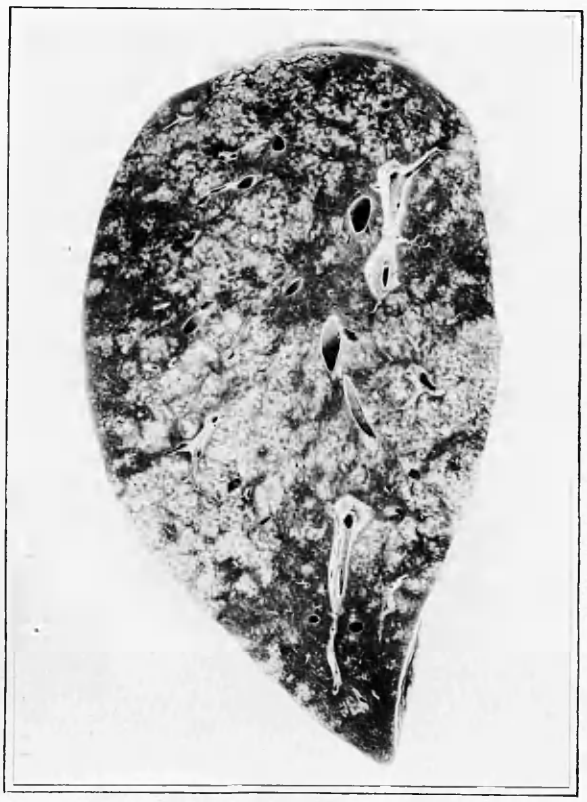
True Acute Yellow Atrophy.

One case of true acute yellow atrophy of the liver has been encountered in a pregnant woman during the present study (Case 186) and an account of the clinical, biochemical and pathological findings is included here for comparison with those of the previous case (185).

The patient, a girl of 17 years, was 7 weeks pregnant when admitted to hospital. A week before admission she had noticed slight yellowness of her conjunctivae and had had slight vomiting and diarrhoea with light yellow stools. Jaundice deepened but she had no more sickness.

25. 6.40. Admitted to hospital. She was deeply jaundiced, the urine contained albumen +, and a trace of bile pigment, but no other abnormality was made out on general examination. T. 97.6; P. 92; R. 20. B.P. 120/65.

PLATE V.



Liver in True Acute Yellow Atrophy.

Naked eye view showing red and yellow patches.

(Case 136)

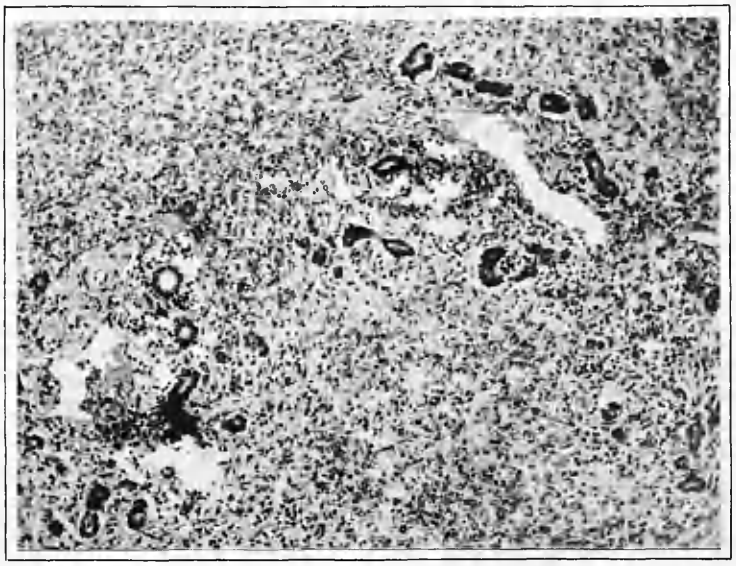


PLATE VI.

Liver in True Acute Yellow Atrophy. (Case 186)

Low power view from red area showing absence of liver cells and numerous newly formed bile ducts.

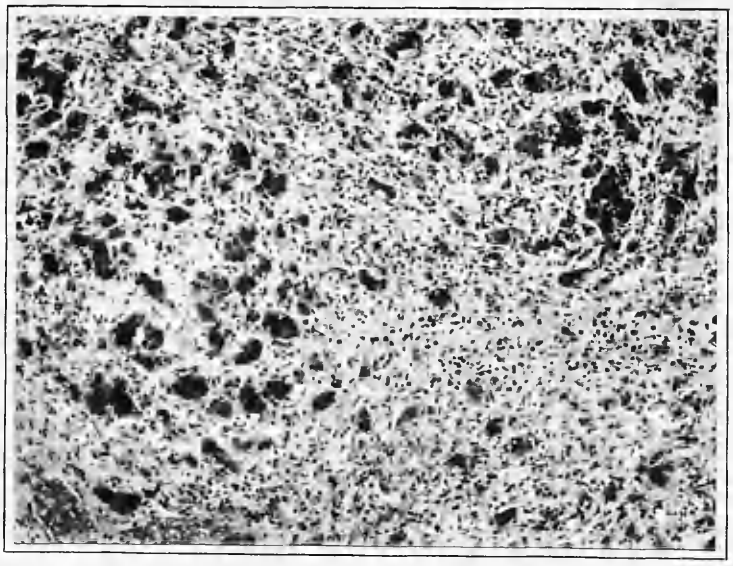


PLATE VII.

Liver in True Acute Yellow Atrophy. (Case 186)

Low power view from yellow area showing small groups of surviving liver cells with strongly basophil staining reaction.

26. 6.40. She appeared well and her mental condition was normal. Her stools were light clay coloured. She had no sickness during the next few days. The jaundice was still very deep.

29. 6.40 at 2 p.m. The patient suddenly became very irritable and screamed loudly when touched or disturbed.
11.30 p.m. She had become stuperose and would not speak but when roused she was maniacal. She had no vomiting. 300 c.c. 50% glucose saline + 10 units of insulin were given intravenously.

30. 6.40. 10 a.m. She was somewhat improved and was now conscious and able to speak. Her stool was of normal colour and the urine deeply bile-stained. Later in the morning she began to vomit and gradually relapsed into a semi-conscious state becoming maniacal when roused. The pulse rate rose to 130 per minute but the temperature remained normal. 30% glucose was given intravenously.

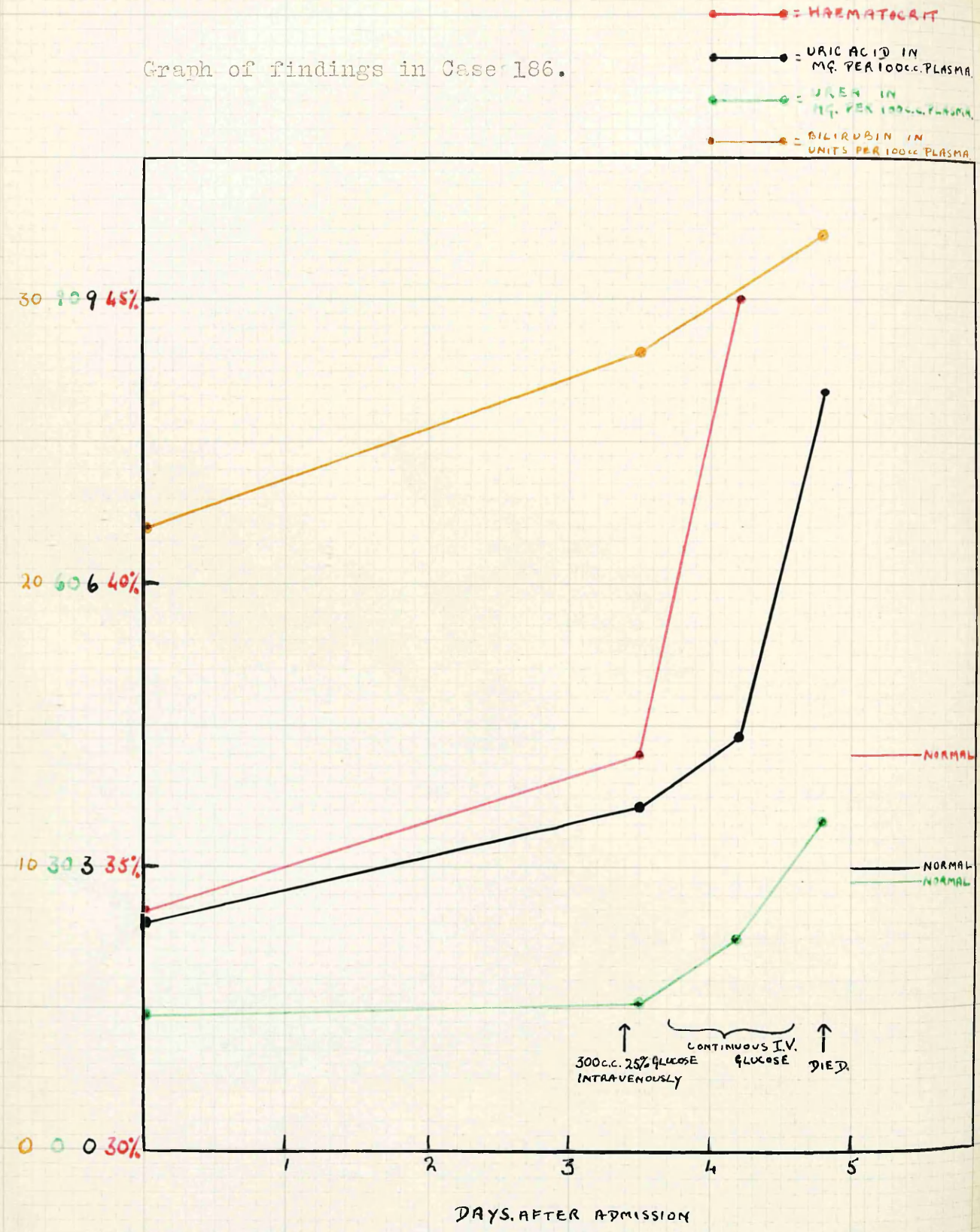
1. 7.40. T. 98.2; P. 140; R. 24. She remained deeply comatose throughout the day and exhibited Cheyne Stokes respiration. Jaundice appeared to be deeper. Continuous intravenous 30% glucose was given with no effect. The urinary volume passed was large and the urine contained a large amount of bile pigment. At 9 p.m. she had twitchings of her face and severe right-sided clonic spasms of the body and limbs. T. 102.4; P. 144; R. 24.

2. 7.40. The patient died at 1.45 a.m.

At the autopsy, performed 1¼ hours after death, the liver weighed only 700 gms. It was shrunken and the peritoneal surface was wrinkled. The cut surface was dark red in colour with a mottling of irregular yellow patches - see Plate V. Microscopically there was widespread necrosis of liver cells. In the red areas only shadows of the columns remained - see Plate VI., and in the yellow areas there were small groups of surviving liver cells; these cells showed a strongly basophil staining reaction - see Plate VII. Over large areas the necrotic debris had been absorbed, its place being occupied by dilated sinusoids packed with blood. There was some

CHART XXII.

Graph of findings in Case 186.



infiltration of small round cells and polymorphs from the portal tracts. Large numbers of new-formed bile ducts surrounded the portal tracts in many places - see Plate VI., and there was some increase in the periportal fibrous tissue. Sudan staining revealed the presence of droplets of fat in many of the surviving liver cells. The kidneys showed no abnormality. In the lungs there was a very haemorrhagic broncho-pneumonia. The mucosa of the duodenum was swollen and inflamed. The uterus contained an early gestation sac about 2 cm. diameter.

It will be seen that the post-mortem findings are typical of true acute yellow atrophy, such as occurs either idiopathically or as the result of poisoning by various substances - e.g. dope, T.N.T., cincophen etc.; and in this case there were grounds for suspecting that some chemical or drug had been taken although no history of this could be obtained from the relatives.

The biochemical findings are given below and are shown in graph form opposite - see Chart XXII.

Biochemical Findings in Case 186.

	27.6.40.	10 p.m. 30.6.40.	12 m.d. 1.7.40.	3 a.m. P.M. 2.7.40.
Haematocrit.	34%	37%	45%	
Uric Acid.	2.6	3.6	4.4	8.0
Urine.		0.03		0.056
Urea.	14.3	16.	23.	35.
Urine.		0.46		
Bilirubin.	22	28		32

Intravenous glucose, 30.6.40, at 8 p.m.

On 27.6.40, ten days after the onset of jaundice and other symptoms, the blood investigation revealed no abnormality, apart from a direct positive reaction to the van den Bergh test with only a ½ minute delay, and a very high plasma bilirubin (22 units); the haematocrit reading was normal and the plasma uric acid and urea were within normal limits although the urea value was rather low. The stools contained no bile pigment and at this stage a diagnosis of catarrhal jaundice was made. After the patient had become acutely ill, blood investigation showed a rise in haematocrit value from 34% to 37% (probably less than the actual rise because the specimen of blood was withdrawn 2 hours after 300 c.c. of 30% glucose had been given intravenously). The blood sedimentation rate of this specimen was nil. at 1 hour, suggesting a great increase in fibrinogen. Plasma uric acid and urea were still within normal limits while the plasma bilirubin had increased. Just before death there was a marked increase in blood concentration, the plasma uric acid showed a rapid increase to 8.0 mg. per 100 c.c. and the plasma urea rose to 35 mg. per 100 c.c. Uric acid and urea percentages in the urine were low.

The striking feature in both the clinical and biochemical findings in this case was the absence of signs of severe liver damage until 48 hours before death. It is quite clear, however, from the histological appearance of the liver that the massive necrosis did not take place during the last

few days of life; the extent of the bile duct proliferation and the complete disappearance of the liver cell remnants at most areas indicate that the lesion was probably of 10 to 14 days' duration, i.e. it had occurred about the time of the onset of symptoms. A low blood urea value was to be expected in such a case because it has been shown that urea is formed by the liver (Mann and Magath, 1921).

Numerous reports of cases of acute yellow atrophy have been given in the literature but only two have been found which include biochemical findings in such cases. Stadie and Van Slyke (1920) report the case of a woman aged 29 years who died after 7 days' illness; the liver at post-mortem examination weighed 1,000 gms. and there was extensive necrosis of liver cells although some of the periportal cells appeared healthy. The blood urea nitrogen values during the last 3 days of life were 12, 9 and 16 mg. per 100 c.c. respectively and the urinary urea nitrogen percentage was very low. Blood amino-acid nitrogen values during the last 3 days of life were 14, 17, and 26 mg. per 100 c.c. respectively while urinary amino-acid nitrogen and ammonia ratio were greatly increased. Uric acid was not estimated. The authors regarded these findings as evidence of partial loss of the deaminising power of the liver. Rabinowitch (1929), reports a case of a woman aged 28 years, who died after 3 days' illness. The liver at post-mortem examination weighed 650 gms. and showed necrosis of almost all cells. No blood urea was found in this

case and the blood amino-acid nitrogen value was 216 mg. per 100 c.c. The blood uric acid was 1.96 mg. per 100 c.c. The case of Stadie and Van Slyke and that of Rabinowitch were both apparently more acute than the present case. Unfortunately blood amino-acids were not determined in the present case but the blood and urinary urea findings are similar to those of the cases just mentioned and tend to support the conclusion of previous workers with regard to the part played by the liver in urea metabolism.

The uric acid findings were very unexpected. The work of Bollman, Mann and Magath (1925), and Gremels and Bodo (1926), indicates that in dogs uric acid is destroyed in the liver; and in man it is generally accepted that liver damage results in an increase in blood uric acid. The present findings do not support such a hypothesis. It is possible that, in the present case, regeneration of liver cells was sufficient to deal with the endogenous uric acid and to form a certain amount of urea; certainly the surviving liver cells, seen microscopically, appear to be young and were presumably active. The low uric acid value found in Rabinowitch's case, however, suggests that another explanation is necessary since the liver damage was gross and there was no sign of any cell regeneration.

In contrast to the non-protein nitrogen findings, the plasma bilirubin values, in the present case, were directly proportional to the severity of the disease. During the

period of examination the plasma bilirubin rose steadily from 22 units to 32 units at death. The plasma gave a positive direct reaction to the van den Bergh test with only ½ minute delay on 27.6.40, about 10 days after the onset of symptoms, and this finding, along with the absence of pigment in the stools led to the erroneous diagnosis of catarrhal jaundice. In the terminal stage of the disease the plasma gave a positive direct reaction to the van den Bergh test with 1½ minute delay and the stools contained bile pigment.

The main points of interest in this case of true acute yellow atrophy may be summarised as follows:

I) The absence of clinical signs and symptoms, apart from the jaundice, for about 12 days after the onset of the liver damage.

II) No increased blood concentration until about 48 hours before death.

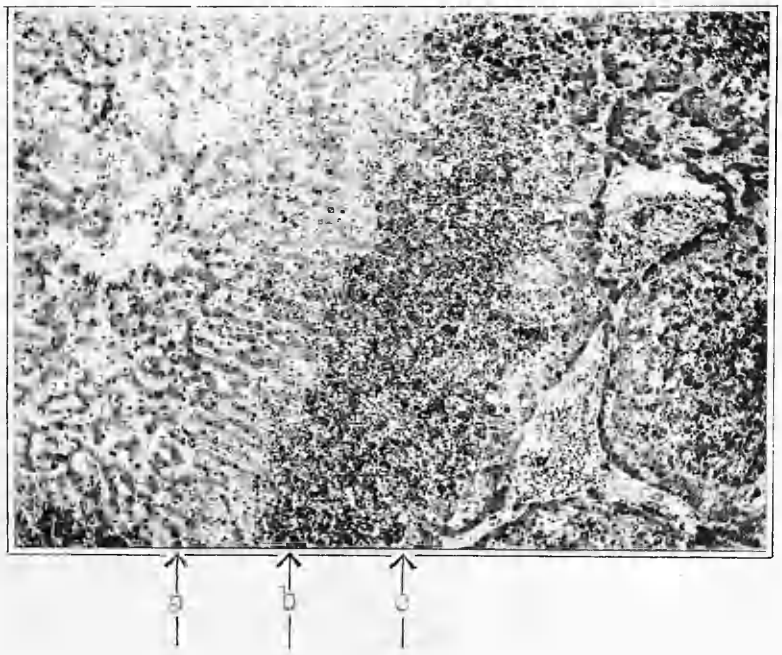
III) Normal plasma uric acid and low blood urea values until just before death; a retention of both plasma uric acid and urea followed the increase in blood concentration.

IV) A steadily rising plasma bilirubin content throughout the illness and a positive delayed direct reaction to the van den Bergh test.

Delayed Chloroform Poisoning.

Poisoning due to chloroform causes yet another type of liver lesion and cases of delayed chloroform poisoning

PLATE VIII.



Liver in Delayed Chloroform Poisoning showing mid-zonal necrosis.

a , Surviving centrolobular cells.

b , Band of necrosis.

c , Surviving periportal cells.

(Case 137)

Low power view.

are occasionally met with in the puerperium. Sheehan (1940), has pointed out that the acidosis and dehydration resulting from a long labour, make perturient women specially susceptible to the ill effects of this drug. He has found that the liver lesion in these dystocia cases is a mid-zonal necrosis which spreads centrally in severe cases.

A fatal case, case 187, of delayed chloroform poisoning was examined during the present study but no biochemical findings were obtained from it. The patient had been given chloroform for 20 minutes on two occasions. She had had moderately severe vomiting for 48 hours before the first administration of chloroform and this sickness continued during the next day when labour began. Vomiting was severe and the patient was very acidotic. Chloroform was again given for the delivery after a 4 hour labour. The patient's condition after delivery was very poor. She died 6 hours after the delivery and about 72 hours after the first administration of chloroform. At the autopsy, the liver weighed 1,220 gms. Microscopically there was very definite mid-zonal necrosis with fatty degeneration of the centrolobular cells; a microphotograph of this is shown in Plate VIII. for comparison with the liver lesions in cases 185 and 186.

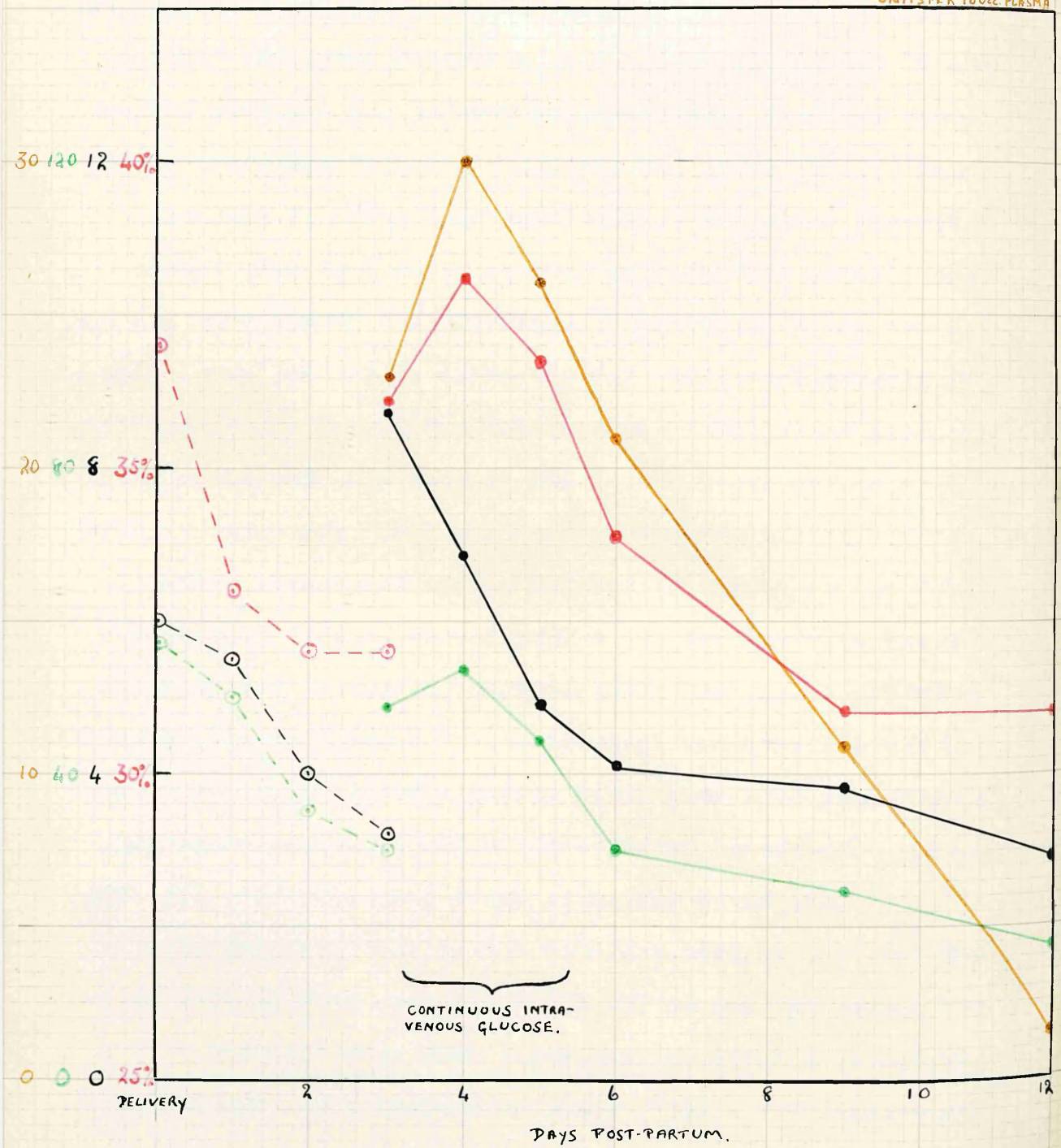
Three very severe but non-fatal cases, (188, 189 and 190), and two milder cases, (135 and 191), of delayed chloroform poisoning have been investigated during the present study and it is interesting to compare the biochemical

changes found in these cases with those found in the cases of the two other types of liver damage, previously described. In each of the severe cases chloroform was given for the delivery after a long labour; the mean length of labour for the 3 cases was 52 hours. All the patients vomited frequently during labour and were, therefore, dehydrated and acidotic before delivery. In cases 189 and 190 chloroform was given on two different occasions during labour and again at delivery. Case 189 was delivered by forceps after a 52 hour labour and was under chloroform anaesthesia for 50 minutes; 200 c.c. 50% glucose were given intravenously at the delivery. One of the mild cases (191) received chloroform for 25 minutes after a 51 hour labour; 200 c.c. 50% glucose had been given half-an-hour before the anaesthetic was started. The other mild case (135) was an eclamptic treated by the Stroganoff method. Chloroform was given for a few minutes on four different occasions during and after labour. Labour lasted 24 hours and the delivery was spontaneous. This was the only eclamptic patient out of a very large series treated by the Stroganoff method, who showed definite signs of chloroform poisoning.

All these patients, cases 188, 189, 190, 191, ~~211~~, ~~212~~ and 135, vomited a little during the first two days of the puerperium and on the third day they were noticed to be jaundiced; temperature and pulse rate were elevated - Temperature 99° - 101°, pulse rate about 120. The severe cases became restless and later delirious; two of them, cases

CHART XXIII.

Composite Graph of findings in three cases of severe Delayed Chloroform Poisoning. (Cases 188, 189 & 190. Average length of labour: 52 hours.)



188 and 189, were completely comatose by the fourth day post partum. The temperature ranged from 103° to 105° and the pulse was very weak and rapid - often over 160 per minute. Continuous intravenous glucose was started on the third day post partum: slight improvement (fall in temperature and pulse rate and signs of returning consciousness) began on the fifth day post partum and persisted. In the mild cases the jaundice lasted only a few days and there were no mental symptoms; in the severe cases the jaundice became very deep and lasted for about 2 weeks. Bile pigments were present in both urine and faeces. The biochemical findings throughout the course of the disease in each case are given in the appendix. A composite chart of the changes in the various blood fractions in the three severe cases is given in Chart XXIII. Investigation of these cases was not started until the 3rd day post partum and therefore the changes occurring in the blood fractions immediately after delivery are not known. In the graph, however, the expected changes in haematocrit, plasma uric acid and urea in a normal case with a 52 hour labour, during the first 3 days post partum, are indicated with dotted lines, for comparison with the findings in the present cases; the values have been calculated from the data recorded in Sections II. and IV. It will be noticed that the haematocrit values during the acute stage of the illness were relatively high and that improvement coincided with a decrease in the haematocrit values. In each case the plasma gave a positive delayed direct van den Bergh reaction and the plasma bilirubin

was very high (24 to 32 units in severe cases); the plasma bilirubin appeared to vary with the severity of the clinical condition. Plasma uric acid and urea were raised in each case. The plasma uric acid values were relatively higher than the urea values in individual cases and the maximum urea value was usually reached a day later than the maximum uric acid value. A fall in the plasma uric acid level occurred in each case at least 24 hours before there was any other biochemical or clinical sign of improvement; apart from this, however, the plasma uric acid and urea values followed the haematocrit changes.

In each case treatment with intravenous glucose was instituted as soon as the diagnosis was made: in three of the cases the diagnosis was suggested from the biochemical findings before there was any jaundice clinically. In the severe cases continuous drip transfusions were given. All the patients recovered in spite of the fact that two of them, cases 188 and 189, were moribund for more than 24 hours. The favourable results in these cases appeared to be due to the early and continued intravenous glucose therapy and they support the view expressed by Sheehan (1940) and Townsend (1939) that the prognosis in cases of delayed chloroform poisoning, even with much liver destruction, should not be unduly pessimistic.

The main biochemical findings in the blood in these cases of delayed chloroform poisoning are

I) An increase in haematocrit value.

II) High plasma uric acid and moderately raised blood urea values. A fall in plasma uric acid value was found to be the first sign of improvement; it occurred while the urea value was still rising.

III) A positive delayed direct reaction to the van den Bergh test and very high plasma bilirubin values. The plasma bilirubin values were directly proportional to the severity of the clinical condition.

Discussion.

The main findings in these three types of liver damage, as shown by the present cases, are summarised in the following table IX.

Table IX.

	Obstetric Acute Yellow Atrophy.	True Acute Yellow Atrophy.	Delayed Chloroform Poisoning.
iver Lesion.	Centrolobular cytoplasmic change. No necrosis.	Widespread azonal necrosis.	Mid-zonal necrosis with fatty change in surviving cells.
sociated nal changes.	Degeneration of tubular epithelium and early interstitial fibrosis.	Kidney apparently normal.	Marked fatty change in the epithelium of the 1st convoluted tubules.
lood concentration.	Markedly increased.	Unchanged.	Increased.
lasma uric acid.	Very high.	Normal.	High.
lood Urea.	High.	Slightly subnormal.	Moderately high.
an den Bergh eaction.	Positive direct with 2 minutes delay.	positive direct with 1½ minutes delay.	Positive direct with ½ minute delay.
lasma Bilirubin.	High.	High.	High.

In each of these conditions there are definite pathological changes in the liver although in obstetric acute yellow atrophy there is no necrosis of liver cells. The only biochemical abnormality common to the three conditions is the raised plasma bilirubin and in each case the height of the plasma bilirubin was a good index of the severity of the disease. The changes in the blood non-protein nitrogen fractions, however, appear to have no direct relation to the extent of the liver damage. In each condition there are at least three factors which might influence the blood non-protein fractions and these are, - liver damage, kidney damage and anhydraemia. In the case of true acute yellow atrophy of the present series the only one of these factors present, in the early stages of the disease, was the liver damage and at this time the blood urea value was normal. Immediately before death, however, a marked increase in blood concentration developed and there was a simultaneous rise in both the plasma uric acid and urea - presumably the result of the anhydraemia. The uric acid findings in this case are specially interesting; they indicate that gross liver damage does <u>not</u> cause an increase in plasma uric acid but that a rise in plasma uric acid may develop, in such a case, as a result of anhydraemia. The fact that an actual rise in blood urea occurred terminally indicates that liver damage - as widespread as in the present case - is insufficient to cause much reduction in urea formation. It may therefore be concluded that even gross liver damage has little <u>direct</u> effect on the plasma uric acid

and urea levels in the blood.

In obstetric acute yellow atrophy and delayed chloroform poisoning there is a definite retention of non-protein nitrogen fractions in the blood. The changes in plasma uric acid and urea again appear to have a definite relation to the changes in blood concentration but in these cases there is associated renal damage which is presumably the primary cause of the retention.

No investigation of acid-base balance has been made in the present cases although the possibility that some of the changes may have resulted from an upset of this balance has not been overlooked. The increase in blood concentration, found in many of these cases, did not result from prolonged vomiting or dehydration; it was presumably due to some acid base imbalance.

Summary.

The biochemical findings in a fatal case of obstetric acute yellow atrophy and in one of true acute yellow atrophy and in five non-fatal cases of delayed chloroform poisoning are recorded; pathological findings in the two former cases and in one fatal case of delayed chloroform poisoning are also given. The height of the plasma bilirubin is found to be the best index of the severity of the liver damage. In each condition there was increased blood concentration at some time during the illness

and changes in the non-protein nitrogen fractions of the blood appeared to depend directly on changes in blood concentration rather than on the extent of the liver damage. A high plasma uric acid was not found to be the invariable result of liver damage.

S E C T I O N VII.

CHANGES IN BLOOD BIOCHEMISTRY ABOUT THE TIME OF DELIVERY WITH SPECIAL REFERENCE TO OBSTETRIC SHOCK.

During recent years attention has been drawn to the similarity of the syndrome of adrenal cortex insufficiency to secondary shock and it has been suggested that the symptoms and signs of secondary shock are due to temporary exhaustion of the adrenal cortex.

In 1933 Swingle, Pfiffner, Vars, Bott and Parkins investigated the function of the adrenal cortical hormone and the cuase of death from adrenal insufficiency, using adrenalectomised dogs which had been kept alive and normal for several years with injections of cortical extract. Withdrawal of the hormone caused a progressive decrease in blood volume and a fall in blood pressure until death occurred 8 to 12 days later. Haemoconcentration and increased blood viscosity were marked, depending on the decrease in plasma volume. The heart rate varied inversely with the blood pressure as did blood non-protein nitrogen and urea values. The dogs in this shock-like condition were found to be unable to dilute their blood at all. If a small haemorrhage occurred their condition became rapidly worse and death occurred in a short time; even when fluid was given intra-

:peritoneally no blood dilution occurred. When cortical hormone was injected, however, the fluid was absorbed, blood dilution occurred and the animal recovered. One function of the adrenal cortical hormone therefore appeared to be the regulation and maintenance of a normal circulating volume. The authors emphasize the similarity between the symptoms and signs of adrenal insufficiency and those of secondary shock. Lock (1932) drew attention to the similarity of the clinical picture of adrenal insufficiency to that of diseased conditions in which a loss of inorganic base played an important part, e.g. high intestinal obstruction, cholera, and diabetic acidosis - such pathological states being characterised by prostration, dehydration, nitrogen retention, low plasma bicarbonate and severe "shock". He found low blood sodium and high blood potassium in 3 cases of Addison's disease. Loeb, Atchley, Benedict and Leland (1933) made electrolyte balance studies on dogs before and after adrenalectomy and found a fall in blood sodium, chloride and bicarbonate with an increase in blood potassium and non-protein nitrogen to occur after adrenalectomy. The fall in blood sodium was shown to result from an excessive loss of sodium in the urine and it was suggested that the adrenal glands must have a regulatory effect on sodium metabolism and renal function. The relation of the adrenal cortex to the sodium metabolism has been further investigated by many other workers (Loeb, Atchley, Gutman&Jillson, 1933; Zwemer,

1934; Zwemer and Sullivan, 1934; Loeb, 1935; Hartman, Lewis and Toby, 1938. High salt diets have been used with some success in the treatment of Addison's disease and other types of adrenal cortical insufficiency (Rogoff and Stewart, 1928; Loeb, 1932; Harrop, Weinstein, Soffer and Trescher, 1933; Zwemer, 1934). More recently active adrenal cortical extracts have been prepared and are now used in the treatment of all types of adrenal cortical insufficiency (Harrop and Weinstein, 1932; Harrop, Weinstein, Soffer and Trescher, 1933; Kendall, 1935; Swingle, Parkins, Taylor and Hays, 1938). The marked similarity of the symptoms and signs of the adrenal deficiency syndrome to those of surgical shock has suggested that adrenal exhaustion may be a factor in the etiology of traumatic and surgical shock. Many experimental studies in dogs in which adrenal cortical hormone was used in the treatment of shock, induced by trauma and by adrenaline injections, have been performed with successful results (Swingle, Parkins, Taylor and Hays, 1937 a & b; Parkins, Swingle, Taylor and Hays, 1938). Recent work by Swingle, Parkins, Taylor and Hays (1937 a & b, and 1938) has tended to indicate that adrenal cortical hormone has a direct action in the low blood pressure of shocked dogs apart from any control it exerts on fluid and electrolyte balance.

The biochemical changes in the blood resulting from adrenal cortex insufficiency are summarised by Loeb (1935) as follows:-

I) Decrease in serum sodium.

II) Increased blood concentration.

III) Retention of non-protein nitrogen fractions.

IV) Increase in blood potassium.

At present very little information has been published with regard to the effect of adrenal cortical hormone in cases of shock in human beings. Reed (1938), used adrenal cortical extract in 50 surgical cases - some after shock had developed and some prophylactically. The treated cases were found to improve quickly after operation - pulse rate and heart action were stabilised. In one or two cases biochemical findings were noted: in untreated shock cases blood sodium and chloride fell markedly but non-protein nitrogen did not rise: in treated shock cases blood sodium and chloride remained unchanged. Wilson, Rowley and Gray, (1936), Wilson, MacGregor and Stewart (1937), and Wilson and Stewart (1939) studied changes in blood chemistry in cases of shock due to burns and found a definite fall in serum sodium while serum potassium, blood chloride, non-protein nitrogen and urea increased. They report that injections of desoxy-:corticosterone, a synthetic adrenal cortical preparation, caused rapid restoration to normal of the serum sodium level and other blood abnormalities but that this drug had only an occasional beneficial effect on the circulatory failure in these shock cases.

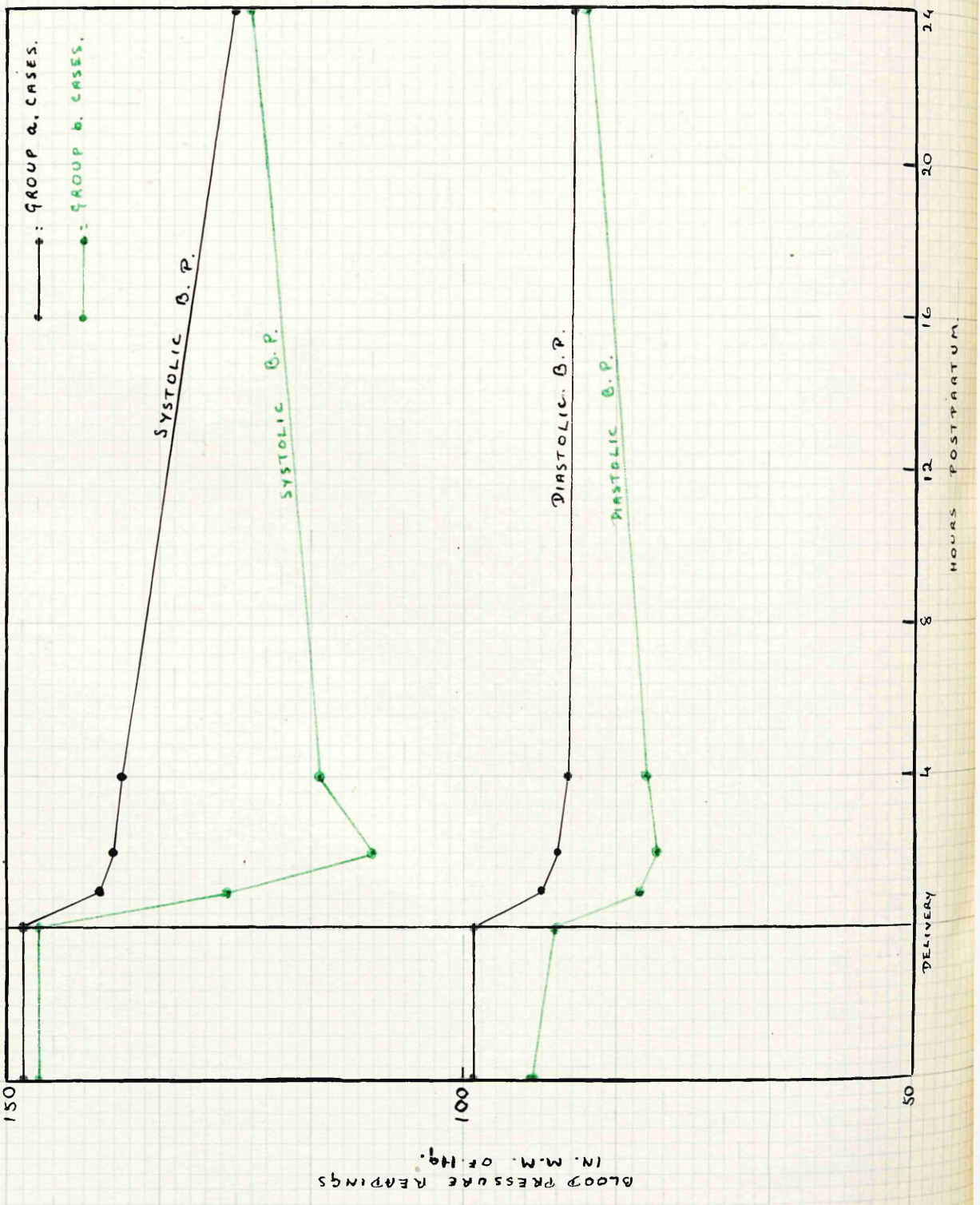
A study of cases of obstetric shock reveals several factors which suggest that adrenal cortical exhaustion

may possibly play a part in this syndrome. There is a close resemblance between the clinical condition of shocked patients and that of animals suffering from adrenal cortex insufficiency; blood pressure falls very low and even very slight haemorrhage rapidly causes death. Freeman (1933), working with cats found that prolonged hyperactivity of the sympathetic nervous system brought about a decrease in blood volume and what he calls a "pre-shock state"; if the animal lost a very small amount of blood, when in this condition, a precipitate fall in blood pressure resulted and death occurred in a very short time. In certain obstetric patients there is prolonged sympathetic overactivity, due to the anxiety and foreboding of the days before the onset of labour, which makes them less able to stand the exertion and trauma of delivery; such very nervous patients are especially liable to develop obstetric shock. Increased adrenalin output is presumably a factor in causing this type of shock;,Parkins, Swingle, Taylor and Hays (1938) produced shock in dogs by giving large doses of adrenalin and they found that this type of shock responded well to treatment with adrenal cortical hormone.

In the present investigation a clinical and biochemical study of some patients suffering from varying degrees of obstetric shock has been made in an attempt to detect any evidence of adrenal exhaustion in this condition. The changes occurring in haematocrit values, plasma sodium, whole blood chlorides and blood urea have been determined in

CHART XXIV.

Mean Blood Pressure Changes from Cases of Groups a & b.



a series of 26 pregnant women about the time of delivery. A careful watch was kept on the clinical condition of each patient for signs of collapse, especially during the first few hours after delivery; blood pressure readings and pulse rate were taken at half hourly intervals in most cases. The patients have been divided, from a clinical standpoint, into 3 groups:-

a) 12 Cases (192 to 203) with no post-partum collapse.

b) 8 Cases (204 to 211) with slight post-partum collapse.

c) 6 Cases (212 to 217) with marked post-partum collapse.

Each group includes patients delivered spontaneously and by forceps, patients with short and long labours and toxaemic and non-toxaemic patients. In group c. there are 2 cases of retained placenta.

Blood specimens were taken off during labour, usually in the second stage and at approximately 1, 2, 4 and 24 hours after delivery. In group c. cases however, it was found to be impossible to obtain specimens of blood at regular intervals of time because the patients were so collapsed and because in most cases blood transfusions were being given. Occasional specimens were obtained, however, and if each case is considered separately the changes occurring can be followed.

Mean systolic and diastolic blood pressure readings from the patients of groups a. and b. are charted in Chart XXIV. to show the changes occurring during the period of

investigation. The absolute values charted are not of importance as both groups included several patients suffering from hypertensive toxaemia, causing a wide range of values. As might be expected there was a slight and gradual post-partum fall in systolic blood pressure in the normal cases and a sudden post-partum fall with subsequent rise in the systolic blood pressures of the cases with slight post-partum collapse. The wide variation between the two systolic curves in Chart XXIV. is significant, but the diastolic curves do not show the same difference. As a rule pulse rate varied inversely with the blood pressure readings.

The mean haematocrit changes in the same patients during the first four hours post-partum are shown in Table X.

Table X.

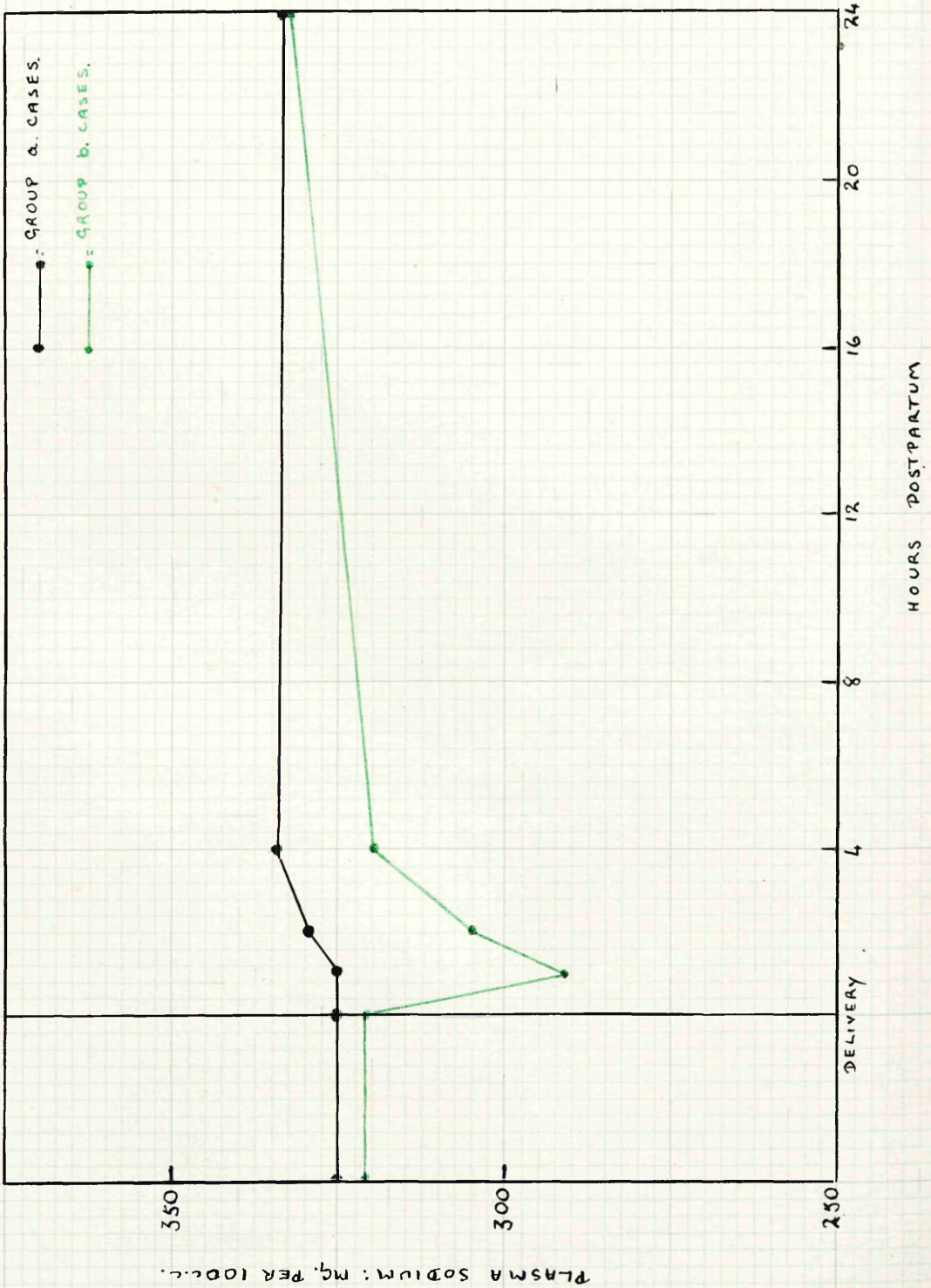
Mean Haematocrit values from Groups a. and b. before and after delivery.

		Antepartum.		Postpartum.			
	No. of Cases.	Immediately before delivery.		1 hr.	2 hr.	4 hr.	24 hrs.
		Mean.	Range.				
Group a.	11	38%	31 to 43	37%	36%	35.5%	31%
Group b.	6	42%	34 to 50	41%	41%	39.5%	33%

The mean values from the group a. cases show the normal post-partum decrease described in Section IV.; in the group b. cases high values are maintained for several hours after delivery. The range of ante-partum values in each group is wide but high values predominate in group b.; they are

CHART XXV.

Mean Plasma Sodium Changes from Cases of Groups a & b.



presumably due to diminished blood volume. It will be shown later that corresponding ante-partum changes in the plasma sodium occur in many of these cases.

The plasma sodium estimations were made by Rourke's modification of Kramer and Gittleman's pyroantimonate method (1928). In the present investigation the mean plasma sodium value from 30 pregnant women, near term or very early in labour was 332 mg. per 100 c.c. with a range of 321 mg. to 347 mg. per 100 c.c. The mean plasma sodium findings in patients of groups a. and b. during the first 4 hours post partum, are shown in Chart XXV. and Table XI.

Table XI.

Mean Plasma Sodium and Whole Blood Chloride changes in Groups a. and b.

	Group a.				Group b.			
	Plasma Sodium in mg. per 100 c.c.		Whole Blood Chloride in mg. per 100 c.c.		Plasma Sodium in mg. per 100 c.c.		Whole Blood Chloride in mg. per 100 c.c.	
	Mean.	Range.	Mean.	Range.	Mean.	Range.	Mean.	Range.
Before delivery.	325	311 to 344	558	481 to 656	321	301 to 337	514	428 to 603
½ hour post partum.	325	317 to 339	573	475 to 632	291	266 to 305	528	480 to 612
2 hours post partum.	329	319 to 348	578	510 to 632	305	285 to 330	537	445 to 612
3 hours post partum.	333	319 to 352	594	542 to 648	320	301 to 337	538	516 to 709
4 hours post partum.	332	319 to 350	631	565 to 664	333	320 to 346	575	498 to 630

CHART XXVI: Findings from Individual Cases of Group a.

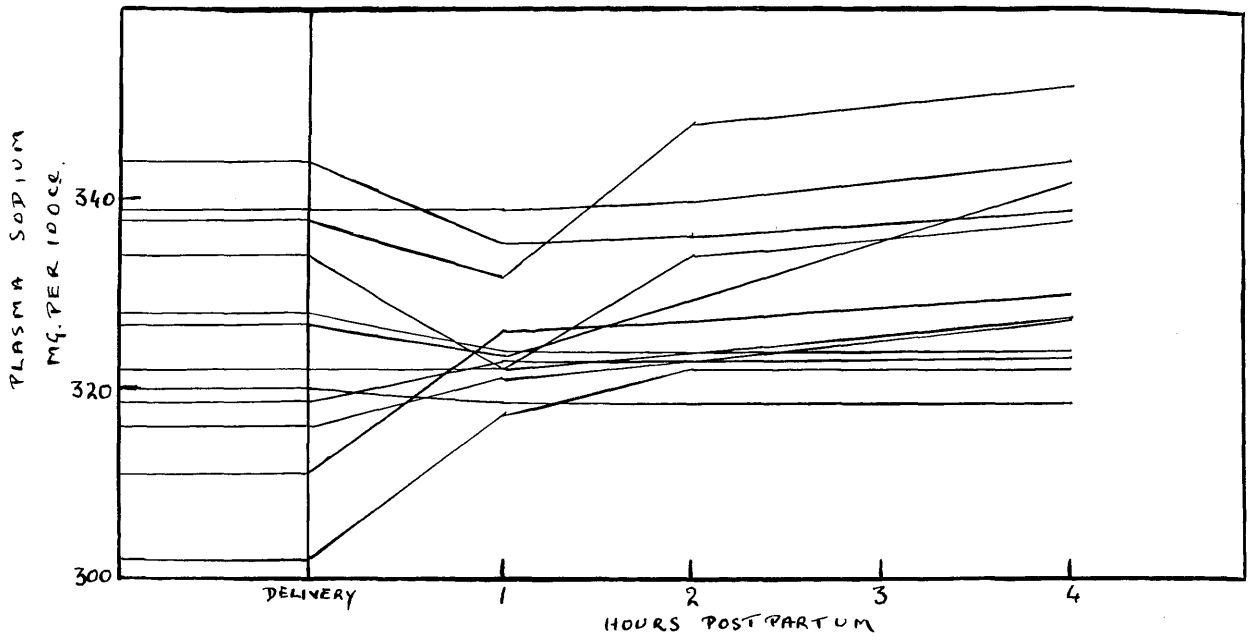
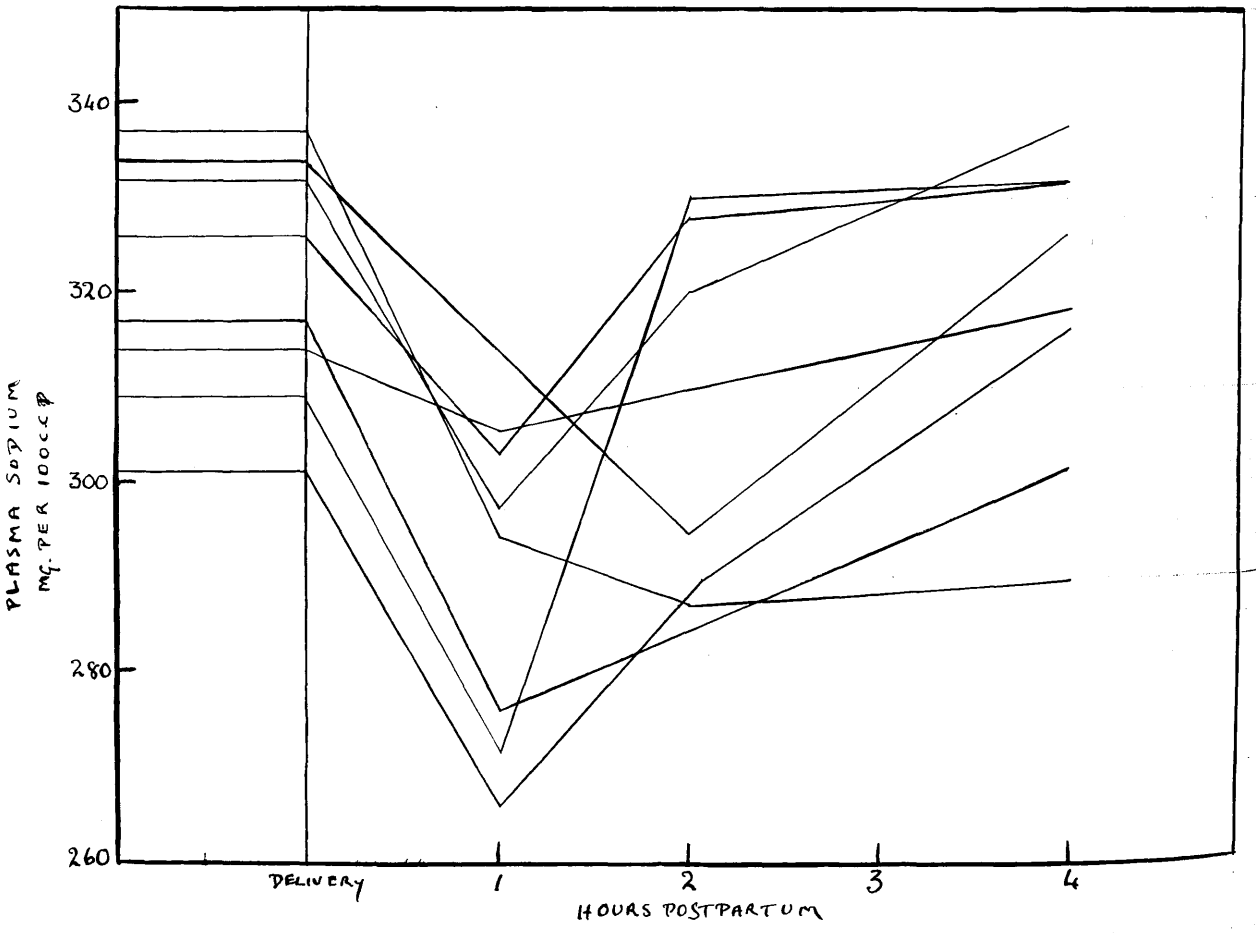


CHART XXVII: Findings from Individual Cases of Group b.



It will be noted that there is a significant drop in the plasma sodium values after delivery in the cases of group b., with return to normal at 4 hours post partum. The mean plasma sodium values for the 12 normal cases show practically no change during the same period. In Charts XXVI. and XXVII. the values from the individual patients of groups a. and b. respectively have been plotted separately in order to demonstrate the consistency of the results and the timing of the fall in plasma sodium values. In most cases the lowest values occur at 1 hour post partum with a rapid return to normal levels at 2 hours post partum. In a few cases the fall continues till 2 hours post partum and the values return more slowly to normal. All values are within normal limits at 24 hours post partum. It is of interest to note that in both groups a. and b. there are a few cases with low plasma sodium values before delivery; in Chart XXVI. (from group a.) the values from such cases show a rise towards normal levels immediately after delivery while in Chart XXVII. (from group b.) the curves from all cases fall immediately post partum. These cases with low ante-partum plasma sodium values all had high ante-partum haematocrit values. The significance, ~~therefore~~, of this finding will be discussed later.

Whole blood chloride determinations carried out at the same times as the plasma sodium determinations and on the same patients do not reveal similar changes. It was not

found possible, in the present circumstances, to estimate plasma chlorides because of the difficulty in maintaining anaerobic conditions. The estimations of whole blood chloride were made by Whitehorn's modification of Volhard's method (1921). The mean results from patients of both groups a. and b. show a steady rise in whole blood chloride from before delivery to 24 hours post partum. The results are given in Table XI. There are wide variations in the whole blood chloride values obtained from individual patients of both groups due to factors not under consideration at present, so that the difference between the mean values of the two groups is not of significance. The blood urea findings were very variable and in most cases the changes could be regarded as a result of the length of the labour. In 4 of the severely shocked patients (cases 213, 214, 216 and 217), however, there was a greater rise during the period of shock than could be accounted for by the labour. In such cases the increase in the blood urea is presumably the result of the shocked condition.

The values for haematocrit, plasma sodium and blood pressure from the individual patients of group a., which have been used to construct the various graphs, are shown together in the composite table, Table XII., in order that the relation between the various changes in each individual patient may be compared. A similar table, Table XIII. is given for the findings from group b. cases. It will be

Table XII.

12 Cases with No Post-Partum Collapse - Group a.

	Before Delivery			1 hr. post-partum			2 hrs post-partum			4 hrs post-partum			24 hrs. post-partum.		
Case No.	Blood Pressure.	Pl. Sodium mg. per 100 c.c.	Haem't. %	Blood Pressure.	Pl. Sodium mg. per 100 c.c.	Haem't. %	Blood Pressure.	Pl. Sodium mg. per 100 c.c.	Haem't. %	Blood Pressure	Pl. Sodium mg. per 100 c.c.	Haem't. %	Blood Pressure.	Pl. Sodium mg. per 100 c.c.	Haem't. %
192	110/90	319	42.5	130/80	324	42	105/65	324	42	110/75	323	42	120/80	319	35
193	130/110	338	39	120/95	332	40	123/95	348	39.5	110/95	352	37	120/70	332	30
194	130/80	344	40	85/65	335	39.5	115/85	336	40	115/80	339	40	120/90	344	35
195	145/80	327	37	150/105	323	36	-	-	-	140/90	341	33	125/80	336	28
196	185/115	302	41	165/110	317	40	150/110	323	39	140/90	323	39	125/100	337	35
197	180/130	339	32.5	175/125	339	28	-	340	27	160/120	344	26	145/100	324	20
198	165/120	320	42	135/80	319	38	-	319	40	150/100	319	41	140/100	320	35
199	155/90	334	40	160/95	322	38	150/90	334	37	150/90	339	35	130/80	350	32.5
200	-	311	44	-	324	47	-	-	-	-	-	-	-	337	41
201	165/100	320	31	160/90	322	32	170/95	324	33	160/90	328	35	140/90	324	30
202	115/70	316	31	125/70	322	31	115/60	323	31	115/60	328	30	110/60	337	28
203	-	328	38.5	-	324	36	-	324	35	-	-	-	-	320	32

Table XIII.

8 Cases with Slight Post-Partum Collapse - Group b.

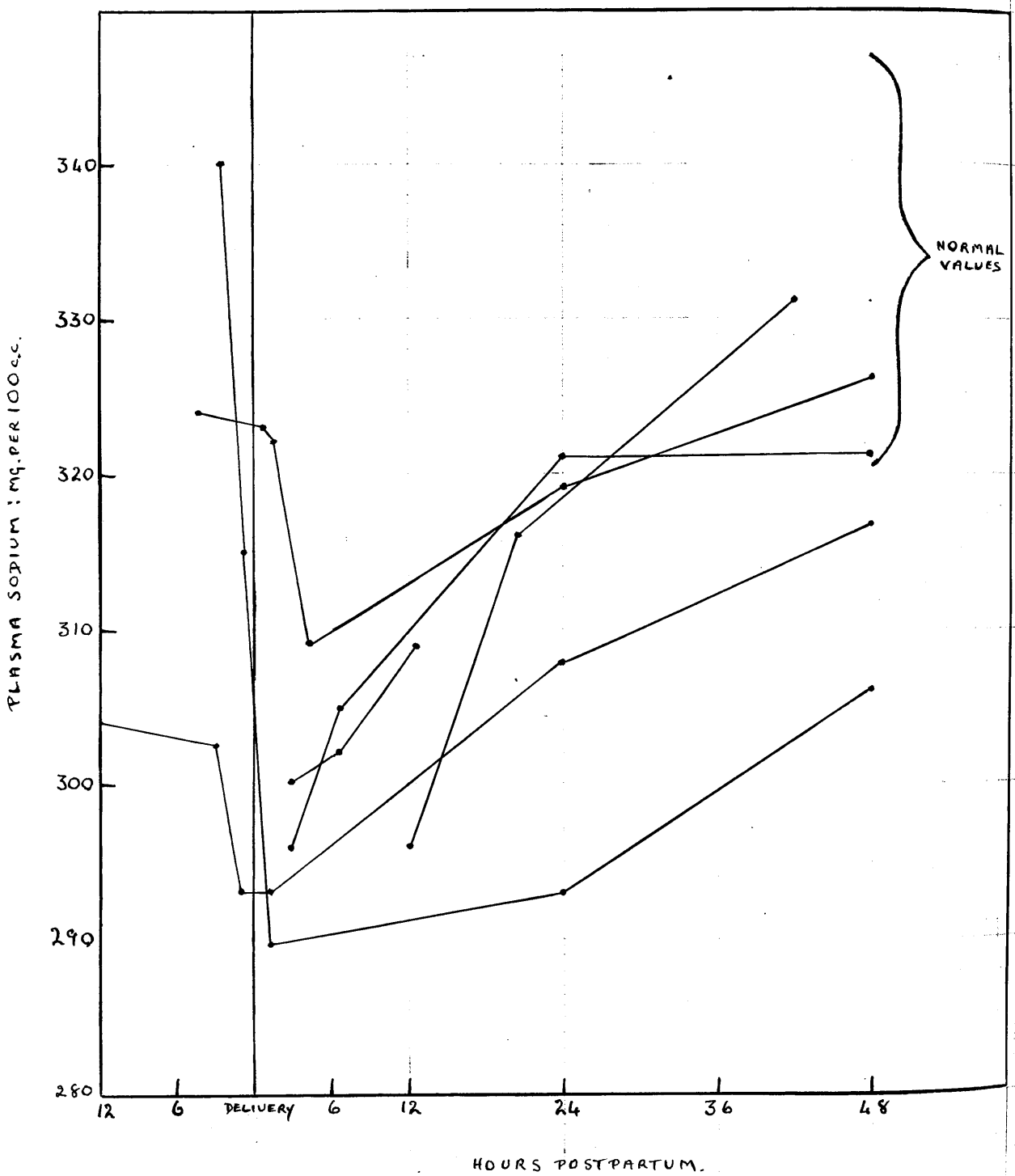
Case No.	Before Delivery			1 hr. post-partum			2 hrs. post-partum			4 hrs post-partum			24 hrs. post-partum		
	Blood Pressure.	Pl. Sodium mg. per 100 c.c	Haemt. %	Blood Pressure.	Pl. Sodium mg. per 100 c.c	Haemt. %	Blood Pressure.	Pl. Sodium mg. per 100 c.c	Haemt. %	Blood Pressure.	Pl. Sodium mg. per 100 c.c	Haemt. %	Blood Pressure.	Pl. Sodium mg. per 100 c.c	Haemt. %
204	-	301	43.5	-	266	42	-	288	42	-	316	40	-	320	36
205	145/75	309	41.5	105/70	272	42	90/55	330	42	100/70	332	41	128/70	334	32
206	145/90	317	33.5	135/75	275	35	90/60	-	-	105/70	311	32	110/60	343	27
207	140/90	332	41	105/65	297	40	115/60	320	40	115/60	337	40	120/70	346	36
208	130/60	337	43	105/45	294	41	100/60	287	-	98/65	-	-	110/70	335	35
209	185/120	314	37	115/100	305	31	150/115	310	30	150/115	318	28	140/100	324	23
210	140/110	326	40	145/100	303	38	110/80	328	38	120/90	332	34	135/90	332	31
211	160/100	334	50	150/120	315	48	125/115	295	48	-	326	45	115/80	326	35

noticed from these tables that in individual cases there is neither a constant proportion between the decrease in plasma sodium and the fall in blood pressure, nor does the minimum plasma sodium value coincide with the lowest blood pressure reading. In all cases with slight collapse, however, a fall in blood pressure, a rise in pulse rate, and a lowering of plasma sodium level occur immediately post partum.

It has not been found possible to arrange the results from the cases of group c., those with severe post-partum collapse, in tables or graphs. Each case must be studied by itself. As one would expect, during the period of severe shock the blood pressure was always extremely low e.g., readings of 40/30 - 55/50 were often recorded and occasionally no reading at all could be obtained. It was noticed in this group of cases that a small pulse pressure was an important sign of commencing or established shock and that a decrease in pulse pressure was more significant than a fall in systolic blood pressure. As in group b. cases there was no absolute proportion between the blood pressure and the plasma sodium level. In 6 patients during severe shock the plasma sodium ranged from 289 to 300 mg. per 100 c.c. with a mean value of 297 mg. per 100 c.c. These values are subnormal but there was no definite relation between the degree of shock and the plasma sodium value; several patients from group b. had lower plasma sodium values during their slight collapse than had any of the patients of group c. In the latter group,

CHART XXVIII.

Graph of Plasma Sodium Findings in 6 cases of severe postpartum collapse.



however, a longer time elapsed before the plasma sodium regained normal levels.

The plasma sodium changes about the time of delivery in the 6 cases of group c. are shown in Chart XXVIII. and the other findings are given in the appendix. It will be noticed that the plasma sodium values were very low after delivery in all cases. Three of the cases (212, 213 and 214) were only admitted to hospital after delivery. They were severely shocked and in each there had been severe haemorrhage; the haematocrit values in these cases were therefore low.

In case 212 the plasma sodium remained low and the patient died in a few hours in spite of blood transfusion being given. Post-mortem examination revealed an adherent placenta and signs of obstetric shock.

Case 213 remained severely shocked for several hours and during this time the plasma sodium value was subnormal. The next day the patient was improved and the plasma sodium value was normal.

In case 214 normal plasma sodium values were regained 16 hours after delivery and the signs of shock had disappeared.

Case 215, a pre-eclamptic, showed marked signs of exhaustion during labour. She was extremely anxious, cried without cause and complained of complete exhaustion. The pulse rate was rapid throughout labour and before delivery the pulse pressure became very small. The actual haematocrit readings were not greatly raised but they were relatively very high as compared with the value obtained on the day after delivery; that is, the post-partum decrease in values was excessive considering that there was no loss of blood at delivery. The plasma sodium values in this case fell during labour and became very low at the time of delivery. The patient became collapsed about 1½ hours after an easy forceps delivery but recovered with treatment.

Case 216 became collapsed 1 hour after delivery but the plasma sodium values remained within normal limits till at least 2 hours after delivery. At 4 hours post partum the plasma sodium value had fallen considerably but regained

normal levels by the next day.

Case 217 became extremely shocked 3 hours after delivery. She improved slightly with treatment but collapsed several times later. Her general condition remained extremely poor and she died 2 days later. Post-mortem examination revealed generalised peritonitis, - apparently due to direct spread from uterus and the broad ligament, - and patchy liver necrosis. Plasma sodium values showed a marked fall in this case, immediately post partum in spite of the administration of intravenous saline (there is usually a rise in plasma sodium for 1 to 2 hours after intravenous saline), and the values remained low during the long period of collapse.

Discussion.

The results recorded here indicate that, in women who show signs of slight or severe post-partum collapse, the marked fall in blood pressure is accompanied by a fall in the plasma sodium level. The fall in blood pressure is approximately proportional to the degree of collapse, as one might expect, but the decrease in plasma sodium is not so related. It is very difficult to assess the significance of the transitory lowering of the plasma sodium in these cases. This finding, however, tends to support the suggestion of previous workers, that shock results from an acute adrenal cortex insufficiency; the haematocrit and blood urea changes in the present cases, although not very striking, are at least consistent with such a theory. In Addison's disease the blood sodium often remains fairly normal until just before death but in such cases the destruction of the adrenal glands is very slow and the excessive sodium

excretion is compensated. It was not found possible, in the present study to determine the sodium excretion in the urine with any accuracy because of the difficulty in collecting all the urine passed about the time of delivery. The present results, however, are similar to those recorded by Reed (1938), in cases of surgical shock, and those of Wilson and Stewart (1939), in cases of burn shock, and it is possible that both the blood pressure and the plasma sodium changes are the result of adrenal cortex insufficiency.

On this theory, the high ante-partum haematocrit values and the low ante-partum plasma sodium values found in some cases, might, perhaps, be regarded as an indication of a pre-shock state, resulting from prolonged sympathetic overactivity before, and exhaustion during labour (on an anology with Freeman's experiments mentioned above). The development of definite post-partum collapse or shock, in these cases, would then depend on the amount of trauma or haemorrhage sustained at delivery. High ante-partum haematocrit values and low plasma sodium values are not, of course, an invariable finding in cases which later develop obstetric shock; for example, in anaemic women, the haematocrit values would be low even if some reduction in blood volume had taken place, and also, in many cases the trauma and haemorrhage sustained at delivery are sufficient to cause shock in the absence of predisposing factors. The conception of a pre-shock state is suggested only to explain the post-partum

CHART XXIX.

Blood pressure and pulse rate fluctuations in four casesof severe shock: effect of Eschatin administration.

collapse which occurs in certain cases after relatively easy deliveries; case 215, already described, is an example of this.

In several of these cases of obstetric shock treatment with "eschatin", an adrenal cortical extract, was tried. The results were not dramatic but in a few cases they were encouraging. The changes in systolic and diastolic blood pressure and pulse rate in four of the cases, before and during the period of shock and treatment, are given in Chart XXIX.

Case 213, was admitted to hospital very shocked; blood transfusion was given on admission but the patient collapsed again soon after. 8 c.c. eschatin were given intravenously when no blood pressure reading could be obtained and the patient was pulseless; an hour later the blood pressure was 35/30 and the pulse was very faint and irregular. Manual removal of the placenta was carried out at this stage; no anaesthetic was required as the patient was unconscious. Blood transfusion was again started but only 200 c.c. had run in when the needle blocked. Two hours after the eschatin injection the blood pressure was 50/28 and pulse rate 176. 5 c.c. eschatin were then given and the blood pressure continued to rise slowly throughout the evening and the patient gradually recovered.

In cases 215 and 216 the eschatin was given soon after the onset of collapse and it can be seen, from Chart XXIX., that a rise in blood pressure followed: the general condition

showed gradual improvement.

In case 216 no treatment apart from the eschatin was given; the blood pressure rose gradually for 4 hours after the eschatin injection but later fell slightly; a second injection of eschatin was given and within half an hour the blood pressure had begun to rise.

In case 218, a similar result was obtained - see Chart XXIX. - but no biochemical investigation was carried out.

In case 217, the eschatin appeared to have little effect on the blood pressure or general condition but it was found later that the signs of collapse were due to peritonitis.

In several other cases general improvement followed treatment with eschatin although it was difficult to estimate the part played by the eschatin as other forms of treatment were also given.

It is very difficult to assess the value of a new treatment, especially of a condition such as obstetric shock in which one can never estimate the spontaneous powers of recovery of the individual. The present cases suggest that adrenal cortical extract may be of value but much further investigation is necessary before any conclusions can be drawn. It would appear that response to the injection of extract does not begin for $\frac{1}{2}$ to 2 hours after the injection and then improvement is very gradual. This means that unless the extract is administered early, death may occur before

the action takes place. Prophylactic treatment with adrenal cortical extract will require to be investigated.

Summary.

A clinical and biochemical study has been made, about the time of delivery, on a series of patients including cases with no post-partum collapse, cases with slight post-partum collapse and cases with severe post-partum collapse. A decrease in plasma sodium is found in every case with clinical signs of collapse. A steady rise in whole blood chloride occurs in all cases from before delivery to 24 hours post partum. Changes in whole blood chloride are not found related to shock. Blood pressure, pulse, haematocrit, plasma sodium and urea findings in collapsed cases are consistent with the theory that temporary exhaustion of the adrenal cortex is a factor in obstetric shock. Treatment with adrenal cortical extract may be of value in some cases.

S E C T I O N VIII.

GENERAL SUMMARY AND CONCLUSIONS.

Individual summaries are appended to each of the principal sections of this thesis and therefore only a very short account of the contents of the work is given here.

In the introductory section the various theories relating to the etiology of eclampsia are mentioned, and the divergence of opinion of investigators with regard to the question of retention of non-protein nitrogen fractions in the hypertensive toxaemias of pregnancy is noted. The lack of detailed investigation of the changes occurring in the blood of individual patients throughout the course of the disease is stressed.

In Section II. the changes in the plasma uric acid and urea during the last few weeks of pregnancy, during labour and throughout the puerperium are studied in normal and non-convulsive toxaemic patients. A rise in these non-protein nitrogen fractions is found to occur always during labour and the height of the rise is dependent on the length of the labour in each case. The values return to normal levels during the first few days of the puerperium. These changes in plasma uric acid and urea are quite unrelated to toxaemia or other factors. The importance of these results

is that any interpretation of biochemical findings about the time of delivery which does not take into consideration the changes which have been shown to result from labour itself, is liable to be fallacious.

A similar detailed investigation of eclamptic patients is recorded in Section III. A series of eclamptic cases is studied before the onset of fits and it is shown that, contrary to the statements of previous authors, a high plasma uric acid value in a pre-eclamptic case is not an index of impending eclampsia. A marked and rapid increase in plasma uric acid level is shown to occur, in every case, immediately after the onset of fits and the extent of the rise is directly proportional to the number of the fits. The plasma urea findings do not show any such definite changes; in some cases, however, the urea level rises considerably late in the disease and this rise is related to a period of oliguria; in such patients the high plasma uric acid values are maintained or even increase after the fits have ceased until the urinary output is increased. Labour is found to cause the same changes in plasma uric acid and urea in eclamptic patients as is shown to occur in normal and non-convulsive toxaemic cases. Convulsions in non-pregnant cases and in animals are followed by changes in the plasma uric acid similar to those found in the eclamptic cases; it is therefore concluded that convulsions, per se, result in a rapid and marked rise in plasma uric acid and the cause of

this rise is discussed.

In Section IV. variations in blood concentration, during the last few days of pregnancy, during labour and throughout the puerperium, as measured by changes in the haematocrit value, are studied in a series of normal pregnant women, pre-eclamptic and chronic toxaemic patients. In the normal cases, a definite increase in blood concentration is shown to occur during the last few days before delivery, reaching a maximum value about the time of delivery. There is rapid blood dilution during the first 3 days post partum and after this, the blood gradually returns to normal. The changes involve the transfer of 20 to 25% of the fluid of the plasma from the blood into the tissues and back again. Pre-eclamptic cases show similar changes and in addition, there is an increase in blood concentration ante-partum related to the severity of the toxaemia in each case; oedematous cases have relatively lower haematocrit values. The chronic toxaemic cases are found to be of 2 types - those without anaemia and those with severe anaemia or hydraemia. Those without anaemia closely resemble the pre-eclamptic cases in their haematocrit changes while the anaemic cases show the normal haematocrit changes about the time of delivery but do not show any rise in values during the puerperium. The post-partum dilution of the blood is increased in patients in whom there is oedema before delivery, haemorrhage at delivery or infection in the puerperium.

Section V. consists of some biochemical studies in several different conditions characterised by severe vomiting. In early pregnancy the most important cause of severe vomiting is hyperemesis gravidarum. High haematocrit values, raised plasma uric acid and urea values are found in a group of patients suffering from severe hyperemesis; increased plasma bilirubin occurs in a few cases. This nitrogen retention disappears rapidly as the anhydraemia is relieved. The severity of the clinical signs in any individual case is found to depend on the degree of anhydraemia and the development of Wernicke's encephalopathy. Several fatal cases are described to illustrate the relative importance of these two factors.

In late pregnancy, cases with severe vomiting as their main symptom are found to be of several different types: they are classified in four groups.

I) Prolonged Hyperemesis:- In this group of cases the biochemical findings are the same as those in the severe hyperemesis cases and appear to be the direct result of the vomiting. The changes disappear rapidly when intravenous glucose is administered. It is concluded that these patients are suffering from hyperemesis gravidarum which has continued into the late months.

II) Pyelitis of Pregnancy:- In this group of cases the characteristic biochemical findings are, a very low haematocrit value in spite of severe vomiting, normal plasma uric acid

and urea values except late in the disease in severe cases when a retention of both non-protein nitrogen fractions is found, and, in some cases, a positive immediate direct reaction to the van den Bergh test and a slight increase in plasma bilirubin. A very marked increase in the non-protein nitrogen fractions of the blood is found to occur during labour in these cases.

III) "Erythroblastosis foetalis":- Severe vomiting is found to occur in a group of patients associated with foetal and placental abnormalities. Plasma uric acid values are very high and plasma urea moderately raised in each case. Slight jaundice and raised plasma bilirubin are found early in the disease. The etiology of the condition is not known.

IV) A miscellaneous group:- including cases of hypertensive toxaemia, acute yellow atrophy of pregnancy and idiopathic ones. No biochemical findings are given for cases of this group.

In Section VI. conditions with jaundice as a prominent feature are dealt with. The etiology of the slight icterus, positive van den Bergh and raised plasma bilirubin, found in some cases of eclampsia, hyperemesis, and pyelitis is discussed.

A fatal case of obstetric acute yellow atrophy is described with the biochemical and pathological findings; death in this case was due to shock and a large paraganglioma was found at the autopsy. It is suggested that the

"erythroblastosis foetalis" group of late vomiting cases may be non-fatal examples of obstetric acute yellow atrophy.

A fatal case of true acute yellow atrophy of the liver occurring during pregnancy is next described and the biochemical and pathological findings are given in detail.

The biochemical findings in several non-fatal cases of delayed chloroform poisoning are recorded and the changes during treatment are followed. The rapid improvement which occurs in such cases following the administration of continuous intravenous glucose is emphasized.

A comparison is then made between the biochemical and pathological findings in these three types of liver damage - obstetric acute yellow atrophy, true acute yellow atrophy and delayed chloroform poisoning. The height of the plasma bilirubin is found to be the best index of the severity of the disease. It is shown that gross liver damage does not necessarily result in a high plasma uric acid.

Section VII. includes a short resumé of the experimental and clinical research which has indicated a relationship between surgical or secondary shock and adrenal cortex insufficiency. The biochemical changes in the blood which have been shown to result from adrenalectomy are given. It is noted that there is a close resemblance between the clinical condition of patients suffering from obstetric shock and that of animals with adrenal insufficiency. Changes in blood pressure, pulse rate, haematocrit, whole blood

chloride, plasma sodium and urea are studied in patients about the time of delivery and in the early puerperium. In cases with slight or severe collapse biochemical changes suggestive of a temporary adrenal cortical insufficiency are found to occur; the most important finding is a fall in plasma sodium in shocked cases. The existence of a "pre-shock" state in some patients is suggested. Improvement resulted in a few cases of obstetric shock after treatment with adrenal cortical extracts.

BIBLIOGRAPHY.

1. Anselmino, K.J. and Hoffmann, F., Arch. f. Gyn., 1931, cxlvii, 604.
2. Belt, H.E. and Powell, T.O., Surg. Gyn. and Obst., 1934, lix, 9.
3. Benedict, S.R. J. Biol. Chem., 1922, li, 187 and 1922, liv, 233.
4. Bokelmann, O. and Rother J., Zeitschr. f. Geburtsh u. Gyn., 1925, lxxxix, 72.
5. Bollman, J.L., Mann, F.C. and Magath, T.B., Am. J. Physiol., 1925, lxxii, 629.
6. Burian, R. and Schnur, H., Pflügers Arch., 1910, lxxxvii, 239.
7. Cadden, J.F. and Faris, A.M., Am. J. Obst. and Gyn., 1938, xxxvi, 77.
8. Cadden, J.F. and McLane, C.M., Surg. Gyn. and Obst., 1934, lix, 177.
9. Cadden, J.F. and Stander, H.J., Am. J. Obst. and Gyn., 1939, xxxvii, 37.
10. Caldwell, W.E. and Lye, W.G., ibid., 1921, ii, 17.
11. Carton, P., Quoted by Denecke.
12. Cruickshanks, J.N., Hewitt, J. and Couper, K.L., Med. Research Council Report, 1927, No. 117.
13. Cushing, H., Am. J. Path., 1934, x, 145
14. Denecke, Hinselmann's "Die Eklampsie", Bonn 1924.

15. Dieckmann, W.J., Am. J. Obst. and Gyn., 1933, xxvi, 543.

16. Dieckmann, W.J., ibid., 1935, xxix, 472.

17. Dieckmann, W.J. and Crossen, R.J., Am. J. Obst. and Gyn., 1927, xiv, 3.

18. Dieckmann, W.J. and Wegner, C.R., (a) Arch. Int. Med., 1934, liii, 71.

19. Dieckmann, W.J. and Wegner, C.R., (b) loc. cit., p. 188.

20. Dienst, A., Arch. f. Gyn., 1902, lxv, 369.

21. Dienst, A., ibid., 1927, cxxxii, 288.

22. Duncan, C. and MacLachlan, G.R., Am. J. Obst. and Gyn., 1933, xxv, 157.

23. Dunn, J.S. and Polson, C.J., J. Path. and Bact., 1926, xxix, 337.

24. Eckelt, K., Zeitschr. f. Geburtsh. u. Gyn., 1919, lxxxi, 1.

25. Eisenberg, E.E. and Wallerstein, H., Arch. Path., 1932, xiv, 818.

26. Elden, C.A., Sinclair, F.D. and Rogers, W.C., J. Clin. Invest., 1936, xv, 317.

27. Fikentscher, R., Arch. f. Gyn., 1939, clxviii, 331.

28. Fitzgerald, J.E. and Webster, A., Am. J. Obst. and Gyn., 1938, xxxvi, 460.

29. Folin, O., Bergland, H. and Derick, C., J. Biol. Chem., 1924, lx, 361.

30. Freeman, N.E., Am. J. Physiol., 1933, ciii, 185.

31. Frey, W., Arch. f. Gyn., 1923, cxx, 83.

32. Glassman, O., Surg. Gyn. and Obst., 1938, lxvi, 858.

33. Gremels, H. and Bodo, R., Proc. Roy. Soc. B., 1926, c, 336.

34. Harrop, G.A. and Weinstein, A., J. Exper. Med., 1932, lvii, 305.

35. Harrop, G.A., Weinstein, A., Soffer, L.J. and Trescher, J.H., J. Amer. med. Assoc., 1933, c, 1850.

36. Hartman, F.A., Lewis, L.A. and Toby, C.G., Endocrinology, 1938, xxii, 207.

37. Hegglin, R. and Nabholz, H., Zeitschr. f. Klin. Med., 1938, cxxxiv, 161.

38. Hellmuth, K., Arch, f. Gyn., 1923, cxviii, 18.

39. Herold, L., Arch. f. Gyn., 1939, clxviii, 509.

40. Heynemann, T., from Hinselmann's "Die Eklampsie", Bonn, 1924.

41. Hinselmann, "Die Eklampsie", Bonn, 1924.

42. Hofbauer, J., Zentralbl. f. Gyn., 1918, xlii, 745.

43. Hofbauer, J., Am. J. Obst. and Gyn., 1933, xxvi, 311.

44. Holst, E.J., Acta. Med. Scand., 1938, xciv, 510.

45. Hovarth, M., Quoted by Denecke.

46. Howard, J.E. and Barker, W.H., Bull.John Hopkins' Hosp., 1937, lxi, 371.

47. Kendall, E.C., J. Amer. med. Assoc., 1935, cv, 1486.

48. Killian, J.A. and Sherwin, C.P., Am. J. Obst. and Gyn., 1921, ii, 6.

49. Lever, Guy's Hosp. Rep. Lond., 1843, viii, 493.

50. Liepmann, W., Zentralbl. f. Gyn., 1906, xxx, 693.

51. Loeb, R.F., Science, 1932, lxxvi, 420.

52. Loeb, R.F., Proc. Soc. Exper. Biol. and Med., 1933, xxx, 808.

53. Loeb, R.F., J. Amer. med. Assoc., 1935, civ, 2177.

54. Loeb, R.F., Atchley, D.W., Benedict, E.M. and Leland J., J. Exper. Med. 1933, lvii, 775.

55. Loeb, R.F., Atchley, D.W., Gutman, E.B. and Jillson, R., Proc. Soc. Exper. Biol. and Med., 1933, xxxi, 130.

56. Maclean, M., Arch. Dis. Childh., 1936, xi, 247.

57. Mahnert, A., Arch. f. Gyn., 1921, cxiv, 168.

58. Mann, F.C. and Magath, T.B., Am. J. Physiol., 1921, lv, 286.

59. Meyer, P.J., Arch. f. Gyn., 1887, xxxi, 145.

60. Miller, J.R., Keith, N.M. and Rowntree, L.G., J. Amer. med. Assoc., 1915, lxv, 779.

61. Nasse, H., Arch, f. Gyn., 1876, x, 315.

62. Oberling, C. and Jung, G., Bull. Soc. d'Obst. et Gynec., 1927, xvi, 279.

63. Oberst, F.W. and Plass, E.D., J. Biol. Chem., 1935, cix, 71.

64. Oberst, F.W. and Plass, E.D., Am. J. Obst. and Gyn., 1936, xxxi, 61.

65. Parkins, W.M., Swingle, W.W., Taylor, A.R. and Heys, H.W., Am. J. Physiol., 1938, cxxiii, 668.

66. Payer, A., Arch. f. Gyn., 1904, xxviii, 496.

67. Peckham, C.H., Am. J. Obst. and Gyn., 1929, xvii, 776.

68. Plass E.D., Bull. Johns Hopkins' Hosp., 1924, xxxv, 345.

69. Plass, E.D. and Bogert, L.J., loc. cit., p. 361.

70. Plass, E.D. and Matthews, C.W., Am. J. Obst. and Gyn., 1926, xii, 346.

71. Rabinowitch, I.M. J. Biol. Chem., 1929, lxxxiii, 333.

72. Reed, F.R., Am. J. Surg., 1938, xl, 514.

73. Regnault, Quoted by Payer.

74. Rogoff, J.M. and Stewart, G.N., Am. J. Physiol., 1928, lxxxiv, 649.

75. Rosenau, M.J. and Anderson, J.F., J. Med. Research, Bost., 1908, xix, 37.

76. Rourke, M.D., J. Biol. Chem., 1928, lxxviii, 337.

77. Schoenholz, L., Arch. f. Gyn., 1929, cxxxviii, 596.

78. Schwarz, O. and Dieckmann, W.J., Am. J. Obst. and Gyn., 1929, xviii, 515.

79. Sheehan, H.L., (a) J. Obst. and Gyn. B.E., 1939, xlvi, 218.

80. Sheehan, H.L., (b) loc. cit., p. 685.

81. Sheehan, H.L., J. Obst. and Gyn. B.E., 1940, xlvii, 49.

82. Siedentopf, H.W., Am. J. Obst. and Gyn., 1932, xxiv, 696.

83. Skajaa, K., Acta Obstet. et Gynec. Scand., 1929, viii, 371.

84. Smith, G. van S. and Smith, O.W., Proc. Soc. Exper. Biol. and Med. 1933, xxx, 918.

85. Smith, G. van S. and Smith, O.W., Am. J. Physiol., 1934, cvii, 128.

86. Solomons, B., J. Obst. and Gyn. B.E., 1922, xxix, 416.

87. Solomons, B., Am. J. Obst. and Gyn., 1933, xxv, 172.

88. Stadie, W.C. and van Slyke, D.D., Arch. Int. Med., 1920, xxv, 693.

89. Stander, H.J., Bull. Johns Hopkins' Hosp., 1924, xxxv, 132.

90. Stander, H.J., Ashton, P. and Cadden, J.F., Am. J. Obst. and Gyn., 1932, xxiii, 461.

91. Stander, H.J. and Cadden, J.F., (a) Am. J. Obst. and Gyn., 1934, xxviii, 61.

92. Stander, H.J. and Cadden, J.F., (b) loc. cit., p. 856.

93. Stander, H.J., Duncan, E.E. and Sisson, W.E., Bull. Johns Hopkins' Hosp., 1925, xxxvi, 411.

94. Stander, H.J. and Radelet, A.H., ibid., 1926, xxxviii, 423.

95. Swingle, W.W., Parkins, W.M., Taylor, A.R. and Hays, H.W. (a) Proc. Soc. Exper. Biol. and Med., 1937, xxxvii, 601.

96. Swingle, W.W., Parkins, W.M., Taylor, A.R. and Hays, H.W. (b) loc. cit., p. 675.

97. Swingle, W.W., Parkins, W.M., Taylor, A.R. and Hays, H.W. Am. J. Physiol., 1938, cxxiii, 659.

98. Swingle, W.W., Pfiffner, J.J., Vars, H.M., Bott P.A. and Parkins, W.M., Science, 1933, lxxvii, 58.

99. Thomson, K.J., Hirscheimer, A., Gibson, J.G. and Evans, W.A., Am. J. Obst. and Gyn., 1938, xxxvi, 48.

100. Titus, P., Willetts, E.W. and Lightbody, H.D., Am. J. Obst. and Gyn., 1930, xix, 16.

101.. Townsend, E., B.M.J., 1939, ii, 558.

102. Van Slyke, D.D. and Kugel, V.H., J. Biol. Chem., 1933, cii, 489.

103. Veit, Quoted by Stander in "Williams Obstetrics", 1936, New York.

104. Volhard, F., Hinselmann's "Die Eklampsie", Bonn, 1924.

105. Walthard, B., Arch. f. Gyn., 1922, cxvi, 68.

106. Wells, A.H. and Boman, P.G., J. Amer. med. Assoc., 1937, cix, 1176.

107. Whitehorn, J.C., J. Biol. Chem., 1921, xlv, 449.

108. Williams, J.L., J. Amer. med. Assoc., 1921, lxxvi, 1297.

109. Williams, E.C.P., J. Obst. and Gyn. B.E., 1923, xxx, 189.

110. Wilson, W.C., Rowley, G.D. and Gray, N.A., Lancet, 1936, ccxxx, 1400.

111. Wilson, W.C., MacGregor, A.R. and Stewart, C.P., Brit. J. Surg., 1937, xxv, 826.

112. Wilson, W.C. and Stewart, C.P., Trans. Edin. Medico-Chirurg, 1939, cxviii, 153.

113. Wuth, O., Bull. Johns Hopkins' Hosp., 1926, xxxviii, 389.

114. Young, J., Trans. Edin. Obst. Soc., 1927, xlvii, 61.

115. Zangemeister, W., Zeitschr. f. Geburtsh.u. Gyn., 1903, xlix, 92.

116. Zangemeister, W., Zeitschr. f. Geburtsh. u. Gyn., 1904, xlix, 98.

117. Zangemeister, W., ibid., 1919, lxxxi, 491.

118. Zangemeister, W., Quoted by Heynemann, 1924.

119. Zunst, L., Zentralb. f. Gyn., 1911, xxxv, 1365.

120. Zwemer, R.L., Endocrinology, 1934, xviii, 161.

121. Zwemer, R.L. and Sullivan, R.C., loc. cit., p. 97.

APPENDIX.

A P P E N D I X

The biochemical findings from all the cases quoted throughout the work are given in detail in this section. They are grouped as follows:-

25 Normal cases, Nos.1 to 25.

27 Mild Pre-eclamptic cases, Nos.26 to 52.

29 Severe Pre-eclamptic cases, Nos.53 to 82.

20 Chronic Toxaemic cases, Nos.83 to 103.

42 Eclamptic cases, Nos.104 to 145.

10 Azoman cases, Nos.146 to 155.

16 Hyperemesis cases, Nos.156 to 171.

4 Late hyperemesis cases, Nos.172 to 174.

6 Pyelitis cases, Nos.175 to 180.

4 'Erythroblastosis foetalis' cases, Nos.181 to 184.

1 Case of Obstetric Acute Yellow Atrophy, No.185.

1 Case of True Acute Yellow Atrophy, No.186.

5 Delayed Chloroform Poisoning cases, Nos.187 to 191.

26 Obstetric Shock cases, Nos.192 to 217.

The results are given in tabular form. Plasma uric acid, urea and sodium and whole blood chlorides are expressed in mg. per 100 c.c.; Plasma bilirubin is expressed in units per 100 c.c.

25 NORMAL CASES

CASE 1. Primigravida. Slightly contracted pelvis. Labour 96 hours. Delivered by forceps with no undue haemorrhage.

Date.	4/10/38	12/10/38	13/10/38	14/10/38	11 a.m. 15/10/38	11 a.m. 16/10/38	17/10/38	18/10/38	19/10/38	21/10/38
Haem^{t}	34%	39%	36%	34.5%	35%	36.5%	34%	33%	35%	35%
Uric Acid.	4.2mg%	4.8mg%	6.5mg%	7.7mg%	9.6mg%	10.4mg%	7.9mg%	6.3mg%	5.2mg%	4.2mg%
Urea		20.57%			77.57%	10 47%	637%		217%	

Delivered 12.30p.m.

CASE 2. Primigravida. Slightly contracted pelvis. Labour over 48 hours. Delivered by forceps with no undue haemorrhage.

Date.	31/5/38	8/6/38	17/6/38	28/6/38	29/6/38	30/6/38. 10 a.m.	12.40p.m.	3.10 p.m.	1/7/38	2/7/38
Haem^{t}	31%	30%	31%	36.5%	36%	38%	38.5%	37%	31%	27.5%
Uric Acid.	4.6mg%	5.2mg%	5.2mg%	6.5mg%	7.1mg%	7.6mg%	8.3mg%	8.8mg%	8.8mg%	6.6mg%
Urea.					28.3mg%			38.5mg%	18.5mg%	

Delivered 12.30 p.m.

Date.	4/7/38	7/7/38.
Haem^{t}.	29.5%	30.5%
Uric Acid.	5.5	4.1

CASE 3. Primigravida. Labour 13½ hours. Spontaneous delivery.

Date.	22/10/38	25/10/38. 11 a.m.	3 p.m.	8 p.m.	26/10/38	27/10/38	29/10/38	3/11/38.
Haem^{t}.	33%	35%	39%	36.5%	34%	36%	35%	38%
Uric Acid.	4.2	4.6	5.1	5.2	5.4	5.0	4.6	4.5
Urea	26.3	28		22				
Bili-rubin.	<1	3.5	2.2	<1	<1			

Delivered 12.30 p.m.

CASE 4. Primigravida. Normal. Labour 45 hours. Delivered spontaneously with moderate post-partum haemorrhage.

Date.	23/9/38	4 p.m. 27/9/38	11 a.m. 28/9/38	29/9/38	30/9/38	1/10/38	3/10/38	5/10/38	10/10/38.
Haem^{t}.	29%	28.5%	29%	23%	20%	19.5%	19%	28%	26.5%
Uric Acid.	5.2	5.8	8.5	9.2	7.8	6.4	4.2	4.2	3.8
Urea.		16	32	55	39		21		

Delivered 8.30 p.m.

CASE 5. Primigravida. Normal. Delivered spontaneously. Labour 14 hours. Slight P.P.H.

Date.	30/4/38.	3/5/38	6/5/38. 10 a.m.	2.30 p.m.	3.30 p.m.	9 p.m.	7/5/38	8/5/38	9/5/38	11/5/38.	14/5/38.
Haem^{t}.	36%	38.5%	39.5%	40%	38%	33%	30%	28.5%	30%	30%	32%
Uric Acid.	3.5	2.5	3.3	3.8	4.1	4.5	3.8	3.3	2.8	2.9	3.0

Delivered 3.5 p.m.

CASE 6. Primigravida. Normal. Labour 31 hours. Delivered by Forceps. Moderate P.P.H. and mild infection in the puerperium.

Date.	21/7/38	2/8/38	3/8/38. 10 a.m.	2 p.m.	5 p.m.	4/8/38	5/8/38	6/8/38	8/8/38	12/8/38.
Haem^{t}.	32%	32.5%	35%	33%	32%	27%	24.5%	24%	23.5%	25%
Uric Acid.	4.2	5.4	7.6	8.3	8.7	8.8	5.7	4.8	3.9	3.8

Delivered 1 p.m.

CASE 7. Primigravida. Normal. Labour 16 hours. Spontaneous delivery.

Date.	10 a.m.	12 a.m.	2 p.m.	22/3/38. 4.30 p.m.	5.30 p.m	9.30 p.m.	23/3/38	24/3/38	26/3/38.
Haemt.	34%	34%	35%	39.5%	36%	38%	33%	33.5%	38%
Uric Acid.	4.3			4.7		5.0	4.3	4.4	4.3

Delivered 5 p.m.

CASE 8. Primigravida. Slightly contracted pelvis. Labour 24 hours. Delivered by forceps with no undue haemorrhage.

Date.	6/10/38	10/10/38	15/10/38	11a.m. 16/10/38	17/10/38	18/10/38	19/10/38	21/10/38	24/10/38	31/10/38
Haem^{t}.	34.5%	32%	33%	36%	30%	24%	24%	23.5%	27%	32.5%
Uric Acid.	4.2	5.2	5.1	6.4	7.2	7.2	5.7	4.6	3.9	4.5
Bilirubin.				1.9	1.6	<1				

Delivered 6 p.m.

CASE 9. Primigravida. Normal. Labour 21 hours. Spontaneous delivery.

Date.	8/7/38	10a.m. 15/7/38	12.5 p.m.	4 p.m.	16/7/38	17/7/38	18/7/38	20/7/38	22/7/38.
Haem^{t}.	34.5%	38%	40%	37%	32%	30%	29.5%	30%	31%
Uric Acid.	3.6	4.4		4.9	4.5	3.5	3.6	3.6	3.5
Bilirubin.		1.4	1.4	1.6	1.4	<1			

Delivered 12.15 p.m.

CASE 10. 2nd. Para. Normal. Labour 43 hours. Delivered by forceps with no undue haemorrhage.

Date.	6/10/38	7/10/38. 10 a.m.	7/10/38. 4.30 p.m	8/10/38	9/10/38	10/10/38	11/10/38	12/10/38	14/10/38.
Haem^{t}.	33%	36%	35%	33%	29.5%	28%	28.5%	29%	31%
Uric Acid.	4.0	5.3	5.6	6.4	6.0	5.7	5.6	5.2	4.5
Urea.	15.5								

Delivered 6.30 p.m.

CASE 11. Primigravida. Normal. Labour 20 hours. Spontaneous delivery.

Date.	14/7/38	1/8/38. 10 a.m.	1/8/38. 4.30 p.m.	2/8/38	3/8/38	4/8/38	5/8/38	8/8/38	10/8/38.
Haem^{t}.	29.5%	30%	29%	27%	26%	25%	25%	29%	31%
Uric Acid.	4.0	4.9	4.8	5.2	4.8	4.5	4.2	3.8	3.5

Delivered 7.30 p.m.

CASE 12. 2nd Para. Normal. Labour 4 hours. Spontaneous delivery.

Date.	22/10/38	25/10/38 11 a.m.	25/10/38 8 p.m.	26/10/38	27/10/38	29/10/38	3/11/38.
Haem^{t}.	31%	36%	40%	36%	36.5%	36%	37%
Uric Acid.	3.2	3.5	4.0	4.0	4.2	4.2	3.4
Urea.	18.7	19.3	18.5				

Delivered 6.20 p.m.

CASE 13. Primigravida. Normal. Labour 19 hours. Spontaneous delivery.

Date.	4/5/38. 10.30 a.m	4/5/38. 2.30 p.m.	5/5/38	6/5/38	7/5/38	9/5/38.
Haem^{t}.	34%	32%	37.5%	32%	31.5%	32%
Uric Acid.	4.1	4.0	5.1	5.5	5.1	4.1

Delivered 12 M.N.

CASE 14. Primigravida. Normal. Labour 35 hours. Delivered by forceps with no undue haemorrhage.

Date.	24/5/38. 10 a.m.	24/5/38. 5 p.m.	25/5/38 10 a.m.	25/5/38 12.10 p.m.	25/5/38 4 p.m.	26/5/38	27/5/38	28/5/38	30/5/38.	1/6/38.
Haem^{t}.	31.5%	31%	31%	30.5%	30.5%	25%	25%	24.5%	24.5%	28%
Uric Acid.	4.6		5.0	5.3	5.1	5.2	5.7	5.6	5.4	5.6

Delivered 12 M.D.

CASE 15. Primigravida. Normal. Labour 13 hours. Delivered by forceps with no undue haemorrhage.

Date.	6/10/38.	12/10/38 3 p.m.	13/10/38	14/10/38	15/10/38	17/10/38	20/10/38.
Haem^{t}.	35%	37%	33.5%	32%	31%	32.5%	35%
Uric Acid.	3.9	4.0	4.0	3.9	3.4	3.4	3.5

Delivered 6.45 p.m.

CASE 16. Primigravida. Normal. Labour 61 hours. Spontaneous delivery.

Date.	7/7/38	21/7/38	23/7/38 2.5 a.m.	23/7/38 10 a.m.	24/7/38	25/7/38	27/7/38	29/7/38.
Haem^{t}.	35%	34%	38%	38.5%	33.5%	34%	35%	35.5%
Uric Acid.	3.9	5.2	7.0	7.0	6.1	5.9	4.8	4.9

Delivered 2 a.m.

CASE 17. Primigravida. Normal. Labour 23 hours. Spontaneous delivery.

Date.	23/9/38	3/10/38. 11.30 a.m.	3/10/38. 4.30 p.m.	4/10/38	5/10/38	6/10/38	8/10/38	12/10/38.
Haem^{t}.	36.5%	38%	39.5%	39.5%	38%	34.5%	35%	40.5%
Uric Acid.	3.9	4.6	4.8	4.8	4.8	4.5	4.7	4.6

Delivered 10.30 p.m.

CASE 18. Primigravida. Normal. Labour 9 hours. Spontaneous delivery.

Date.	23/5/38	10 a.m. 25/5/38	26/5/38	27/5/38	28/5/38	30/5/38	1/6/38.
Haem^{t}.	34%	39%	34%	31%	30.5%	31%	32%
Uric Acid.	4.0	5.0	5.2	4.2	3.8	3.5	3.5

Delivered 6 a.m.

CASE 19. Primigravida. Normal. Labour 7 hours, Spontaneous delivery.

Date.	21/7/38.	26/7/38.	27/7/38.	28/7/38.	29/7/38.	1/8/38.	5/8/38.
Haem^{t}.	32%	39%	33%	33%	32%	33%	32%
Uric Acid.	4.5	4.4	4.6	4.1	4.2	3.9	3.5

Delivered 6.50 a.m.

CASE 20. 2nd Para. Contracted pelvis. Labour 23 hours. Delivered by Caesarian section with no undue haemorrhage.

Date.	7/6/38.	17/6/38.	19/6/38.	20/6/38.	21/6/38.	22/6/38.	24/6/38.	27/6/38.	1/7/38.
Haem^{t}.	35%	31.5%	34%	30%	27.5%	26%	23.5%	26%	32.5%
Uric Acid.	3.8	4.4		5.7	6.4	5.0	3.9	2.9	3.0

Delivered 8 p.m.

CASE 21. Primigravida. Normal. Labour 15 hours. Delivered spontaneously.

Date.	8/3/38.	9/3/38.	10/3/38.	14/3/38.
Haem^{t}.	36.5%	29.5%	27.5%	30%
Uric Acid.	2.7	2.9	2.7	3.2

Delivered 4 p.m.

CASE 22. Primigravida. Normal. Labour 8 hours. Delivered spontaneously of a premature child.

Date.	8/3/38.	9/3/38.	10/3/38.	14/3/38.
Haem^{t}.	37%	31%	33%	36%
Uric Acid.	2.7	2.9	2.3	2.8

Delivered 4 a.m.

CASE 23. Primigravida. Normal. Labour 39 hours. Delivered spontaneously of twins.

	25/3/38.		26/3/38.			27/3/38.	
Date.	10 a.m.	2.30 p.m.	10 a.m.	12.30 p.m.	4.30 p.m.	11.30 a.m.	29/3/38.
Haem^{t}.	30.5%	29%	29%	30.5%	33%	30%	26.5%
Uric Acid.	3.9		4.1		5.5	4.7	3.3

Delivered 4.5 p.m.& 4.15 p.m.

CASE 24. Primigravida. Normal. Labour 6 hours. Delivered spontaneously.

Date.	20/12/37.	21/12/37.	23/12/37.	29/12/37.
Haem^{t}.	36%	42.5%	39%	38.5%
Uric Acid.	3.7	3.3	3.02	4.04
	Delivered 4 a.m.			
Hb.	81%	94%	-	87.5
Protein	6.3	6.5	6.4	6.7

CASE 25. 3rd Para. Normal. Labour 20 hours. Delivered spontaneously.

Date.	30/11/37.	3/12/37.	8/12/37.	9/12/37	10/12/37.	13/12/37.	17/12/37.	3/1/38.
Haem^{t}.	37%	36%	35.5%	36%	36%	36%	41%	43%
Protein.		5.8	5.7	6.0	5.7	6.1	6.3	6.5
Uric Acid.		4.8	5.6	5.5	6.5	5.3	5.3	4.6
Hb.		80	79	84	88	82	93.5	85.5

Delivered 2 p.m.

[illegible] Primigravida. S.P. T[illegible] ... and oedema. Labour 36 hours. ... forceps with ... under ...

	2 p.m. 18/3/38	11 a.m. 17/3/38	[illegible]	[illegible]	[illegible]	25/3/38	31/3/38	[illegible]	27/4
[illegible]	76%	88.5%	[illegible]	10.8%	[illegible]	80%	88%	[illegible]	74.4
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

27 MILD PRE-ECLAMPTIC CASES

CASE 26. Primigravida. Slightly raised blood pressure, no albuminuria and no oedema. Labour 72 hours. Delivered by forceps with no undue haemorrhage.

Date.	21/9/38	26/9/38	28/9/38	29/9/38. 10 a.m.	29/9/38. 4.30 p.m.	30/9/38	1/10/38	3/10/38	6/10/38	10/10/38
Haem^{t}.	35%	35%	37.5%	38.5%	38.5%	35%	30.5%	30%	31%	31.5%
Uric Acid.	5.2	5.2	6.7	9.0	9.2	9.6	7.3	3.6	3.5	3.6
Urea				39		41		18.7		
Bilirubin			<1	2.1	1.9	1.6	<1			

Delivered 1.45 a.m.

CASE 27. Primigravida. B.P. $\frac{155}{100}$, slight albuminuria and no oedema. Labour 86 hours. Delivered by forceps with no undue haemorrhage.

Date.	2 p.m. 16/3/39	11 a.m. 17/3/39	18/3/39. 4 p.m.	18/3/39. 11 a.m.	18/3/39. 7 p.m.	20/3/39	21/3/39	23/3/39	27/3/39.
Haem^{t}.	36%	38.5%	39%	38.5%	40%	30%	32%	35%	36.5%
Uric Acid.	5.4	5.6	6.0	7.0	7.9	6.6	4.4	3.2	3.2
Urea	26	21		32	43	40	28	25	

Delivered 9.30 p.m. 19/3/39.

CASE 28. Primigravida. B.P. $\frac{145}{100}$, no albuminuria and slight oedema. Labour 67 hours. Delivered by forceps with moderate P.P.H.

Date.	23/5/38	28/6/38	1/7/38	2/7/38	3/7/38 11 a.m.	4/7/38	5/7/38	6/7/38	8/7/38	11/7/38.
Haem^{t}.	36%	35.5%	38.5%	37%	38.5%	30%	24%	23%	23%	26%
Uric Acid.	3.0	5.3	6.3	6.0	8.0	9.2	6.6	4.9	4.0	3.4

Delivered 2.30 p.m.

CASE 29. Primigravida. B.P. $\frac{140}{100}$, slight albuminuria and marked oedema. Labour 37 hours. Delivered spontaneously.

Date.	3/3/39	6/3/39	8/3/39	9/3/39 10a.m.	2 p.m.	10/3/39	11/3/39	12/3/39	14/3/39	16/3/39.
Haem^{t}.	36%	36%	39%	40.5%	41%	39.5%	34%	34%	34%	39%
Uric Acid.	3.1	3.1	4.0	5.7	6.2	5.4	4.7	3.3	4.5	4.2
Urea.	14	15	19	19	20	29	26	29		29

Delivered 10.5 a.m.

CASE 30. Primigravida. B.P. $\frac{170}{100}$, No Albuminuria and moderate oedema. Labour 41 hours. Delivered spontaneously.

Date.	3p.m. 1/3/39	2/3/39. 10 a.m.	2/3/39. 5 p.m.	3/3/39.	4/3/39	6/3/39	8/3/39	10/3/39.
Haem^{t}.	37%	36.5%	36%	37%	32%	31%	35%	37%
Uric Acid.	3.8	4.6	4.9	5.5	5.4	4.1	5.1	4.6
Urea.	25	26	28	31	32	23	23	29

Delivered 3.30 a.m.

CASE 31. Primigravida. B.P. $\frac{160}{110}$, Slight albuminuria and no oedema. Labour 23 hours but very mild. Delivered spontaneously of macerated foetus.

Date.	1/11/38	8/11/38	16/11/38	17/11/38	18/11/38	19/11/38	21/11/38
Haem^{t}.	38.5%	39%	40%	39%	37.5%	34%	35%
Uric Acid.	3.1	3.0	2.6	3.3	3.3	3.0	2.3
Urea.	7.5						

Delivered 4 a.m.

CASE 32. Primigravida. B.P. $\frac{130}{90}$. Moderate albuminuria and slight oedema. Labour 24 hours. Delivered spontaneously.

Date.	28/2/39	7/3/39	11 a.m. 8/3/39	9/3/39	10/3/39	11/3/39	14/3/39
Haem^{t}.	34%	36%	35%	31.5%	29%	31%	35%
Uric Acid.	3.3	4.2	4.6	5.1	4.7	4.1	4.5
Urea	20	14	19	17	19		

Delivered 5 a.m.

CASE 33. Primigravida. B.P. $\frac{150}{110}$, no albuminuria and no oedema. Labour 65 hours. Delivered by forceps with no undue haemorrhage.

Date.	7/3/39	8/3/39	9/3/39 10 a.m.	9/3/39 2.15 p.m	10/3/39	11/3/39	12/3/39	14/3/39	16/3/39
Haem^{t}.	36.5%	37%	39%	42%	35%	32%	33%	32.5%	33%
Uric Acid.	4.3	5.3	6.7	7.8	5.8	4.6	3.7	3.5	2.6
Urea.	19	26	33	42	47	27	24		

Delivered 11.35 a.m.

CASE 34. Primigravida. B.P. $\frac{150}{100}$. No albuminuria, and slight oedema. Labour 9 hours. Delivered spontaneously.

Date.	9/11/38	15/11/38	10 a.m. 16/11/38	3.30 p.m. 16/11/38	17/11/38	18/11/38	19/11/38	24/11/38.
Haem^{t}.	28%	32%	33%	34%	31.5%	30.5%	32%	36%
Uric Acid.	4.9	4.5	5.1	5.1	4.6	4.0	3.7	3.5
Urea.	23.6		16.5		14			

Delivered 1.10 p.m.

CASE 35. Primigravida. B.P. $\frac{150}{100}$, Slight albuminuria and slight oedema. Labour 34 hours. Delivered by forceps with no undue haemorrhage.

Date.	9/1/39	19/1/39	20/1/39	21/1/39	22/1/39	23/1/39	24/1/39	26/1/39	31/1/39
Haem^{t}.	38%	38%	40%	42%	37%	28%	24%	26.5%	29.5%
Uric Acid.	3.8	3.5	4.6	4.6	5.7	6.0	4.6	3.9	3.7
Urea		23	34	33	48	63	38	29	39

Delivered 1.30 a.m.

CASE 36. Primigravida. B.P. $\frac{145}{100}$. No albuminuria and no oedema. Labour 24 hours. Delivered of twins - one by forceps and one spontaneously. Both children still-born. No undue haemorrhage.

Date.	2/3/39	22/3/39	23/3/39 10 a.m.	23/3/39 2.15 p.m.	23/3/39 4.30 p.m.	24/3/39	25/3/39	27/3/39	29/3/39	2/4/39
Haem^{t}.	35%	34%	38.5%	39%	35.5%	32%	27.5%	26%	28%	27.5%
Uric Acid.	3.0	3.4	4.4	4.4	4.4	4.8	4.2	3.2	2.6	2.7
Urea.	19	26	37	41		56	44	20		22

Delivered 2.45 p.m. & 4.10 p.m.

CASE 37. Primigravida. B.P. $\frac{150}{100}$. Slight albuminuria and oedema. Labour 46 hours. Delivered by forcepswith no undue haemorrhage.

Date.	21/3/39	22/3/39. 10 a.m.	22/3/39. 2 p.m.	22/3/39. 5 p.m.	23/3/39	24/3/39	25/3/39	27/3/39	30/3/39.
Haem^{t}.	39%	37%	39%	37.5%	33%	31.5%	30.5%	30%	34%
Uric Acid.	4.6	7.2	7.6	7.6	6.1	4.3	3.1	3.0	2.8
Urea	33	67	76		6.1	50	44	28	44

Delivered 12 M.D.

CASE 38. Primigravida. B.P. $\frac{145}{100}$ Slight albuminuria and no oedema. Labour 24 hours. Delivered of twins, internal version performed and children delivered by forceps. There was a moderate amount of haemorrhage at delivery and the patient collapsed after delivery. Intravenous glucose and blood transfusion given.

Date.	20/3/39	27/3/39. 10 a.m.	12.15 p.m.	2.15 p.m.	4.15 p.m.	28/3/39	29/3/39	30/3/39	4/4/39.
Haem^{t}.	37.5%	43.5%	40%	37%	36%	29%	27%	27%	26.5%
Uric Acid.	5.1	7.6	8.8	8.5	9.6	8.9	6.6	4.9	3.6
Urea	16	34		41	47	45	35	28	38

Delivered 12 M.D. Collapsed.

CASE 39. Primigravida. B.P. $\frac{130}{90}$ Slight albuminuria and no oedema. Labour 18 hours. Delivered spontaneously with a moderate P.P.H.

Date	14/3/38. 11.30 a.m.	4.30 p.m.	15/3/38	16/3/38	17/3/38	21/3/38
Haem^{t}.	36%	36%	29%	26%	26%	27%
Uric Acid.	5.9	6.0	6.0	5.0	5.2	4.5

Delivered 11.30 p.m.

CASE 40. Primigravida. B.P. $\frac{160}{100}$ Slight albuminuria and oedema. Labour 8 hours. Delivered spontaneously. Retained placenta and severe P.P.H. Blood transfusion given.

Date.	7/3/38	8/3/38	9/3/38	10/3/38	12/3/38	15/3/38	19/3/38.
Haem^{t}.	33%	35%	26.5%	24%	22%	23%	30%
Uric Acid.	5.1	5.1	6.5	6.3	4.5	5.3	5.4

Delivered 4 p.m.

CASE 41. Primigravida. B.P. $\frac{145}{100}$. No albuminuria and slight oedema. Labour 28 hours but very severe. Delivered by forceps with no undue haemorrhage.

Date.	7/6/38	11/6/38	12/6/38	13/6/38	14/6/38	16/6/38	20/6/38
Haem^{t}.	34%	38%	34%	26.5%	26%	28%	30%
Uric Acid.	3.6	4.9	7.2	5.4	4.0	3.2	3.2

Delivered 4 a.m.

CASE 42. Primigravida. B.P. $\frac{148}{80}$. No albuminuria and no oedema. Labour 17 hours. Delivered by forceps with no undue haemorrhage.

Date.	1/3/39	3/3/39. 10 a.m.	3/3/39. 12.15 p.m.	4/3/39	6/3/39	8/3/39	10/3/39.
Haem^{t}.	36%	38%	39%	34%	33.5%	31%	33%
Uric Acid.	4.2	5.1	5.2	5.3	4.5	3.2	3.3
Urea.	32	39	47	49	32	27	22

Delivered 10.50 a.m.

CASE 43. 2nd Para. B.P. $\frac{145}{100}$. Slight albuminuria and no oedema. Labour 2 hours. Spontaneous delivery.

Date.	21/6/38	22/6/38	23/6/38	24/6/38	25/6/38	27/6/38	21/7/38
Haem^{t}.	36%	35%	37%	35%	32%	34%	40%
Uric Acid.	4.0	4.2	4.6	4.4	4.2	2.8	2.3

Delivered 6.45 p.m.

CASE 44. Primigravida. B.P. $\frac{150}{110}$. Slight albuminuria and no oedema. Labour 23 hours. Spontaneous delivery.

Date.	19/5/38 11 a.m.	19/5/38 3.10 p.m.	19/5/38 5.30 p.m.	20/5/38	21/5/38	23/5/38	25/5/38.
Haem^{t}.	35%	36%	34.5%	33%	32%	30.5%	33%
Uric Acid.	5.2	5.4	5.8	5.2	5.2	3.9	3.4

Delivered 3.30 p.m.

CASE 45. Primigravida. B.P. $\frac{145}{100}$. Slight albuminuria and oedema. Labour 27 hours. Premature child. Spontaneous delivery.

Date.	17/5/38 3 p.m.	17/5/38 9 p.m.	18/5/38	19/5/38	20/5/38	21/5/38	24/5/38	27/5/38
Haem^{t}.	39.5%	41%	36.5%	30.5%	28.5%	29.5%	32%	33%
Uric Acid.	4.3	4.2	4.7	4.5	3.9	3.7	3.2	3.2
Urea	18		22			24		

Delivered 1.25 a.m.

CASE 46. Primigravida. B.P. $\frac{130}{80}$ Slight albuminuria and oedema. Labour 17 hours. Spontaneous delivery with slight P.P.H. Mild infection in puerperium.

Date.	20/4/38	21/11/38 10 a.m.	2 p.m.	4.30 p.m.	22/4/38	23/4/38	25/4/38	28/4/38	2/5/38.
Haem^{t}.	29.5%	34%	31%	30.5%	21.5%	16.5%	16.5%	18.5%	21%
Uric Acid.	5.4	5.8	6.8	6.9	6.8	6.0	4.8	5.1	3.5
Bilirubin.	1.5	2.6		2	1.6				

Delivered 1.10 p.m.

CASE 47. Primigravida. B.P. $\frac{145}{100}$. Slight albuminuria and oedema. Labour 20 hours. Spontaneous delivery.

Date.	17/1/38	21/1/38	9/2/38	10/2/38	11/2/38	14/2/38	18/2/38
Haem^{t}.	34.5%	35.5%	37%	34%	32.5%	29%	32%
Hb.	71.5	75	78	71	64	59.5	
Protein	5.3	5.8	6.0				
Uric Acid.	4.6	5.0	7.7	6.8	5.4	4.8	3.7

Delivered 2 p.m.

CASE 48. Primigravida. B.P. $\frac{160}{112}$. Slight albuminuria and oedema. Labour 23 hours. Spontaneous delivery.

Date.	16/2/38	17/2/38	18/2/38	21/2/38	23/2/38	25/2/38.
Haem^{t}.	33%	36%	33.5%	33.5%	37%	35.5%
Uric Acid.	4.7	5.3	4.7	4.0	4.3	4.50
Urea.		19.4				

Delivered 2 a.m.

CASE 49. Primigravida. B.P. $\frac{140}{90}$. No albuminuria and slight oedema. Labour 14 hours. Spontaneous delivery.

	23/4/38.					
Date.	10 a.m.	2.50 p.m.	4.50 p.m.	24/4/38	25/4/38	28/4/38.
Haem^{t}.	27%	28%	27%	26%	23%	24.5%
Uric Acid.	4.2	5.2	4.5	4.2	3.6	3.4

Delivered 2.20 p.m.

CASE 50. Primigravida. B.P. $\frac{145}{100}$. Slight albuminuria and no oedema. Labour 12 hours. Spontaneous delivery.

Date.	1/3/38	5p.m. 2/3/38	3/3/38	7/3/38	9/3/38.
Haem^{t}.	31%	35.5%	29%	26.5%	27%
Uric Acid.	4.3	4.2	4.1	4.5	3.7

Delivered 2 p.m.

CASE 51. 2nd Para. B.P. $\frac{140}{112}$. Slight albuminuria and oedema. Labour 18 hours. Delivered by forceps with moderate P.P.H.

Date.	24/1/38	25/1/38	26/1/38	28/1/38	2/2/38
Haem^{t}.	29.5%	28%	25%	25%	28.5%
Hb.	52.5	48	43.5	43	50
Protein.	7.5	7.4	7.1	6.9	6.7
Uric Acid.	4.6	5.96	5.3	4.9	4.1

Delivered 4 p.m.

CASE 52. Primigravida. B.P. $\frac{138}{90}$, Slight albuminuria and very slight oedema. Labour 10 hours. Spontaneous delivery.

Date.	10 a.m. 22/2/38	2 p.m. 22/2/38	23/2/38	24/2/38	26/2/38.
Haem^{t}.	37%	41%	34%	34%	32%
Uric Acid.	5.3	5.0	5.6	4.2	4.1

Delivered 12.30 p.m.

with no undue haemorrhage.

11/7/38	15/7/38	10a.m. 15/7/38	4p.m. 15/7/38	15/7/38. 10 a.m.	4.30 p.m.	5 p.m.	17/7/38	18/7/38	19/7/38	20/
52%	37%	38%	41%	41.5%	37%	33.5%	34.5%	30.5%	28%	30
d. 6.8	7.1	8.1	8.0	9.1	9.2	8.3	10.4	9.1	7.5	8.
87	80	90		75				80		

Delivered 12.30 p.m.

25/7/38	26/7/38
30.5%	31.5%
4.8	3.7

29 SEVERE PRE-ECLAMPTIC CASES

CASE 53. Primigravida. B.P. $\frac{165}{112}$. Slight albuminuria and marked oedema. Labour 96 hours. Delivered by forceps with slight P.P.H. Infection in puerperium due to haemolytic Streptococci.

Date.	10/6/38	12/6/38	13/6/38	14/6/38. 10 a.m.	14/6/38. 8 p.m.	15/6/38. 10 a.m.	15/6/38. 12.10 p.m.	15/6/38. 3 p.m.	16/6/38	17/6/38	18/6/38	20/6/38
Haem^{t}.	38%	36.5%	35%	36%	37%	37%	36%	35%	30%	26%	26%	24%
Uric Acid.	3.6	4.8	6.0	7.7	9.1	9.6	9.8	10	8.4	5.2	4.0	3.5
Urea.			30				88		94	57		41
Bilirubin.		.5	2.4	2.8	2.8	3.8		4	3.6	2.4	1	3·6

Delivered 12.5 p.m.

CASE 54. Primigravida. B.P. $\frac{200}{110}$. Moderate albuminuria and oedema. Labour 47 hours. Delivered by forceps with no undue haemorrhage.

Date.	11/7/38	13/7/38	15/7/38 10a.m.	15/7/38 4p.m.	16/7/38. 10 a.m.	16/7/38. 1.30 p.m.	16/7/38. 5 p.m.	17/7/38	18/7/38	19/7/38	20/7/38	22/7/38
Haem^{t}.	33%	37%	38%	41%	41.5%	37%	39.5%	34.5%	30.5%	29%	30.5%	32%
Uric Acid.	6.8	7.1	8.1	8.0	9.1	9.2	9.3	10.4	9.1	7.3	5.8	4.6
Urea	27	30	50		73				60			36.5

Delivered 12.50 p.m.

Date.	25/7/38	28/7/38
Haem^{t}.	30.5%	31.5%
Uric Acid	4.3	3.7
Urea.		

CASE 55. Primigravida. B.P. $\frac{180}{100}$ Gross albuminuria and moderate oedema. Labour 48 hours. spontaneous delivery.

	5/5/38			6/5/38							
Date.	10 a.m.	4 p.m.	10 p.m.	6 a.m.	10 a.m.	7/5/38	8/5/38	9/5/38	11/5/38	13/5/38	18/5/38
Haem^{t}.	32%	40%	41%	44%	43%	34.5%	32%	30%	32.5%	35%	35%
Uric Acid.	5.4	5.0	6.3	7.4	7.8	6.4	4.8	3.9	3.9	3.9	4.0
Urea.	15.8			34							

Delivered 4.45 a.m.

CASE 56. Primigravida. B.P. $\frac{150}{110}$. Moderate albuminuria and gross oedema. Labour 6 hours. Delivered by forceps with slight P.P.H. Mild infection in puerperium. Blood transfusion given for anaemia on fourth day post-partum.

		18/5/38.								
Date.	17/5/38	10 a.m.	12.5 p.m.	4 p.m.	19/5/38	20/5/38	21/5/38	23/5/38	30/5/38	10/6/38
Haem^{t}.	24.5%	27%	26.5%	22%	17.5%	15%	15%	19.5%	22%	19%
Uric Acid.	5.2	6.4	6.7	6.9	6.4	6.0	4.7	3.7	3.3	
Urea.	13.5%				22.3		25			

Delivered 12 M.D.

Transfusion 300 cc blood.

CASE 57. Primigravida. B.P. $\frac{160}{110}$. Moderate albuminuria and slight oedema. Labour 27 hours. Delivered by forceps with no undue haemorrhage.

Date.	25/10/38	28/10/38 10 a.m.	3 p.m.	29/10/38	30/10/38	31/10/38	1/11/38	5/11/38	8/11/38
Haem^{t}.	34%	36%	37%	33%	31.5%	30%	33%	35%	35%
Uric Acid.	6.2	8.0	7.9	8.2	6.8	5.5	3.9	3.5	3.3
Urea.	22.8	31		36.5		16	17		

Delivered 4.20 p.m.

CASE 58. Primigravida. B.P. $\frac{184}{70}$ Moderate albuminuria and no oedema. Labour $\frac{3}{4}$ hour. Spontaneous delivery.

Date.	16/1/39	19/1/39	25/1/39	1/2/39	6/2/39	7/2/39	8/2/39	9/2/39	10/2/39	11/2/39	13/2/39	15/2/39	17/2/39
Haem^{t}.	38%	39%	38%	39%	40.5%	45%	40%	38%	37.5%	39%	42%	41.5%	44%
Uric acid.	7.1	8.0	6.6	6.2	7.2	7.4	7.4	7.8	7.8	7.6	7.0	6.2	5.4
Urea.	38	30	26	18	18	38	38.6	39	38	29	31	32	28.5

Delivered 4.15 a.m.

Date.	20/2/39.
Haem^{t}.	44%
Uric Acid	4.6

CASE 59. 2nd Para. B.P. $\frac{150}{110}$. Gross albuminuria and no oedema. Labour 4 hours. Delivered spontaneously of twins.

Date.	30/9/38	8/10/38	24/10/38 2.15 p.m.	25/10/38	26/10/38	27/10/38	28/10/38	2/11/38
Haem^{t}.	34%	32%	36%	38%	35%	31%	33%	38%
Uric Acid.	6.0	5.6	5.7	6.8	7.4	6.9	6.6	5.0
Urea.			39	34	29			31

Delivered 6.30 a.m.

CASE 60. Primigravida. B.P. $\frac{120}{70}$. Gross albuminuria and oedema. Labour 10 hours. Delivered spontaneously of a premature still-born child.

Date.	30/1/39	31/1/39	1/2/39	2/2/39	6/2/39	7/2/39	8/2/39	9/2/39	11/2/39	15/2/39	22/3/39
Haem^{t}.	28.5%	29%	30.5%	31.5%	27.5%	31%	31.5%	30.5%	29%	33.5%	33%
Uric Acid.	6.6	6.7	6.6	6.6	5.3	4.5	4.3	4.1	2.9	3.3	3.5
Urea.	46	36	39		18	16					19.3

Delivered 7 a.m.

CASE 61. Primigravida. B.P. $\frac{170}{120}$. Marked albuminuria and oedema. Labour 4 hours. Delivered spontaneously.

Date.	13/6/38	14/6/38	15/6/38	16/6/38	17/6/38	18/6/38	19/6/38. 12.5 p.m.	19/6/38. 11 a.m.	20/6/38	21/6/38	22/6/38	24/6/38	28/6/38
Haem^{t}.	33%	35%	37%	35%	36%	38%	31%	32%	31%	30%	28.5%	31%	35%
Uric Acid.	6.4	6.0	6.0	6.8	7.6	6.1	6.0	6.0	5.4	4.6	3.9	3.9	3.9
Urea.	29		29.3		46			29.5					

Delivered 10.45 p.m.

CASE 62. Primigravida. B.P. $\frac{150}{110}$. Moderate albuminuria and slight oedema. Labour 12 hours. Spontaneous delivery.

Date.	16/3/39	18/3/39	20/3/39	21/3/39	22/3/39	23/3/39	24/3/39	25/3/39	27/3/39	29/3/39.
Haem^{t}.	35%	33%	32.5%	34.5%	37%	34%	33%	32.5%	36%	35%
Uric Acid.	6.1	6.8	5.7	5.8	6.4	6.4	5.9	4.6	5.7	5.6
Urea.	35	28	17	26	35	37	33	29	30	

Delivered 4 a.m.

CASE 63. Primigravida. B.P. $\frac{170}{120}$. Marked albuminuria and gross oedema. Labour 21 hours. Delivered by forceps with no undue haemorrhage. Albuminuria and oedema increased for 2 days after delivery.

Date.	22/6/38	23/6/38 10 a.m.	23/6/38 11 a.m.	23/6/38 3 p.m.	24/6/38	25/6/38	29/6/38	18/7/38
Haem^{t}.	30.5%	34%	35%	37%	30.5%	31.5%	34%	37%
Uric Acid.	5.1	6.5	6.9	6.0	6.8	7.3	4.8	4.3
Urea.	21		47					
Bili-rubin.	.5		1.5	2.8		1.8		

Delivered 10.40 a.m.

CASE 64. Primigravida. B.P. $\frac{150}{105}$. Gross albuminuria and slight oedema. Labour 48 hours and very severe. Delivered by forceps with no undue haemorrhage. Mild infection post-partum.

Date.	4/10/38	7/10/38	10/10/38	12/10/38	13/10/38. 11 a.m.	13/10/38. 4 p.m.	10a.m. 14/10/38	15/10/38	16/10/38	17/10/38	18/10/38
Haem^{t}.	26%	24%	25%	25%	27%	26%	27%	20%	17.5%	16.5%	17.5%
Uric Acid.	5.7	5.9	6.4	6.0	8.0	8.5	10.8	12.4	10.4	6.6	5.0
Urea.	24		22				89	115	106	57	40

Delivered 10.30 p.m.

Date.	Haem^{t}.	Uric Acid	Urea.
21/10/38	18%	4.2	31

CASE 65. Primigravida. B.P. $\frac{140}{110}$. Marked albuminuria and no oedema. Labour 86 hours - severe vomiting during labour. Delivered by forceps with moderate P.P.H.

Date.	28/12/38	6/1/39	7/1/39. 1.30 a.m.	10 a.m.	8/1/39	9/1/39	10/1/39	12/1/39	19/1/39.
Haem^{t}.	32%	34%		28%	22%	19%	20%	18%	20.5%
Uric Acid.	6.0	10.7	12.1	11.0	11.7	6.3	4.3	3.7	1.8
Urea.	30	81	77	94	104	67	51	36	32
Urine			14 oz.		5oz.	3oz.	19oz	22oz 13 oz.	

Delivered 1 a.m.

CASE 66. Primigravida. B.P. $\frac{160}{100}$. Slight albuminuria and marked oedema. Labour 32 hours. Spontaneous delivery. Mild infection in the puerperium.

Date.	23/4/38 10 a.m.	3 p.m.	4.45 p.m.	24/4/38	25/4/38	26/4/38	28/4/38	31/4/38	4/5/38.
Haem^{t}.	32%	32%	29%	23%	20%	17.5%	17.5%	19%	19%
Uric Acid.	7.2	8.3	6.1	6.9	7.2	6.6	4.2	4.6	5.2
Urea.									
Bilirubin	3.4	4.4	2	1.4					

Delivered 4.20 p.m.

CASE 67. 2nd Para. B.P. $\frac{150}{120}$. Slight albuminuria and marked oedema. Labour 4 hours. Spontaneous delivery.

Date.	3/2/39	4/2/39	6/2/39	7/2/39	8/2/39	10/2/39	13/2/39.
Haem^{t}.	38%	42%	42%	35%	34.5%	35%	40.5%
Uric Acid.	5.0	5.0	5.8	5.9	5.2	4.1	3.4
Urea.	34	36	33		29		23
Urine		8oz.	4oz.				

Delivered 5/2/39 at 12 M/N.

CASE 68. Primigravida. B.P. $\frac{165}{100}$. Moderate albuminuria and oedema. Labour 14 hours. Spontaneous delivery.

Date.	8/11/38	10/11/38	10.15a.m. 11/11/38	3.30p.m. 11/11/38	12/11/38	13/11/38	14/11/38	16/11/38	19/11/38
Haem^{t}.	36%	Pains began 10 p.m.	39%	39%	32%	29%	30.5%	31.5%	31.5%
Uric Acid.	5.0		6.0	6.3	5.7	5.5	4.8	3.7	3.5
Urea.	27		32		45	28	31	27.3	

Delivered 11.30 a.m.

CASE 69. Primigravida. B.P. $\frac{164}{120}$. Marked albuminuria and slight oedema. Labour 16 hours. Spontaneous delivery.

Date.	24/2/39	27/2/39	28/2/39 10 a.m.	1/3/39	2/3/39	3/3/39	6/3/39.
Haem^{t}.	39%	43%	39%	35.5%	33.5%	33.5%	36%
Uric Acid.	4.0	5.7	6.2	5.9	4.7	3.4	3.8
Urea.	20	32	29	24	32	28	27

Delivered 8.30 a.m.

CASE 70. Primigravida. B.P. $\frac{200}{120}$. Slight albuminuria and no oedema. Labour 38 hours. Spontaneous delivery.

Date.	26/7/38	28/7/38	9/8/38	10/8/38. 10 a.m.	12.30 p.m.	3.30 p.m.	11/8/38	12/8/38	13/8/38.
Haem^{t}.	30%	33%	36%	38%	36%	35%	32.5%	31%	31.5%
Uric Acid.	5.2	4.6	6.0	6.8	6.8	7.3	5.6	5.0	3.5
Bilirubin			1.1	2			<1		
Urea.			36						

Delivered 12.35 p.m.

CASE 71. Primigravida. B.P. $\frac{168}{98}$. Slight albuminuria and no oedema. Labour about 86 hours. Delivered of macerated foetus after craniotomy. Moderate P.P.H.

Date.	21/3/39	22/3/39. 10 a.m.	22/3/39. 4.50 p.m.	23/3/39	24/3/39	25/3/39	28/3/39.
Haem^{t}.	36%	36%	33%	24%	24%	25%	27%
Uric Acid.	5.4	6.6	7.2	9.6	6.6	4.6	4.2
Urea.	15	23	30	64	40	30	26

Delivered 12 M.D.

CASE 72. Primigravida. B.P. $\frac{152}{100}$. Moderate albuminuria and no oedema. Labour 23 hours. Spontaneous delivery of a still-born child.

Date.	11/1/39.	13/1/39.	25/1/39	7/2/39	10/2/39	13/2/39	14/2/39	15/2/39	17/2/39.
Haem^{t}.	43%	43%	43%	42%	42%	39%	41%	38%	40%
Uric Acid.	6.8	6.3	6.9	6.6		7.8	6.6	6.1	6.2
Urea.	32	27	29	33		28	21	22	28

Delivered 10.30 p.m.

CASE 73. Primigravida. B.P. $\frac{170}{120}$. Moderate albuminuria and oedema. Labour 24 hours. Spontaneous delivery.

Date.	30/5/38	6/6/38	21/6/38	23/6/38	24/6/38	25/6/38	10.45a.m. 26/6/38	2 p.m. 26/6/38	27/6/38	28/6/38
Haem^{t}.	32.5%	32%	35.5%	36%	35.5%	37%	37%	35%	29%	28.5%
Uric Acid.	4.4	4.8	4.6	4.6	4.7	4.6	4.7	4.9	5.0	5.6

Delivered 26/6/38 10.10 a.m.

Date.	29/6/38	1/7/38	4/7/38
Haem^{t}.	30%	28%	33%
Uric Acid.	4.8	4.8	4.6

CASE 74. Primigravida. B.P. $\frac{180}{120}$. Marked albuminuria and oedema. Labour 38 hours. Delivered Spontaneously.

Date.	1/11/38	10/11/38	11/11/38	11 a.m. 13/11/38	14/11/38	15/11/38 10 a.m.	4 p.m.	16/11/38	17/11/38	18/11/38
Haem^{t}.	35%	35%	33.5%	38.5%	34%	38%	39.5%	34.5%	31%	29%
Uric Acid.	3.5	4.7	4.8	5.4	6.2	6.6	6.5	6.7	4.4	4.2
Urea.	13.7		30	29		38		31.6	14.5	

Worse. Delivered 5 p.m.

Date.	19/11/38	23/11/38
Haem^{t}.	29%	30.5%
Uric Acid.		2.3

CASE 75. Primigravida B.P. $\frac{150}{100}$. Moderate albuminuria and oedema. Labour 23 hours. Spontaneous delivery.

Date.	3/2/39	9/2/39. 10.35 a.m.	9/2/39. 3.30 p.m.	10/2/39	11/2/39	13/2/39	16/2/39	21/2/39.
Haem^{t}.	33%	36%	37%	32%	28%	31%	35%	35%
Uric Acid.	5.8	6.4	7.0	6.1	5.3	3.7	3.8	3.9
Urea.	17	30	36	31	15		29	32

Delivered 10.30 a.m.

CASE 76. Primigravida. B.P. $\frac{160}{120}$. Marked albuminuria and oedema. Labour 3 hours. Spontaneous delivery of a complete miscarriage.

Date.	29/9/38	30/9/38	1/10/38	3/10/38	4/10/38	5/10/38	7/10/38	10/10/38	14/10/38
Haem^{t}.	39.5%	40%	39%	36%	34.5%	34%	32%	33%	32%
Uric Acid.	4.6	4.5	5.1	5.2	4.0	3.6	3.3	2.8	3.0

Delivered 3/10/38 at 7.30 a.m.

CASE 77. Primigravida. B.P. $\frac{190}{140}$. Slight albuminuria and no oedema. Labour 1 hour. Spontaneous delivery.

Date.	31/5/38	1/6/38	2/6/38	4/6/38	6/6/38	8/6/38	11/6/38.
Haem^{t}.	40%	44%	39%	32%	34%	37%	39%
Uric Acid.	6.4	6.2	6.1	5.6	4.3	4.0	4.0

Delivered 2.30 a.m.

CASE 78. 2nd Para. B.P. $\frac{236}{156}$. Moderate albuminuria and no oedema. Labour 6 hours. Spontaneous delivery.

Date.	16/2/38	22/2/38	25/2/38	28/2/38	1/3/38	2/3/38	4/3/38.
Haem^{t}.	36.5%	39%	37%	40%	37.5%	34.5%	33%
Uric Acid.	5.3	4.4	4.7	5.4	4.9	4.6	3.8

Delivered 28/2/38 12.30 a.m.

CASE 79. 2nd Para. B.P. $\frac{200}{110}$. Moderate Albuminuria and oedema. Labour 12 hours. Spontaneous delivery.

Date.	10/1/38	12/1/38	13/1/38	14/1/38	15/1/38	17/1/38	19/1/38.
Haem^{t}.	39%	41.5%	30%	23%	24%	23.5%	24%
Hb.	76	83	56	45.5	47	46	47.5
Protein	6.07	6.28	4.6	4.6	5.0	4.9	5.8
Uric Acid.	6.9	6.6	8.5	7.0	5.4	3.7	3.2
Urea.	60	27	17.4	18.2	34		

Delivered 1 a.m.

CASE 80. Primigravida. B.P. $\frac{180}{120}$. Moderate albuminuria and slight oedema. Labour 14 hours. Spontaneous delivery with a moderate P.P.H.

Date.	4/3/38	9/3/38	11/3/38	12/3/38	14/3/38	15/3/38	16/3/38	18/3/38	21/3/38.
Haem^{t}.	31.5%	31%	31.5%	33.5%	23.5%	22%	23%	23%	25%
Uric Acid.	7.4	9.7	8.3	9.7	10	8.9	8.3	7.7	6.3
Urea.			53	55	46	37	33		
Protein.							6		

Delivered 13/3/38 6 p.m.

CASE 81. Primigravida. B.P. $\frac{170}{110}$ Gross albuminuria and oedema. Labour 48 hours. Delivered by forceps with moderate P.P.H. Mild infection in puerperium.

Date.	4/12/37 10a.m.	4/12/37 6 p.m.	5/12/37	6/12/37	7/12/37	8/12/37	9/12/37	11/12/37	13/12/37	15/12/37	21/12/37	3/1/38
Haem^{t}.	20.7%	20%	24%	24.5%	20.5%	19%	19%	20%	18.5%	18.5%	19.5%	27%
Protein	6.6	6.6	7.0	7.0	5.8	5.95	6.0	5.3	5.0	6.25	6.1	6.56
Uric Acid.	6.4	6.2	6.5	9.4	9.9	8.1	6.5	6.4	4	3.8	3.1	4.0
B.Urea.			37.2	51.4	67.2	51.2	33.2	25.6	18.7	26	24	

Delivered 10.30 p.m.

CASE 82. Primigravida. B.P. $\frac{260}{120}$. Moderate albuminuria and marked oedema. Labour 3 hours. Spontaneous delivery with no haemorrhage.

Date.	2/7/38	6/7/38	13/7/38	20/7/38	25/7/38	28/7/38	30/7/38	1/8/38	2/8/38	4/8/38	5/8/38	8/8/38	11/8/38
Haem^{t}.	35%	38.5%	38%	41%	41%	42%	39%	40%	30%	23%	23%	23.5%	24.5%
Uric Acid.	7.6	6.2	4.9	3.8	4.1	4.5	4.9	5.3	7.2	6.3	5.4	4.0	4.0
Urea	27		15.5		17								

Delivered 8 p.m.

20 CHRONIC TOXAEMIC CASES.

CASE 83. 4th Para. B.P. $\frac{170}{110}$. Moderate albuminuria and gross oedema. Labour 13 hours. Delivered by forceps with no undue haemorrhage.

Date.	6/12/38	12/12/38 10.30 a.m.	12/12/38 2.30 p.m.	13/12/38 10 a.m.	13/12/38 12.15 p.m.	4.45 p.m	14/12/38	15/12/38	17/12/38	19/12/38
Haem^{tt}.	37.5%	39%	37%	39%	39.5%	33%	24%	23.5%	24%	25%
Uric Acid.	4.1	5.7	5.8	5.8	6.2	7.0	7.2	6.3	5.6	4.1
Urea.	17	15		17			52	32	19	

Delivered 2.30 p.m.

CASE 84. 7th Para. B.P. $\frac{230}{142}$. Moderate albuminuria and gross oedema. Labour 14 hours. Spontaneous delivery.

Date.	27/5/38	30/5/38	31/5/38	2/6/38	3/6/38	4/6/38	5/6/38	6/6/38	7/6/38	8/6/38	10/6/38	14/6/38	18/6/38.
Haem^{t}.	43%	44.5%	46%	47%	45%	42%	43%	42%	40.5%	39%	41.5%	43%	42%
Uric Acid.	5.7	5.6	5.4	6.4	6.4	6.6	6.0	5.7	6.0	5.6	5.2	5.6	4.7
Urea.	25.5					38			21				

Delivered 5 a.m.

CASE 85. 3rd Para. B.P. $\frac{180}{110}$. No albuminuria and no oedema. Labour 14 hours. Spontaneous delivery.

Date.	25/5/38	3/6/38 9.30 a.m.	3/6/38 1 p.m.	4/6/38	5/6/38	6/6/38	7/6/38	8/6/38.
Haem^{t}.	29%	33%	30.5%	32.5%	31%	30.5%	30.5%	32%
Uric Acid.	4.9	5.8	6.8	7.0	6.0	5.5	5.2	4.4
Urea.	27.6		39.3	39.3			37.3	

Delivered 12 M.D.

CASE 86. 6th Para. B.P. $\frac{160}{110}$. No albuminuria and marked oedema. Labour 26 hours. Spontaneous delivery.

Date.	9/5/38	10/5/38	12/5/38	13/5/38	14/5/38	17/5/38	19/5/38	24/5/38.
Haem^{t}.	38%	38.5%	37%	37%	32%	28%	32%	32.5%
Uric Acid.	7.3	7.3	8.5	8.2	6.8	4.6	4.2	4.0
Urea.	50.3			13				

Delivered 6.30 a.m.

CASE 87. 6th Para. B.P. $\frac{150}{110}$. No albuminuria and no oedema. Labour 56 hours. Delivered by forceps with no undue haemorrhage.

Date.	16/1/39	18/1/39 2.p.m.	19/1/39	20/1/39	21/1/39	23/1/39	26/1/39.
Haem^{t}.	41%	43%	39%	36%	37%	37%	39%
Uric Acid.	4.7	7.4	7.8	7.7	6.0	6.0	3.7
Urea.	22	57	75	76	48	44	44.6

Delivered 4 p.m.

CASE 88. 2nd Para. B.P. $\frac{154}{100}$. No albuminuria and slight oedema. Previous eclampsia. Labour 4 hours. Spontaneous delivery.

Date.	28/2/39	7/3/39	8/3/39	9/3/39	10/3/39	11/3/39	13/3/39.
Haem^{t}.	33%	41%	42%	39%	37%	37.5%	40%
Uric Acid.	4.2	4.4	4.4	5.1	4.5	4.3	4.6
Urea.	20	19	21	21	27	28	28

Delivered 11.30 p.m.

CASE 89. 4th Para. B.P. $\frac{170}{110}$. Slight albuminuria and oedema. Labour 9 hours. Spontaneous delivery.

Date.	8/6/38	13/6/38	14/6/38 8 p.m.	15/6/38	16/6/38	17/6/38	18/6/38	21/6/38.
Haem^{t}.	28%	28.5%	32%	35%	28%	27%	28%	30.5%
Uric Acid.	4.6	4.3		5.2	4.6	4.2	3.8	3.9

Delivered 1 a.m.

CASE 90. 4th Para. B.P. $\frac{140}{90}$. Slight albuminuria and oedema. Labour 3 hours. Spontaneous delivery.

Date.	10/2/38	14/2/38	15/2/38	17/2/38	21/2/38.
Haem^{t}.	28.5%	30%	29.5%	28.5%	29.5%
Uric Acid.	3.5	4.0	4.5	4.2	4.6
Urea.		18.2			

Delivered 13/2/38 at 10 p.m.

CASE 91. 3rd Para. B.P. $\frac{150}{80}$. Slight albuminuria and gross oedema. Labour 6 hours. Delivered by forceps because of marked maternal distress. Moderate P.P.H.

Date.	17/3/38	18/3/38	19/3/38	21/3/38	22/3/38	23/3/38	24/3/38.
Haem^{t}.	27.5%	27%	29%	23%	20%	21%	21%
Uric Acid.	4.1	4.3	6.9	10.1	9.8	7.0	5.6
Urea.		18.5	14.3	66.3	50	28	

Delivered
20/3/38 8 p.m.

CASE 92. 2nd Para. B.P. $\frac{120}{70}$. Gross albuminuria and oedema. Labour 20 hours. Delivered by forceps with no undue haemorrhage.

Date.	11/3/38	12/3/38	14/3/38	15/3/38	16/3/38	18/3/38	21/3/38	24/3/38.
Haem^{t}.	31%	28%	29%	30%	30.5%	29.5%	30.5%	31%
Uric Acid.	5.9	8.6	4.3	3.6	3.4	2.4	1.5	2.9
Urea.	30	50	28.5	25.9				

Delivered
10.30 p.m.

CASE 93. 5th Para. B.P. $\frac{210}{80}$. Gross albuminuria and oedema. Labour 11 hours. Complete miscarriage.

Date.	24/1/38	26/1/38	28/1/38	29/1/38	31/1/38	2/2/38	4/2/38	8/2/38.
Haem^{t}.	28%	27%	24%	22%	20%	17.5%	16%	18.5%
Hb.	62.5	60	50	44.5	40.5	35	31.5	38
Protein.	5.8	5.8	5.1	5.3	5.3	4.9	5.3	5.3
Uric Acid.	7.96	6.7	10.6	9.5	8.3	7.4	7.6	6.9
Urea.		135	208	208	186	153	96	54
Bilirubin			6.2		1.5			

CASE 94. 3rd Para. B.P. $\frac{180}{100}$. No albuminuria and slight oedema. Labour 18 hours. Spontaneous delivery.

Date.	5/10/38	7/10/38	10/10/38	11/10/38	17/10/38
Haem^{t}.	33.5%	34%	27.5%	24%	29.5%
Hb.	68	68	56	48.5	59.5
Protein.	6.56	6.4	5.9	5.9	6.9
Uric Acid	2.48	2.5	4.0	3.3	3.3
R.B.C.		3,880,000	3,480,000		

Delivered
9/10/38 at 11 p.m.

CASE 95. 3rd Para. B.P. $\frac{210}{100}$. No albuminuria and no oedema. Labour 8 hours. Spontaneous delivery.

Date.	16	18	20	22	24	25	27	30.
Haem^{t}.	39%	41.5%	41%	37.5%	35.5%	36.5%	34.5%	37.5%
Hb.	83.5	86.5	87.5	78.5	74.5		71.5	78.5
Uric Acid.	4.09	3.92	3.92	3.64	3.5	3.1	3.56	3.5
Protein.	6.4	6.6	6.44	6.88	6.33			6.56
Urea.	34.2	27.5	32	27.4		19		

Delivered 10 p.m.

CASE 96. 10th Para. B.P. $\frac{180}{100}$. Gross albuminuria and moderate oedema. Labour 3 hours. Spontaneous delivery.

Date.	21/4/38	22/4/38	24/4/38	25/4/38	26/4/38	28/4/38	30/4/38.
Haem^{t}.	34%	33%	36%	33%	31%	31%	33%
Uric Acid	5.2	6.4	5.4	6.1	5.9	5.2	4.4
Urea.	13.7						

Delivered 23/4/38 at 9.30 p.m.

CASE 97. 4th Para. B.P. $\frac{140}{90}$. Slight albuminuria and oedema. Previous eclampsia. Labour 4 hours. Spontaneous delivery of a still-born child.

Date.	10/2/38	1/3/38	8/3/38	9/3/38	11/3/38	15/3/38.
Haem^{t}.	35%	35%	45%	39%	38.5%	38%
Uric Acid.	3.1	3.7	3.8	4.6	4.2	3.6
Urea.	15.3					

Delivered 8/3/38 at 2 a.m.

CASE 98. 3rd Para. B.P. $\frac{170}{100}$. Moderate albuminuria and no oedema. Labour 7 hours. Spontaneous delivery.

Date.	22/2/38	4/3/38	8/3/38 12.30 p.m.	9/3/38	10/3/38	12/3/38	15/3/38.
Haem^{t}.	32%	33%	39.5%	34%	32%	33%	36%
Uric Acid	2.2	2.7	3.1	3.7	3.8	3.2	3.1

Delivered 8/3/38, 12.30 a.m.

CASE 99. 10th Para. B.P. $\frac{180}{100}$. Slight albuminuria and gross oedema. Labour 2 hours. Spontaneous delivery. The patient had a ? epileptic fit about 18 hours after delivery.

Date.	27/4/38	3/5/38	5/5/38 10.45 a.m.	5/5/38 12.30 p.m.	5/5/38 4.15 p.m.	6/5/38	7/5/38	8/5/38	10/5/38.	20/5/38.
Haem^{t}.	20.5%	21%	25%	25.5%	25.5%	27%	21%	21%	24.5%	24%
Uric Acid.	3.6	2.4	2.4	2.6	2.6	4.3	4.2	3.4	2.2	2.0
Urea.			17.8			24			10.5	

Delivered 10.50 a.m.

fit.

CASE. 100. 3rd Para. B.P. $\frac{150}{100}$. No albuminuria and slight oedema. Labour 27 hours. Spontaneous delivery.

Date.	26/7/38	5/8/38	6/8/38	8/8/38	9/8/38	10/8/38	12/8/38.
Haem^{t}.	36%	37%	39%	33%	32.5%	32%	36%
Uric Acid.	4.0	3.7	4.5	5.0	4.5	3.4	3.0

Delivered 7/8/38 at 1 a.m.

CASE 101. 3rd Para. B.P. $\frac{160}{100}$. Moderate albuminuria and no oedema. Labour 5 hours. Spontaneous delivery.

Date.	12/2/38	14/2/38	15/2/38	17/2/38.
Haem^{t}.	37%	36%	36%	37%
Uric Acid.	4.2	4.3	4.1	4.3
Urea.	20.1	19.3		

Delivered 13/2/38 at 4 p.m.

CASE 102. 2nd Para. B.P. $\frac{184}{120}$. Gross albuminuria and no oedema. Labour 2 hours. Spontaneous delivery.

Date.	26/5/38	27/5/38	28/5/38 7 p.m.	30/5/38	31/5/38	1/6/38	4/6/38	7/6/38	10/6/38.
Haem^{t}.	34%	35.5%	35%	29%	26%	24.5%	25%	27.5%	30.5%
Uric Acid.	5.8	5.4	6.3	5.9	5.6	5.5	5.1	4.8	4.5
Urea.	29.5			34	19				
Bilirubin		≤1	8	.5					

Delivered 11 p.m.

CASE 103. 4th Para. B.P. $\frac{160}{120}$. Moderate albuminuria and no oedema. Labour 4 hours. Spontaneous delivery.

Date.	27/3/38	28/3/38	29/3/38	30/3/38.
Haem^{t}.	41%	40%	37%	32%
Uric Acid.	5.6	5.2	6.3	5.9
Urea.	51.3	69.3	57.3	53

Delivered 4 a.m.

42 ECLAMPTIC CASES

Antepartum Cases.

CASE 104. 2nd Para. She became blind 4 hours before admission, but had no fit. 2 fits after admission. Delivered the next day after 6 hours labour. High B.P. persisted after delivery 240/110 Spontaneous delivery.

Date.	26/7/39 12.40p.m.	2.10p.m.	3.10p.m.	4.30 p.m.	7.25 p.m.	10a.m. 27/7/39	9.30 p.m.	12 M.D. 28/7/39	29/7/39	30/7/39	31/7/39
Haem^{t}.	38%	36%	36%	35%	38%	32%	-	30%	30%	30%	30.5%
Uric Acid.	4.2	5.7	5.9	6.5	6.9	7.6	8.2	8.6	8.4	5.7	5.2
Urea.	25		31		27	35	43	42	34		31

at 8a.m. blind — fit at 1.40p.m. — fit at 7.23p.m. — Delivered 7 p.m.

CASE 105. Primigravida. 4 fits before admission and none subsequently. Delivered the next day after a 7 hour labour. Delivered by forceps with no undue haemorrhage.

Date.	24/1/39 10 a.m.	3 p.m.	25/1/39 10 a.m.	3.10 p.m.	26/1/39	27/1/39	28/1/39	30/1/39	3/2/39.
Haem^{t}.	38%	37.5%	39.5%	42.5%	36.5%	36%	34%	37%	37%
Uric Acid.	8.2	7.8	9.0	8.2	7.8	6.5	5.1	4.0	3.7
Urea	42	40	37		32	16			19

4 fits since 11 p.m. — Delivered 3.15 p.m.

CASE 106. 2nd Para. Admitted after 12 fits in rapid succession. She was semi-conscious and very difficult to manage during the next 2 days. Delivered 4 days after the fits. Labour 3 hours. Spontaneous delivery of a macerated foetus.

Date.	11p.m. 11/6/39	10 a.m. 12/6/39	13/6/39	14/6/39	15/6/39. 10 a.m.	15/6/39. 4 p.m.	16/6/39	19/6/39	22/6/39
Haemt.	-	32%	36%	35%	41%	33%	34%	37%	35%
Uric Acid.	9.5	11.0	9.4	8.1	7.8	6.9	6.3	3.6	4.1
Urea.	22	55	57	31	42		23		27
	12 fits				Delivered 6 a.m.				

CASE 107. Primigravida. Patient had 4 fits on day of admission and then improved. B.P.fell and albuminuria diminished. 24/1/39 B.P. rose again and albuminuria increased and foetal movements ceased. Delivered spontaneously 4 days later after a 38 hour labour of a macerated foetus.

Date.	16/1/39. 10 a.m.	2.30 p.m.	17/1/39	18/1/39	19/1/39	20/1/39	21/1/39	22/1/39	23/1/39	24/1/39	25/1/39	27/1/39.
Haemt.	37%	34.5%	30%	35%	35%	36%	37%	37%	37.5%	40%	39.5%	41.5%
Uric Acid.	7.2	7.0	7.2	6.2	5.7	5.7	5.6	6.9	6.8	5.1	5.2	3.9
Urea.	35		35	27	30	32	30		33	31	22	18
Bilir.	.5	.5	0	1.4	0	0	-	-	-	-	-	-
	3 fits since 3 a.m.	fit 11.30 a.m.									worse	

Date.	28/1/39	29/1/39	30/1/39	31/1/39	2/2/39	8/2/39
Haemt.	41.5%	36%	-	38%	40%	37%
U.Acid.	4.3	3.4		5.4	5.6	3.6
Urea.	49		27	30	23	24
	delivered 9.20 a.m.					

CASE 108. Primigravida. Patient had 2 fits before admission and then improved greatly. B.P. fell gradually during next 9 days. Delivered spontaneously 11 days after admission after a 13 hour labour. Moderate albuminuria and oedema.

Date.	15/7/38	16/7/38	17/7/38	18/7/38	19/7/38	21/7/38	23/7/38	24/7/38	25/7/38 10 a.m.	25/7/38 2.45p.m.	26/7/38.
Haem^{t}.	32%	24%	24.5%	24%	24%	24.5%	24%	25.5%	28%	24%	23%
Uric Acid.	9.5	8.0	6.4	6.0	5.5	5.1	5.0	5.1	5.3	5.6	5.6
Urea.	19								24.5%		38%

2 fits 12 a.m.& 1.30 a.m.

delivered 9.45 a.m.

Date.	27/7/38	28/7/38	1/8/38
Haem^{t}.	21%	21.5%	26.5%
Uric Acid	5.2	3.8	3.6
Urea.	17.5		

CASE 109. Primigravida. Patient had 1 fit before admission. Severe headaches, marked oedema of face and high B.P. persisted till after delivery. Patient delivered spontaneously, 4 days after the fit, after a 20 hour labour.

Date.	22/1/39	23/1/39	24/1/39	25/1/39 2p.m.	26/1/39	27/1/39	28/1/39	30/1/39	2/2/39	9/2/39.
Haem^{t}.	36%	36.5%	37%	38%	37%	37%	37%	39%	44%	40%
Uric Acid	7.0	6.2	6.4	6.7	6.6	5.8	5.0	3.3	3.5	4.6
Urea.	37		34	36	35	23			23.5	

Fit 21/1/39.

delivered 7.30 p.m.

CASE 110. Primigravida. Only 6 months pregnant. Patient had several fits on the day before admission; Semi-conscious on admission, but gradually improved during the next 6 days.

Date.	7.30p.m. 26/7/39	10a.m. 27/7/39	28/7/39	29/7/39	30/7/39	31/7/39.
Haem^{t}.	38%	39.5%	38%	37.5%	37%	38%
Uric Acid.	8.2	8.5	7.5	4.8	4.0	3.8
Urea.	48	40	26			21

fits 25/7/39.

(Undelivered)

CASE 111. Primigravida. 3 fits after admission and then improvement occurred. Delivered 2 days later after a 6 hour labour. Spontaneous delivery.

Date.	10 a.m. 4/7/38	5/7/38	6/7/38. 10 a.m.	4 p.m.	11.15 p.m.	7/7/38	8/7/38	9/7/38	11/7/38.
Haem^{t}.	37%	36.5%	40%	37%	38%	36.5%	33%	35%	33%
Uric Acid.	10.1	11.2	11.5	11.4	11.8	12	8.8	6.3	5.0
Urea.	44.5	44		62.6		58.3	32.5		30.5
Bili-rubin.	3.2	3.2	5.7	5.8	6.6	5.4	3	2.6	.8

3 fits 2 to 5 a.m.

Delivered 10.55 p.m.

CASE 112. 3rd Para. 2 fits on day before admission. Delivered spontaneously of a still-born child on the day of admission after 3 hours labour.

Date.	6/3/39. 10 a.m.	2.45 p.m.	4.30 p.m.	7/3/39	8/3/39	9/3/39	10/3/39	13/3/39	16/3/39.
Haem^{t}.	40%	40%	40%	38%	37%	33%	33%	40%	40%
Uric Acid.	7.2	7.1	6.8	6.0	5.8	4.5	4.3	4.5	4.6
Urea.	39		33	33	22	27	26		48

2 fits 5/3/39

delivered 1.15 p.m.

CASE 113. Primigravida. 6 months pregnant. 3 fits on day before admission. Her condition remained unchanged during the next few days. Delivered spontaneously 9 days after the fits. 4 hours labour. Condition gradually improved after delivery.

Date.	10 a.m. 23/7/38	25/7/38	26/7/38	27/7/38	28/7/38	29/7/38	30/7/38. 10.40a.m.	5 p.m.	1/8/38	2/8/38	4/8/38	8/8/38.
Haem^{t}.	32%	34%	34%	35.5%	34%	35%	39%	32.5%	32%	32%	34.5%	38%
Uric Acid.	7.9	7.7	8.4	8.4	8.2	7.7	7.8	7.6	6.6	5.6	4.9	4.2

3 fits 12.30 p.m. to 6.30 p.m.

Delivered 11 a.m.

CASE 114. Primigravida. 4 fits on day of admission. Gradual improvement during next few days. Developed severe tonsillitis 4 days after admission and had to be transferred to another hospital.

Date.	26/3/38 12.30 p.m.	26/3/38 4.30 p.m.	27/3/38	28/3/38	29/3/38	30/3/38.
Haem^{t}.	34%	35%	36%	35.5%	33.5%	43%
Uric Acid.	5.8	6.4	7.5	6.6	6.0	7.4
Urea.	15.6				18.3	16.5

fits 9.30 & 10.30 a.m. — fit 3 p.m. — fit 6 p.m. — Delivered 31/3/38.

CASE 115. 3rd Para. 6 fits on day of admission and patient remained comatose for many hours. She had improved 2 days after admission and was delivered spontaneously, after a 2 hour labour, 3 days after the fits.

Date.	10.30 a.m. 24/1/38	25/1/38	26/1/38	27/1/38	28/1/38	31/1/38	7/2/38.
Haem^{t}.	34.5%	35%	33%	32.5%	32%	34%	36%
Hb.	69	68.5	64	63.5	63.5	65.5	67.5
Protein.	7.2	7.5	6.9	7.09	6.9	7.2	6.9
Uric Acid.	7.9	9.2	6.2	6.6	5.3	3.5	4.8
Urea.	26	45	47.3	20.6			

4 fits 4a.m.-8a.m. — Fit at 10.5 a.m. — fit 12 M.D. — Delivered 2 A.M.

CASE 116. Primigravida. 1 fit before admission . Improved slightly during next few days. Delivered spontaneously 5 days after admission after a 20 hour labour.

Date.	17/12/38	19/12/38	20/12/38	21/12/38	22/12/38	23/12/38	27/12/38.
Haem^{t}.	34%	34%	32%	37%	39%	38%	35%
Uric Acid.	9.1	9.4	9.2	7.7	7.8	6.3	4.4
Urea.	42	21	20				22

1 fit 16/12/38 at 6p.m.

Delivered 12.40 a.m.

CASE 117. Primigravida. 4 fits before admission. Improved gradually during next few days. Delivered spontaneously 9 days after the fits, after a 6 hour labour. Child stillborn.

Date.	28/2/38	1/3/38	2/3/38	3/3/38	4/3/38	5/3/38	7/3/38	8/3/38	9/3/38	11/3/38.
Haem^{t}.	35.5%	34.5%	34%	36%	36.5%	35.5%	35%	33%	31%	29.5%
Uric Acid.	10.0	9.3	8.5	8.8	8.8	8.0	6.5	6.1	5.2	3.7

4 fits on 26/2/38.

delivered 6/3/38 at 2.20 p.m.

CASE 118. 4th Para. 2 fits before admission. Improved during next few days. Delivered by Caesarian section 5 days after the fits. Marked oedema.

Date.	14/2/38	15/2/38	16/2/38	17/2/38	21/2/38	23/2/38.
Haem^{t}.	32%	32%	31%	32%	32%	31%
Uric Acid.	6.3	5.3	5.2	4.8	3.3	2.5
Urea.	15.8	14				

2 fits 13/2/38 10.30 a.m. & 11.15 a.m.

delivered 18/2/38 at 8 p.m.

CASE 119. Primigravida. 2 fits before admission. Delivered the next day after 24 hours labour. Manual breech delivery. Marked oedema.

Date.	18/1/37. 10 a.m.	18/1/37. 4 p.m.	19/1/37	20/1/37	21/1/37	22/1/37	(22/1/37)	24/1/37	29/1/37.
Haem^{t}.	40.5%	38%	40.5%	37%	30%	31%		30%	34%
Hb.	78.5	74.5	79.5	73	60	62.5		61.5	68.5
Protein	7.0	6.5	7.1	6.4	5.9	6.7		7.6	
Uric Acid.	6.15	5.8	6.15	7.1	5.8	4.2		3.1	4.1
Urea.	30		17.9	18.4	21.9				

2 fits 6.30 a.m.& 9.30 a.m.

Delivered 5.30 a.m.

ECLAMPTIC CASES (Contd)

CASE 120. 3rd Para. Fatal Case - See TEXT.

CASE 121. 2nd Para. Fatal Case - See TEXT.

[illegible] a.m.	4.45 p.m.	5.15 p.m.	9 p.m.
40%	46.3%	45%	60%
[illegible]	8.1	[illegible]	[illegible]
	39		

[illegible] began 1 a.m. Fit at 7.30. p.m. Delivered 8.30 p.m.

Date.	30/4/38	1/5/38	2/5/38	3/5/38	5/5/38	9/5/38	11/5/38.
Haem^{t}.	28%		25%	24%	[illegible]	30%	29.5%
Uric Acid.	10	7.2	6.1	[illegible]	[illegible]	5.4	5.7
Urea.	35.5	30					

ECLAMPTIC CASES

Intrapartum Cases.

CASE 122. Primigravida. Admitted as severe pre-eclamptic case. B.P. ranged from $\frac{160}{120}$ to $\frac{210}{110}$ for several days. Moderate albuminuria and oedema. Patient had one fit during labour 1 hour before delivery. Labour lasted 20 hours and delivery was spontaneous. Improvement began 2 days after delivery.

						28/4/38				29/4/38.	
Date.	22/4/38	23/4/38	24/4/38	25/4/38	26/4/38	10 a.m.	4.45 p.m.	8.15 p.m.	9 p.m.	1 a.m.	10a.m.
Haem^{t}.	36.5%	36%	37%	37%	38.5%	40%	46.5%	45%	40%	40.5%	32%
Uric Acid.	5.8	7.0	6.2	6.1	5.8	6.0	6.1	8.3	8.0	9.1	10.3
Urea.		36					39			49	66.5

Pains began 1 a.m. — Fit at 7.30. p.m. — Delivered 8.30 p.m.

Date.	30/4/38	1/5/38	2/5/38	3/5/38	5/5/38	8/5/38	11/5/38.
Haem^{t}.	26%		23%	24%	26.5%	30%	29.5%
Uric Acid.	10	7.2	5.1	4.1	3.4	3.4	3.7
Urea.	56.5	30					

CASE 123. Primigravida. Admitted as severe pre-eclamptic. She had 4 fits at the onset and during labour. Delivered by forceps after 13 hours labour. No undue haemorrhage.

Date.	26/9/38	3/10/38	5/10/38	7/10/38	9/10/38 11 a.m.	10/10/38	11/10/38	12/10/38	13/10/38	14/10/38
Haem^{t}.	28%	26%	28%	26%	29%	28%	26%	24.5%	24.5%	26%
Uric Acid.	8.0	8.5	6.7	6.4	10.0	11.6	10.5	11.6	10.3	7.6
Urea.	38		21.5		44.3	54	52	53		23.5

2 fits. 2 fits. Delivered 10.30 p.m.

Date.	16/10/38	19/10/38.
Haem^{t}.	27%	29%
Uric Acid.	4.7	3.8

CASE 124. Primigravida. Admitted as pre-eclamptic case but had fits immediately after admission. She had 6 fits in all. Labour began during the fits and lasted 6 hours. Spontaneous delivery.

Date.	11/11/38 2.15 p.m.	3.45 p.m.	4.45 p.m.	7.30 p.m.	12/11/38 10 a.m.	4 p.m.	13/11/38	14/11/38	15/11/38.
Haem^{t}.	40%	37%	38%	37%	34.5%		37%	36.5%	38.5%
Uric Acid.	6.5	7.6	8.8	8.5	10.2	10.0	9.8	8.9	8.1
Urea.	44		44	42	38		32		27

fit 2 p.m. fit 3.15 p.m. fit 4.15 p.m. fit 7.5 p.m. fit 8.30 & 10.30 p.m. Delivered 3.30 a.m.

Date.	16/11/38	18/11/38	21/11/38	24/11/38.
Haem^{t}.	37%	39.5%	40%	42%
Uric Acid.	7.0	5.4	5.7	5.8
Urea.	24.5	25.2	31	27

CASE 125. Primigravida. Admitted as pre-eclamptic with normal blood pressure but marked albuminuria and oedema. She had 1 fit in labour. Labour 6 hours. Spontaneous delivery.

Date.	7/5/38	8/5/38	9/5/38	10/5/38	12/5/38	14/5/38	18/5/38.
Haem^{t}.	25%	21%	19%	19.5%	19%	20.5%	24%
Uric Acid.	5.2	6.3	6.4	5.6	4.4	2.6	3.6

Fit at 7.p.m.
Delivered 7.30 p.m.

CASE 126. 2nd Para. Admitted as severe pre-eclamptic. B.P. $\frac{160}{110}$. Gross albuminuria and oedema. The patient had 2 fits during labour which lasted only about 4 hours. Spontaneous delivery.

Date.	21/6/38	22/6/38	23/6/38	24/6/38 10 a.m.	24/6/38 4 p.m.	25/6/38	26/6/38	27/6/38	30/6/38	6/7/38.
Haem^{t}.	36%	39%	36.5	40%	36%	32%	33%	34%	35%	37%
Uric Acid.	5.1	5.3	5.4	7.0	7.2	6.7	5.8	4.8	3.6	3.4
Urea.	34	43	38	69.5				19		

Fits 6.a.m. & 7.30 a.m.
Delivered 8.30 a.m.

CASE 127. Primigravida. Admitted as normal case but had 2 fits during labour and one 2 hours post-partum. Labour 21 hours. Spontaneous delivery with slight haemorrhage.

Date.	26/4/38 10 a.m.	2.15 p.m.	2.50 p.m.	3.40 p.m.	4.45 p.m.	9 p.m.	27/4/38	28/4/38	29/4/38	2/5/38	5/5/38
Haem^{t}.	31.5	32%	32%	34.5%	27.5%	27.5%	22%	23%	21%	22%	25%
Uric Acid.	4.4	5.3	5.4	5.8	6.2	7.8	5.8	5.2	4.8	3.6	3.9
Urea.	21					29					

Fit 1.30 p.m. Fit 3.7 p.m. Delivered 3.35 p.m. Fit 5 p.m.

CASE 128. Primigravida. Admitted as case of contracted pelvis. She had 8 fits during labour and 3 fits post-partum. Labour lasted 48 hours. Delivered by forceps with no undue haemorrhage.

Date.	19/5/38 4.20 p.m.	5.30 p.m.	9.30 p.m.	20/5/38	21/5/38	23/5/38	25/5/38	28/5/38	1/6/38.
Haem^{t}.	40%	39%	40.5%	37.5%	36%	34%	35%	41%	41%
Uric Acid.	10.2	11.5	11.4	10.2	9.2	6.2	3.2	3.8	3.8
Urea.	90.3		97.3			35.5	30.5		

8 fits. 12 M.D. to 4.10 p.m. Delivered 5 p.m. Fit 7.20 p.m. Fits 9.30 p.m. & 11.30 p.m.

CASE 129. Primigravida. Patient had 4 fits during labour. Labour lasted 26 hours. Delivered by forceps with no undue haemorrhage. Marked Oedema.

Date.	21/5/39 9.15 p.m.	21/5/39 9.45 p.m.	22/5/39 10. a.m.	23/5/39	24/5/39	25/5/39	27/5/39	29/5/39	1/6/39
Haem^{t}.	-	-	-	35.5%	40%	39%	36%	32%	33%
Uric Acid.	7.1	7.3	11.0	7.4	6.4	5.4	4.0	3.3	4.2
Urea.	40		67	47	60	51	48	46	39

Fit 8.8 p.m. & 8.30 p.m.

Fit 10.30 p.m. and 11.5 p.m. Delivered 12.30 a.m.

CASE 130. 2nd Para. 2 fits before admission. Labour 44 hours. Spontaneous delivery.

Date.	6/5/38 10.30 a.m.	6/5/38 9 p.m.	7/5/38	8/5/38	9/5/38	10/5/38	12/5/38	17/5/38	7/6/38.
Haem^{t}.	46%	47%	41%	35%	35%	33.5%	34%	32%	36%
Uric Acid.	8.6	9.3	9.4	8.8	6.2	5.9	5.1	5.0	5.2
Urea.	17.6		47.3			18			

2 Fits 12.30 a.m. & 1 a.m.

Delivered 6.20 a.m.

CASE 131. Primigravida. Patient had 3 fits on day of admission after the onset of labour. Labour 12 hours. Manual breech delivery with no undue haemorrhage.

Date.	10 a.m. 13/5/38	14/5/38	17/5/38	19/5/38	24/5/38.
Haem^{t}.	40%	32%	34%	34%	35%
Uric Acid.	7.2	8.1	6.0	3.6	3.2

3 Fits 2 to 4 a.m.

Delivered 11.20 a.m.

CASE 132. Primigravida. Patient had 4 fits during labour. Labour 16 hours. Delivered by forceps with no undue haemorrhage. Moderate oedema.

Date.	26/1/39 12.15 p.m.	2.20 p.m.	4.30 p.m.	9.40 p.m.	27/1/39 10 a.m.	3 p.m.	28/1/39	29/1/39	30/1/39	1/2/39	4/2/39
Haem^{t}.	35.5%	37%	34%	35%	28.5%	28%	31.5%	29%	29.5%	31%	32%
Uric Acid.	9.8	9.2	9.8	10.2	12.2	10.6	8.3	4.8	3.5	3.3	2.7
Urea.	34.5	26	28	32	34	43	31	34	22		
Urine vol.					15		38	76	109		

Fits 9 a.m. & 12.10 p.m.

Fit 12.15 p.m.

Fit 9.30 p.m.

Delivered 10.40 p m.

CASE 133. Primigravida. Patient had 9 fits before and during labour. Labour 8 hours. Delivered by forceps with no undue haemorrhage.

	19/9/38			20/9/38								
Date.	10 a.m.	4.30 p.m.	7.30 p.m.	10 a.m.	4 p.m.	21/9/38	22/9/38	23/9/38	24/9/38	27/9/38	29/9/38	4/10/38.
Haem^{t}.	38.5%	37%	35%	38%	35.5%	34.5%	35%	32%	33%	32.5%	32%	31%
Uric Acid.	7.8	8.4	8.4	7.6	7.1	5.0	3.4	2.6	2.1	1.8	2.0	2.2
Urea.	24						27					
Bilirubin.	2.8	2	2			2.4	2.5	1.9	1.4	1.6		

Fits from 4.30 a.m. till 4.40 p.m.

Delivered 7.3 p.m.

CASE 134. Primigravida. Patient had one fit during labour. Very little urine was passed during the first day post partum. Labour 19 hours. Spontaneous delivery. Marked oedema.

	18/4/39			19/4/39								
Date.	10 a.m.	12 m.d.	3 p.m.	10 a.m.	4 p.m.	20/4/39	21/4/39	22/4/39	25/4/39	27/4/39	1/5/39	6/6/39.
Haem^{t}.	44%	44%	43%	39%	39%	39%	34%	34%	35%	33%	30%	29%
Uric Acid.	9.1	9.1	9.2	10.4	10.2	10.0	8.9	7.8	4.6	3.5	4.0	
Urea.	45	35	44	102	111	140	130	101	63	58	63	

Fit 7.a.m.

Delivered 7.30 p.m.

CASE 135. 3rd Para. She had 4 fits during labour. Labour 24 hours. Spontaneous delivery. This patient was treated by the Strogonoff method and had chloroform 4 times during labour. She became slightly jaundiced on the third day post-partum and remained so for several days.

	13/6/39		14/6/39							
Date.	5.30 p.m.	11 p.m.	10 a.m.	3.30 p.m.	15/6/39	16/6/39	17/6/39	18/6/39	19/6/39	22/6/39.
Haem^{t}.	35%	-	31%	30%	29%	29.5%	30%	-	31.5%	28.5%
Uric Acid.	7.9	8.3	9.4	9.2	9.6	7.4	3.2	2.8	2.4	3.1
Urea.	26	31	41	50		34			24	20
Bilirubin.	<1	<1	<1	<1	1.8	7.8	9	11	7	<1

4 Fits since 2 p.m.

Delivered 3.30 a.m.

CASE 136. Primigravida. Patient had 3 fits in labour. Labour 30 hours. Manual breech delivery.

	30/6/39				
Date.	10 a.m.	2 p.m.	1/7/39	3/7/39	5/7/39
Haem^{t}.	45%	41%	-	35%	33%
Uric Acid.	12.5	12.8	-	6.8	5.0
Urea.	24	27	32	26	44
Bilirubin.	3.6		<1		

3 Fits
10 p.m. to 4 a.m.
Delivered 5.30 a.m.

CASE 137. 2nd Para. Patient had 2 fits during labour. Labour lasted 8 hours. Spontaneous delivery.

Date.	10 a.m. 25/1/38	26/1/38	27/1/38	28/1/38	31/1/38	4/2/38.
Haem^{t}.	38.5%	31%	28%	28.5%	28%	30%
Hb.	84.5	66.5	59.5	59.5	58.5	65
Protein.	5.58	5.3	5.4	5.6	5.7	6.4
Uric Acid.	5.9	7.9	7.4	7.0	3.7	3.8
Urea.		56	32	17.3		

Fit 3 a.m.
Fit 2 p.m. Delivered 10 p.m.

Post-Partum Eclamptics.

CASE 138. Patient delivered after 11 hours labour and had 2 fits during the first 4 hours post-partum.

Date.	20/4/38 10 a.m.	2.30 p.m.	21/4/38	22/4/38	25/4/38	29/4/38.
Haem^{t}.	42%	39.5%	34%	33%	36%	38%
Uric Acid.	5.7	7.2	7.4	6.4	2.7	2.9
Urea.	39		42	18.7		

Delivered 5 a.m.
Fits 7.30 a.m. and 9.30 a.m.

CASE 139. 8th Para. Patient was delivered at home and had 3 fits immediately post-partum. Urinary output was small for a few days after admission.

Date.	1/5/39	2/5/39	3/5/39	4/5/39	5/5/39	8/5/39.
Haem^{t}.	37%	36%	34%	33.5%	35%	35.5%
Uric Acid.	7.6	7.0	5.8	4.5	3.7	2.7
Urea.	108	106	65	49	44	36

3 Fits
10 p.m.-12 m.n.
Delivered 8 p.m.

CASE 140. 2nd Para. Delivered of twins and then had 4 fits in rapid succession. Strogonoff treatment administered. Labour 3 hours.

	21/4/39									
Date.	8.40 a.m.	10 a.m.	12 m.d.	2 p.m.	4.30 p.m.	22/4/39	24/4/39	25/4/39	27/4/39	29/4/39.
Haem^{t}.	-	39%	-	41%	40.5%	37%	34.5%	35%	33%	33%
Uric Acid.	10.2	11.1	9.5	10.3		11.6	7.7	5.4	4.1	3.7
Urea.	80	92			80	59	45	34	28	

Fits at 5, 6,
7 & 8 a.m.
Delivered twins 20/4/39 11.p.m.
and 21/4/39 at 12-15 a.m.

CASE 141. Primigravida. Fits began at delivery and she had 5 in about 2 hours. Labour lasted 8 hours.

Date.	25/7/39 9.45 a.m.	12.30 p.m.	3 p.m.	26/7/39	27/7/39	28/7/39	29/7/39	31/7/39.
Haem^{t}.	38%	38%	37%	35%	35.5%	31%	33%	34%
Uric Acid.	7.8	7.8	7.7	7.4	7.1	6.5	4.9	4.1
Urea.	23		26	35	35.5	26	26	

Delivered 6.30 a.m.
Fits at 6.30, 7.15, 8.20, 9.5 and 9.40 a.m.

CASE 142. Primigravida. Admitted as normal case. Spontaneous delivery after an 18 hour labour. She had 1 fit 8½ hours after delivery.

Date.	12/5/39 3.50 p.m.	4.30 p.m.	8 p.m.	13/5/39	14/5/39	15/5/39	16/5/39	18/5/39	25/5/39.
Haem^{t}.									40%
Uric Acid.	6.4	7.2	7.8	7.5	6.2	5.5	5.4	3.8	4.2
Urea.	47	46	44	48	58	39	36	28	

Delivered 8 a.m.
Fit
3.40 p.m.

CASE 143. Primigravida. This patient was in hospital for several days before delivery - as a severe pre-eclamptic case. B.P. ranged from $\frac{172}{106}$ to $\frac{204}{140}$. Moderate albuminuria and oedema.

She was delivered spontaneously after an 8 hour labour, 10 minutes later she had a fit and this was followed by 3 more. She remained unconscious for a long time after these fits but by the next day she was conscious, felt well and her B.P. was $\frac{132}{88}$.

Date.	31/1/39	1/2/39	2/2/39	3/2/39	4/2/39	6/2/39 10 a.m.	6/2/39 3.5 p.m.	6/2/39 3.25 p.m.	6/2/39 4.40 p.m.	6/2/39 5.30 p.m	6/2/39 8 p.m.
Haem^{t}.	34.5%	33%	33%	36%	36%	37%	39%	39%	39%	39%	39%
Uric Acid.	4.3	4.9	5.2	5.1	4.8	5.3	6.2	6.6	7.9	8.4	8.7
Urea.	29		29	25	27	34		28.6	24.6	30.3	

Fit 3.10 p.m. Fit 3.45 p.m. Fit 4.30 p.m. Fit 8.12 p.m.

Delivered 3 p.m.

Date.	6/2/39 10.15 p.m.	7/2/39 → 10 a.m.	4 p.m.	8/2/39	9/2/39	10/2/39	11/2/39	13/2/39	15/2/39	20/2/39.
Haem^{t}.	39%	37%	34.5%	34%	32%	33%	34%	37.5%	40%	39%
Uric Acid	8.6	8.2	7.7	6.8	4.9	4.4	4.3	3.7	4.1	4.0
Urea.	34.6	47	37	43	39.7	36	35	36	32.4	

CASE 144. Primigravida. Admitted as normal case. Delivered spontaneously after 9 hours labour. 2½ hours after delivery she suddenly began to have severe convulsions and had 12 fits in 1½ hours. B.P. 190/118. She regained consciousness soon after the last fit and rapidly improved.

Date.	13/3/39 After 2nd fit	After 7th fit	11.30 a.m.	4.30 p.m.	7.30 p.m.	14/3/39	15/3/39	17/3/39	20/3/39.
Haem^{t}.			38%	35%	34%	33%	35%	33%	35%
Uric Acid	3.0	4.3	8.9	7.5	6.4	5.3	3.8	3.3	3.3
Urea.	22		33			36			23

Delivered 4.30 a.m.
12 fits from 7.a.m.
to 8.30 a.m.

CASE 145. Primigravida. Fatal case. See text.

Date.	2 p.m. 23/12/38	p.m. 24/12/38.
Haem^{t}.	34%	-
Uric Acid.	8.3	14.2
Urea.	57	112

Delivered 6 p.m.
3 fits since
2 a.m.

10 AZOMAN CASES.

CASE 146. Non-pregnant female with schizophrenia. First treatment with Azoman. Patient had 2 severe convulsions. Latent period between injection and first fit was 5 secs. First fit lasted 32 seconds and was followed by $2\frac{1}{2}$ minutes coma; patient then had a second fit lasting 42 seconds with $8\frac{1}{2}$ minutes coma.

	23/6/39.					
Date.	11.20 a.m.	11.25 a.m.	11.50 a.m.	2.30 p.m.	8.30 p.m.	24/6/39.
Haem^{t}.	41%	2 Fits.	46%	38%	44%	39%
Uric Acid.	3.3		4.9	8.7	8.1	6.4
Urea.	25		26	25	24	37

CASE 147. Male with manic-depressive psychosis. First treatment with Azoman. Patient had 1 fit lasting 55 secs. and followed by 3 minutes coma. Latent period between injection and fit was 30 seconds.

	29/9/39				30/9/39.
Date.	11.10 a.m.	11.15 a.m.	11.40 a.m.	2.40 p.m.	11.10 a.m.
Haem^{t}.	46%	1 Fit.	52%	46%	47%
Uric Acid.	3.0		5.1	7.5	5.6
Urea.	24		26	28	34

CASE 148. Non-pregnant female with schizophrenia. Second treatment with Azoman. Patient had 1 fit lasting 36 seconds and followed by 2½ minutes coma. Latent period 6 seconds.

	29/6/39.			30/6/39
Date.	11.20 a.m.	11.25 a.m.	3 p.m.	11 am.
Haem^{t}.	42%	1 Fit.	44%	41%
Uric Acid	3.7		5.3	4.3
Urea.	27		26	35

CASE 149. Non-pregnant female with depressive psychosis. Third treatment with Azoman. Patient had 1 fit lasting 50 seconds followed by 7 minutes coma. Latent period 6 seconds.

	11/7/39.				12/7/39
Date.	10.30 a.m.	10.32 a.m.	11 a.m.	2.30 p.m.	10.30 a.m.
Haem^{t}.	40%	1 Fit.	40%	41%	39%
Uric Acid.	2.7		3.6	4.0	3.3
Urea	13		19	14	16

CASE 150. Non-pregnant female with depressive psychosis. First treatment with Azoman. Patient had 1 fit lasting 86 seconds followed by 4½ minutes coma. Latent period 20 secs.

	3/7/39				
Date.	10.15 a.m.	10.17 a.m.	10.45 a.m.	2 p.m.	4/7/39.
Haem^{t}.	42%	1 Fit.	45%	41.5%	41%
Uric Acid.	3.7		5.0	5.6	3.9
Urea.	39		34	37	33

CASE 151. Non-pregnant female with manic-depressive psychosis. 14th treatment with Azoman. Patient had 1 fit lasting 42 seconds and followed by 9 minutes coma. Latent period, 8 seconds.

	10/7/39.				10.15a.m.
Date.	10.25 a.m.	10.30 a.m.	11 a.m.	2.30 p.m.	11/7/39
Haem^{t}.	43%		43%	43%	45%
Uric Acid.	2.6	1 FIT.	3.7	4.1	3.5
Urea.	18		18	21	25

CASE 152. Non-pregnant female with schizophrenia. First treatment with Azoman. Patient had 1 fit lasting 50 seconds and followed by 3½ minutes coma. Latent period 18 seconds.

	7/7/39.				8/7/39
Date.	11.15/a.m.	11.17 a.m.	11.50 a.m.	3 p.m.	11 a.m.
Haem^{t}.	48%		48.5%	43.5%	45%
Uric Acid.	3.8	1 FIT.	5.0	5.9	4.8
Urea.	19		16	20	28

CASE 153. Non-pregnant female with manic-depressive psychosis. 10th treatment with Azoman. Patient had 1 fit lasting 34 seconds and followed by 8 minutes coma. Latent period 90 secs.

	26/6/39				
Date.	11.20 a.m.	11.25 a.m.	11.50 a.m.	3.30 p.m.	27/6/39
Haem^{t}.	41%		42%	42%	39%
Uric Acid	2.6	1 FIT.	3.4	4.5	3.5
Urea.	16		14	16	20

CASE 154. Male with manic-depressive psychosis. 16th treatment with Azoman. Patient had twitchings for 5 minutes but did not convulse or lose consciousness.

	21/6/39.			
Date.	11.15 a.m.	11.20 a.m.	11.45 a.m.	2.45 p.m.
Haemt.	48%	NO FITS.	48%	45%
Uric Acid.	3.1		2.9	3.2
Urea.	18		14	13

CASE 155. Male with manic-depressive psychosis. 5th treatment. Patient had twitchings for 5 minutes but did not convulse or lose consciousness.

	15/6/39.			
Date.	11.15 a.m.	11.20 a.m.	11.45 a.m.	3 p.m.
Haemt.	50%	NO FITS.	51.5%	48%
Uric Acid.	4.5		4.0	4.3
Urea.	39		33	36

16 HYPEREMESIS CASES

CASE 156. 3rd Para. Admitted to hospital when 8 weeks pregnant. She had had severe vomiting for 5 weeks. Continuous intravenous glucose saline was given for 2 days.

Date.	14/12/38	15/12/38	16/12/38	17/12/38	19/12/38.
Haem^{t}.	52%	48%	42%	37%	32%
Uric Acid.	10.9	9.9	5.6	3.5	2.2
Urea.	191	171	108	71	33
Urine	4 oz.	18oz	24oz	37oz	49oz.
	I.V.glucose saline.				

CASE 157. 2nd Para. Admitted to hospital when 11 weeks pregnant. She had had severe vomiting for 5 weeks. She improved rapidly without intravenous fluid.

Date.	16/3/39	17/3/39	18/3/39	20/3/39	22/3/39.
Haem^{t}.	44%	37%	35%	41.5%	40%
Uric Acid.	7.5	5.8	4.4	2.3	1.8
Urea.	66	58	38	24	23
Bilirubin.	4	2.4	-	-	-
	P112.	88			

No intravenous fluid.

CASE 158. 2nd Para. Admitted to hospital when 10 weeks pregnant. She had had severe vomiting for 6 weeks. Intravenous glucose saline was given twice.

Date.	7/3/39	8/3/39	9/3/39	11/3/39.
Haem^{t}.	40.5%	30.5%	28%	29%
Uric Acid.	6.3	3.1	1.7	1.8
Urea.	64	40	36	22
Bilirubin.	2.5	1.8	<1	

CASE 159. Primigravida. Admitted to hospital when 12 weeks pregnant. She had had severe vomiting for 7 weeks and was slightly jaundiced.

Haematocrit	Uric Acid	Urea	Bilirubin
44%	4.0	12	3.5

CASE 160. Primigravida. Admitted to hospital when 9 weeks pregnant. She had been vomiting for 3 weeks.

Haematocrit	Uric Acid	Urea.
43.5%	2.2	27

CASE 161. 3rd Para. Admitted to hospital when 14 weeks pregnant. She had been vomiting for 6 weeks.

Haematocrit	Uric Acid	Urea
39%	2.7	16

CASE 162. 2nd Para. Admitted to hospital when 16 weeks pregnant. She had been vomiting for 10 weeks and was very dehydrated.

Haematocrit	Uric Acid	Urea
50%	4.8	73

CASE 163. 2nd Para. Admitted to hospital when 10 weeks pregnant. She had had severe vomiting for 4 weeks.

Haematocrit	Uric Acid	Urea
42%	6.9	76

CASE 164. Primigravida. Admitted to hospital when 12 weeks pregnant. She had been vomiting for 4 weeks and the vomiting continued after admission for several weeks although it was never severe.

Date.	15/11/39	23/11/39
Haem^{t}.	34%	37%
Uric Acid.	8.2	8.9
Urea	36	40

CASE 165. 3rd Para. Admitted to hospital when 15 weeks pregnant; She had had severe vomiting for 7 weeks. Pulse rate was 160 and she was slightly jaundiced. She improved rapidly when intravenous fluid was given.

Haematocrit	Uric Acid	Urea	Bilirubin
45%	16.4	136	6

CASE 166. 1st Para. Admitted to hospital when 8 weeks pregnant. She had had some vomiting for 4 weeks.

Haematocrit	Uric Acid
38%	3.3

CASE 167. 2nd Para. Admitted to hospital when 10 weeks pregnant. She had had some vomiting for 4 weeks.

Haematocrit	Uric Acid
40.5%	4.3

CASE 168. FATAL CASE - See TEXT.

CASE 169. FATAL CASE - See TEXT.

CASE 170. FATAL CASE - See TEXT.

CASE 171. FATAL CASE - See TEXT.

4 LATE HYPEREMESIS CASES

CASE 172. 4th Para. Admitted to hospital when 6 months pregnant. She had had vomiting throughout the early months and was very apathetic. She had slight jaundice and marked nystagmus. Pulse rate 150. Continuous intravenous glucose saline was given for 36 hours and there was marked improvement.

	2/12/38.		3/12/38		8p.m.	10a.m.						
Date	11 a.m.	3 p.m.	10 a.m.	7.30 p.m.	4/12/38	5/12/38	6/12/38	7/12/38	8/12/38	9/12/38	10/12/38	13/12/38
Haem^{t}.	46%	43%	38%	36%	38%	37%	36%	34.5%	34%	-	31.5%	32%
Uric Acid.	16.6	17	16.2	13.4	12.0	10.1	7.1	5.7	4.4	3.3	3.6	2.7
Urea.	149	147	159	155	120	110	66	60	30	24	23.3	29
Bili-rubin	12.2			9.6	12.8	10.2	7.0	4.4	2.3		.6	
Urine	1oz.	8oz		29oz	44oz.							
		I.V. 5% glucose in saline.										

Date.	19/12/38
Haem^{t}.	34%
Uric Acid	2.5
Urea.	21

CASE 173. 3rd Para. Admitted to hospital when 6½ months pregnant. She had had vomiting throughout the early months. She had marked nystagmus. Pulse rate 120. Continuous intravenous glucose saline was given for 48 hours and she improved greatly.

Date.	13/1/39	14/1/39	16/1/39	17/1/39	18/1/39 2 p.m.	19/1/39	22/1/39	26/1/39
Haemt.	44%	39%	36.5%	35%	36%	35%	37%	33%
Uric Acid.	14.6	10.2	6.2	6.3	5.2	5.3	4.0	3.3
Urea.	149	68	16	15		18		
Urine.	14oz.	59oz	67oz	35oz				

I.V.glucose saline.

Delivered 18/1/39, 1.30 p.m.

CASE 174. 5th Para. Admitted to hospital when 7½ months pregnant. She had had intermittent vomiting throughout the early months. She had slight jaundice. Pulse rate 120. Continuous intravenous glucose saline given for 36 hours.

Date.	4/8/38	6/8/38	8/8/38	9/8/38	10/8/38.
Haemt.	36%	30.5%	29.5%	27%	25.5%
Uric Acid.	5.9	3.5	2.5	1.8	1.9
Urea.	14.5				
Bilirubin.	5.6	2.4	1.8	.8	

I.V.glucose saline.

Delivered 7/8/38 7.30 p.m.

6 PYELITIS CASES

CASE 175. Primigravida. This patient had severe vomiting and backache from the 5th month of pregnancy. Urine was loaded with pus. She gradually became slightly jaundiced and vomiting continued. Labour was induced at the 7th month because of severity of condition. She became more acutely ill during labour and recovered very slowly.

Date.	1/11/38	2/11/38	5/11/38	9/11/38	10/11/38	11/11/38	12/11/38	14/11/38	15/11/38	16/11/38
Haem^{t}.	31%	27%	25%	29%	29%	30%	31.5%	33%		
Uric Acid.	4.5	6.0	8.4	7.4	6.2	5.3	5.3	4.9		
Urea.	41	58	95	105	98	98	92	66	57.5	45
Bilirubin	6.8	7		1.5						

delivered 4/11/38 at 6 p.m.

Date.	21/11/38
Haem^{t}.	28%
Uric Acid	3.5
Urea.	23

CASE 176. Primigravida. This patient had severe vomiting and lumbar pain from the 5th month of pregnancy. The urinary output was small and the urine was loaded with pus. Her condition became worse after admission. Dye excretion tests for renal function gave poor results. Labour was induced but the patient died soon after.

Date.	16/1/39	22/1/39	24/1/39	25/1/39	10a.m. 26/1/39	7.30 p.m.
Haem^{t}.	30%	30%	26.5%	28%	27%	
Uric Acid.	3.9	8.4	11.4	14.8	13.7	14.1
Urea.	30	59	132	193	211	231

delivered 12 M.D.

CASE 177. Primigravida. This patient had severe vomiting from the 3rd month of pregnancy and was admitted to hospital about the 5th month. She never had any renal symptoms and the urine contained only a moderate amount of pus. Vomiting continued and she had nystagmus and slight jaundice. Hysterotomy was performed but she died 2 days later - see text.

Date.	10/1/39	16/1/39	18/1/39	P.M. 19/1/39
Haem^{t}.	31%	31%	39.5%	-
Uric Acid.	3.2	3.3	5.7	12.1
Urea.	15	30	54	138
Bilirubin.	3	3.5	3	3.5

hysterotomy.

CASE 178. Primigravida. Admitted to hospital at term with severe vomiting but no pyuria. Delivered spontaneously after 5 hours labour. Next day she passed urine loaded with pus and her vomiting ceased.

	7/12/38							
Date.	10 a.m.	2.30 p.m.	4.45 p.m.	10 p.m.	8/12/38	9/12/38	10/12/38	15/12/38.
Haem^{t}.	27.5%	28%	32%	-	29%	27.5%	27%	26%
Uric Acid	8.6	9.4	9.1	8.0	6.9	4.2	3.7	2.5
Urea.	30	33	34		22	19	19	-
Bilirubin.	4.4		4.4		3.2	1.8	0.8	

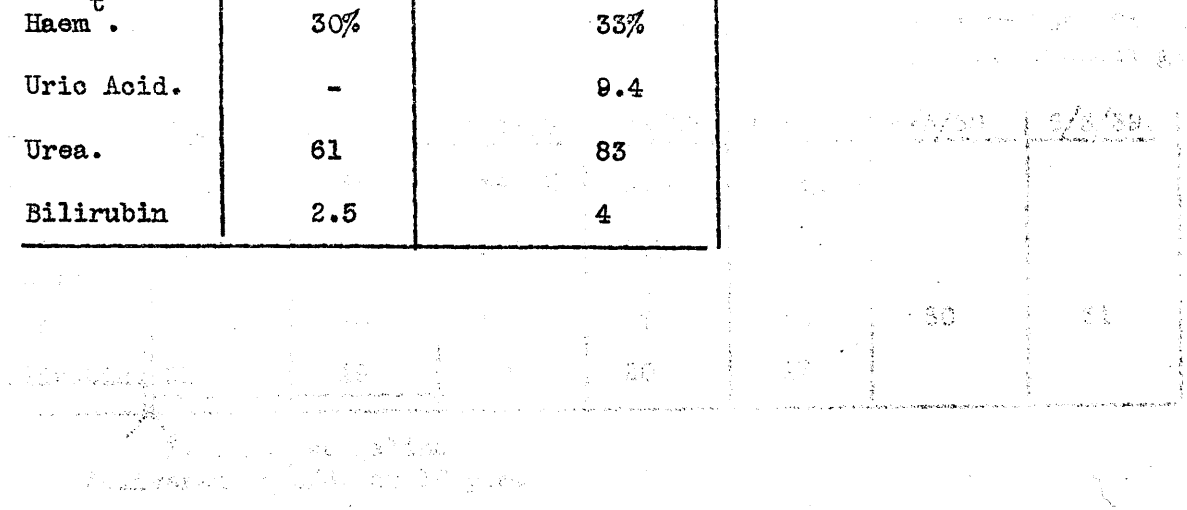
Delivered 4.45 p.m.

CASE 179. Primigravida. This patient had severe vomiting during the 6th and 7th months of her pregnancy and had marked pyuria. Her temperature was irregular for several weeks. She was under observation during the 7th, 8th and 9th months of her pregnancy.

Date.	11/1/39	13/1/39	16/1/39	20/3/39.
Haem^{t}.	29.5%	30%	28.5%	29.5%
Uric Acid.	3.4	3.7	3.2	4.1
Urea.	17	16	-	17

CASE 180. Primigravida. Admitted in 7th month of pregnancy. She had had abdominal pain and sickness for many weeks and her temperature was raised. She was delivered prematurely and became very ill after delivery and died 2 days post-partum - see text.

Date.	12/10/39	17/10/39.
Haem^{t}.	30%	33%
Uric Acid.	-	9.4
Urea.	61	83
Bilirubin	2.5	4



4 "ERYTHROBLASTOSIS FOETALIS" CASES.

CASE 181. 5th Para. Admitted to hospital when 7 months pregnant. B.P. $\frac{160}{100}$. She had had severe vomiting for 7 days. Delivered after 9 hours labour - see Text.

Date.	1/7/38 (11 a.m)	2/7/38	4/7/38	5/7/38	7/7/38	9/7/38.
Haemt.	37.5%	29.5%	26.5%	27%	25.5%	28%
Uric Acid.	11.2	12.0	7.1	5.9	4.6	4.2
Urea.	73.3		41.3	20.5		
Bilirubin.	2.9	2.4	1			

delivered 12.55 p.m.

CASE 182. Primigravida. Patient had severe vomiting and some diarrhoea for a week before admission. She became jaundiced after delivery. Continuous intravenous glucose saline was given for several days. Improvement was gradual.

Date.	27/2/39	28/2/39	1/3/39	2/3/39	3/3/39	4/3/39	6/3/39.
Haemt.	33%	36%	34.5%	34%	33%		
Uric Acid.	7.7	5.2	3.3	3.3	3.1		
Urea.	97	70	60	77	82	60	31
Bilirubin.	22	19	20	20	17		

I.V.glucose saline delivered 26/3/39 at 10 p.m.

CASE 183. 11th Para. Patient had severe vomiting when 6½ months pregnant. She became slightly jaundiced. Labour was induced. Intravenous glucose was given for 24 hours after delivery.

Date.	26/1/38	31/1/38	1/2/38	2/2/38	4/2/38	8/2/38.
Haem^{t}.	33%	36.5%	34%	34.5%	29%	28.5%
Hb.	71.5	79	70	71	59.5	58
Protein.	6.4	6.5	5.8	5.4	4.9	5.2
Uric Acid.	9.9	7.1	6.5	5.4	4.5	3.3
Bilirubin.	4.7	6			0	
Urea.	22.3					

Delivered 31/1/38 at 6 a.m.

CASE 184. 4th Para. Patient had severe vomiting when 7½ months pregnant. She became slightly jaundiced. Labour was induced and continuous intravenous glucose was given for 3 days after delivery.

Date.	11.30a.m. 27/12/37	4 p.m. 27/12/37	28/12/37	29/12/38	31/12/38
Haem^{t}.	43%	44%	37%	33.5%	27%
Uric Acid.	13.3	13.0	11.4	7.1	3.8
Urea.			97	73	30
Protein.	6.2	6.3	6.1	5.9	5.2

Delivered 1 p.m. Continuous I.V. glucose saline.

CASE 185. - See text for clinical and biochemical findings.

CASE 186. - See text for clinical and biochemical findings.

Delayed Chloroform Poisoning Cases.

CASE 187. - See text for clinical and pathological findings.

CASE 188. - Clinical description given in text.

Date.	18/10/38	19/10/38	20/10/38	21/10/38	24/10/38	8/11/38.
Haem^{t}.	41%	41%	34.5%	31%	30%	32%
Uric Acid.	8.8	7.7	4.8	4.0	3.7	5.1
Urea.	70	76	69	22		19
Bilirubin.	28	32	23	20	6.0	1.8

restless comatose

Continuous I.V. glucose saline.

Delivered 14/10/38.

CASE 189. Clinical description given in text.

Date.	2/10/40	3/10/40. 10 a.m.	3 p.m.	4/10/40	5/10/40	7/10/40	10/10/40.
Haem^{t}.	35%	38%	35.5%	36%	34%	32%	29%
Uric Acid.	10.4	7.8	5.8	6.4	6.0	5.0	3.5
Urea.	43	41	34	39	39	29	15
W.B.Cl.	533	580	621	551	560	-	-
Bilirubin.	16	24	21	16 .6	16	10	3.5

restless — comatose — conscious

continuous I.V.glucose saline

delivered 29/9/40.

CASE 190. Clinical description given in text.

Date.	2p.m. 27/6/39	28/6/39	29/6/39	30/6/39	3/7/39.
Haem^{t}.	32%	34%	39.5%	37%	32%
Uric Acid.	6.6	4.8	3.3	-	2.9
Urea.	31	45	30	-	-
Bilirubin.	18	26	24	21	14

Delivered 25/6/39.

I.V.glucose given.

CASE 191. Clinical description given in text.

Date.	2/10/40.	3/10/40.	4/10/40.
$Haem^t$.	37%	34%	38%
Uric Acid.	9.7	8.4	3.8
Urea.	30	17	19
Bilirubin.	8	7.7	2
	jaundiced	improved.	

delivered 29/9/40.

I.V.glucose saline.

26 OBSTETRIC SHOCK CASES

CASE 192. 3rd Para. Delayed labour. Labour 34 hours. Intravenous glucose given during labour and at delivery. Delivered by forceps with no undue haemorrhage.

Date.	7/3/40. 10 a.m.	1.45 p.m.	3.45 p.m.	8/3/40. 10 a.m.
Haem^{t}.	42.5%	42%	42%	35%
Blood Pressure.	110/90	130/80	110/75	120/80
Pulse Rate.	120	120	100	80
Sodium.	319	324	323	319
W.B. Cl.	481	540	595	673

300 cc I.V. 20% glucose at 1 a.m.

I.V. glucose 12.15 p.m.

delivered 12.40 p.m.

CASE 193. 2nd Para. Delayed labour. Labour 72 hours. Delivered by forceps with no undue haemorrhage.

Date.	1/4/40. 10.15 a.m.	10.50 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	2/4/40. 10 a.m.
Haem^{t}.	39%	41.5%	40%	39.5%	37%	30%
Blood Pressure.	130/110	120/95	120/95	123/95	110/95	120/70
Pulse Rate.	120	120	100	100	88	88
Sodium.	338	336	332	348	352	332
W.B. Cl.	533	542	475	510	542	586
Urea.	52	79	48	54		58

Delivered 10.45 a.m.

CASE 194. Primigravida. Contracted pelvis. Labour about 96 hours but never severe. Delivered by forceps with no undue haemorrhage.

Date.	2/5/40. 10 a.m.	2 p.m.	3 p.m.	5 p.m.	3/5/40 10 a.m.
Haem^{t}.	40%	39.5%	40%	40%	35%
Blood Pressure.	130/80	85/65	115/85	115/80	120/90
Pulse Rate.	96	100	92	88	72
Sodium	344	335	336	339	344
W.B.Cl.	540	516	540	605	565
Urea.	46	53			41

delivered 1.10 p.m.

CASE 195. Primigravida. A mild pre-eclamptic case. Labour 48 hours. Delivered by forceps with no undue haemorrhage.

Date.	6/5/40. 10 a.m.	4.55 p.m.	8 p.m.	7/5/40. 10 a.m.
Haem^{t}.	37%	36%	33%	28%
Blood Pressure.	145/80	150/105	140/90	125/80
Pulse Rate.	96	100	80	72
Sodium.	327	323	341	336
W.B. Cl.	445	618	586	605
Urea.	26		35	31

delivered 4.25 p.m.

CASE 196. Primigravida. A severe pre-eclamptic case. Labour 30 hours. Spontaneous delivery.

Date.	29/5/40. 10 a.m.	11.30 p.m.	12.30 p.m.	2.30 p.m.	30/5/40. 10 a.m.
Haem^{t}.	41%	40%	39%	39%	35%
Blood Pressure.	185/115	165/110	150/110	140/90	125/100
Pulse Rate.	120	108	108	100	88
Sodium.	302	317	323	323	337
W.B. Cl.	560	580	551	560	568

delivered 10.30 a.m.

CASE 197. Primigravida. A severe pre-eclamptic case. Labour 24 hours. Spontaneous delivery. Placenta retained for 1 hour and then expressed. Moderate P.P.H.

Date.	30/5/40. 10 a.m.	11.45 a.m.	12.55 p.m.	2.45 p.m.	4.30 p.m.	31/5/40. 10 a.m.
Haem^{t}.	32.5%	29%	28%	26%	25%	20%
Blood Pressure	180/130	175/125	-	170/120	160/120	145/100
Pulse Rate.	140	140	140	120	120	140
Sodium.	339	331	339	344	335	324
W.B. Cl.	606	612	638	621	647	664

delivered 11.15 a.m.

CASE 198. Primigravida. A severe pre-eclamptic case. Labour 30 hours. Delivered by forceps with no undue haemorrhage.

Date.	11/6/40. 10.25 a.m.	10.45 a.m.	11.30 a.m.	12.30 p.m.	2.30 p.m.	12/6/40. 10 a.m.
Haemt.	42%	42%	39%	38%	41%	35%
Blood Pressure.	165/120	-	135/80	135/80	150/100	140/100
Pulse Rate.	80	80	88	80	80	80
Sodium.	315	320	325	319	319	320
W.B. Cl.	610	615	597	597	600	630
Urea.	32		39		38	38

delivered 11 a.m.

CASE 199. Primigravida. A mild pre-eclamptic case. Labour 18 hours. Spontaneous delivery.

Date.	26/3/40. 10.22 a.m.	11.20 a.m.	12.20 p.m.	2.20 p.m.	27/3/40	28/3/40.
Haemt.	40%	38%	37%	35%	32.5%	32%
Blood Pressure.	155/90	160/95	150/90	150/90	130/80	-
Pulse Rate.	110	110	100	88	72	-
Sodium.	334	322	334	339		350
W.B. Cl.	586	568	586	603		628
Urea.	24			38		

Delivered 10.20 a.m.

CASE 200. Primigravida. A severe pre-eclamptic case. B.P. $\frac{252}{160}$. Gross albuminuria and oedema. Labour 12 hours. Spontaneous delivery.

Date.	26/2/40	27/2/40	28/2/40. 10 a.m.	28/2/40. 4.30 p.m.	28/2/40. 7 p.m.	29/2/40	1/3/40	5/3/40.
Haem^{t}.	42%	41%	45%	44%	47%	41%	39%	34%
Sodium.	339	329	318	311	324	337	340	356
Plasma Cl.	615	625	586	638	618	603	603	
W.B. Cl.	489	498	454	670	476	489	533	506
Urea.	53	58	39		69	50		26
Uric Acid.	8.6	8.6						

delivered 6 p.m.

CASE 201. Primigravida. A severe pre-eclamptic case. 24 hours labour. Placenta retained for 2 hours and then delivered with no haemorrhage.

Date.	11/7/40. 10 a.m.	11/7/40. 3 p.m.	11/7/40. 4 p.m.	11/7/40. 5 p.m.	12/7/40. 10 a.m.
Haem^{t}.	31%	32%	35%	35%	30%
Blood Pressure	$\frac{165}{160}$	$\frac{160}{90}$	$\frac{170}{95}$	$\frac{160}{90}$	$\frac{140}{90}$
Pulse rate.	120	120	100	88	96
Sodium.	320	322	324	334	324
W.B. Cl.	568	590	-	587	621
Urea.	19		20		

delivered 1.55 p.m.

CASE 202. Primigravida. Normal. Labour 13 hours. Delivered by forceps with no undue haemorrhage.

Date.	11/7/40. 10 a.m.	11.45 a.m.	12.30 p.m.	2.30 p.m.	3.30 p.m.	12/7/40. 10 a.m.
Haem^{t}.	31%	31%	31%	31%	30%	28%
Blood Pressure	115/70	-	125/70	115/60	115/60	110/60
Pulse Rate.	88	100	80	80	76	80
Sodium.	316	320	322	323	328	337
W.B.Cl.	516	568	545	603	594	621
Urea.	23			23		

delivered 11.30 a.m.

CASE 203. Primigravida. Normal. Labour 18 hours. Spontaneous delivery.

Date.	1/2/40. 10.30 a.m.	2.15 p.m.	2/2/40. 10 a.m.
Haem^{t}.	38.5%	35%	32%
Sodium.	328	324	320
W.B. Cl.	656	832	664

delivered 12.35 p.m.

CASE 204. 2nd Para. The patient had a slight ante partum haemorrhage but was delivered spontaneously after a 20 hour labour. Half an hour after delivery she became rather collapsed and vomited a little. She recovered without any active treatment.

	29/1/40.				30/1/40
Date.	11.20 a.m.	12.15 p.m.	2.15 p.m.	4.15 p.m.	10 a.m.
Haem^{t}.	43.5%	42%	42%	40%	36%
Sodium.	301	266	311	320	320
Pl.Chloride.	603	612	612	630	630

delivered 11.15 a.m.

CASE 205. Primigravida. Patient exhausted in second stage of labour. Labour 44 hours. Delivered by forceps with slight P.P.H. She was collapsed for 2 to 3 hours after the delivery.

	19/1/40.				20/1/40.
Date.	10.10 a.m.	12.40 p.m.	2.40 p.m.	4.40 p.m.	10 a.m.
Haem^{t}.	41.5%	42%	42%	40%	32%
Blood Pressure	145/75	105/70	90/55	128/70	128/70
Pulse Rate.	120	132	120	120	100
Pl. Sodium.	309	272	342	332	334
Pl. Nall.	603	621	611	605	621
W.B. Nall.	428	480	445	516	498
Urea.	26			21	

delivered 11.40 a.m.

CASE 206. Primigravida. Patient very exhausted during labour and had considerable sickness. Labour 35 hours. Manual breech delivery with no undue haemorrhage. Patient moderately collapsed for several hours after delivery.

Date.	22/1/40	23/1/40.			24/1/40.
	12.30 p.m.	10 a.m.	12 M.D.	4.30 p.m.	12 M.D.
Haemt.	33%	33.5%	35%	32%	27%
Blood Pres.		145/90	135/75 90/60	105/70	110/60
Pulse Rate.	112	136	140	120	96
Sodium.	317	317	275	311	343
W.B.Cl.	510	490	516	551	542
Plas.Cl.	691	621	621	621	612
Urea.	24	17	17		
	I.V. 20% glucose 11 a.m.		I.V.glucose 11 a.m. delivered 11.30 a.m.		

CASE 207. Primigravida. Labour 30 hours. Manual breech delivery with no undue haemorrhage. Patient slightly collapsed for 2 to 3 hours post-partum.

Date.	29/1/40.				31/1/40.
	11.20 a.m.	12.15 p.m.	2.15 p.m.	4.15 p.m.	12 M.D.
Haemt.	41%	40%	40%	39%	36%
Blood Pressure.	140/90	105/65	115/60	115/60	120/70
Pulse Rate.	96	120	120	100	72
Sodium.	332	297	333	337	346
Plasma Cl.	595	595	612	709	618
W.B. Cl.	540		551	575	560
	delivered 11.30 a.m.		600 cc I.V.glucose 10% saline.		

CASE 208. Primigravida. Labour 72 hours. Patient was very exhausted during labour. Delivered by forceps with no undue haemorrhage. She was very dazed for several hours after delivery and vomited several times. Placenta was retained for 4 hours. After the delivery of the placenta she gradually recovered.

Date.	5/2/40. 10 a.m.	6/2/40. 10 a.m.	6/2/40. 1.45 p.m.	6/2/40. 3.30 p.m.	7/2/40. 10.30 a.m.
Haem^{t}.	42%	43%	41%	-	35%
Blood Pressure.	130/60	130/60	105/45	98/65	110/70
Pulse Rate.	84	120	152	140	88
Sodium.	330	337	309	287	335
Plasma Cl.	647		673		621
W.B. Cl.	524		489		586
Urea.	28		50		

delivered 1.15 p.m.

placenta delivered 5.30 p.m.

CASE 209. Primigravida. Admitted as a severe pre-eclamptic case. Labour 72 hours. Delivered by forceps with moderate P.P.H. She appeared to be somewhat collapsed for 1 hour after delivery.

Date.	4/3/40	5/3/40	6/3/40. 10.40 a.m.	6/3/40. 11.35 a.m.	6/3/40. 2.15 p.m.	7/3/40. 11 a.m.
Haem^{t}.	36%	33%	37%	31%	28%	23%
Blood Pressure.	160/120	160/100	185/120	115/100	150/115	140/100
Pulse Rate.	80	84	80	140	140	100
Sodium.	319	342	314	305	314	324
W.B. Cl.	498	516	498		550	575
Urea.	39		30		35	

delivered 11.5 a.m.

CASE 210. Primigravida. Admitted as a mild pre-eclamptic case. Labour 48 hours. Delivered by forceps with no undue haemorrhage. She was sick and collapsed for about 2 hours after delivery.

Date.	11/3/40. 10.45 a.m.	11.45 a.m.	12.45 p.m.	2.15 p.m.	4.15 p.m.	12/3/40. 10 a.m.
Haem^{t}.	40%	39%	37%	38%	34%	31%
Blood Pressure	140/110	145/100	110/80	120/90	120/90	135/90
Pulse Rate.	112	120	140	120	96	80
Sodium	326	311	303	331	332	332
W.B. Cl.	516	489	481	489	568	603
Urea.	16		28		15	13

delivered 11.15 a.m.

CASE 211. Primigravida. Admitted as a pre-eclamptic case. Labour 36 hours. Delivered of twins - both spontaneously. She became rather collapsed after the second delivery.

Date.	7/5/40. 10.30 a.m.	11.15 a.m.	1 p.m.	3.30 p.m.	5 p.m.	7 p.m.	8/5/40. 10 a.m.
Haem^{t}.	49%	50%	50%	47%	48%	45%	35%
Blood Pressure.	160/100	145/90	160/100	150/120	125/115	-	115/80
Pulse Rate.	120	120	120	120	120	112	80
Sodium.	309	343	334	335	295	326	326
W.B. Cl.	460	533	551	556		488	550
Urea.	39		48				

1st Child delivered 10.45 a.m.

2nd Child delivered 2.55 p.m.

CASE 212. Clinical description given in text.

Date.	2/2/40. 10.30 a.m.	11.45 a.m.	P.M. 2 p.m.
Haem^{t}.	27%	35%	
Sodium.	300	302	309
Plasma Cl.	625	638	670
Urea.	19		

delivered 7.40 a.m. transfusion 800cc blood. died 1.30 p.m.

CASE 213. Clinical description given in text and blood pressure readings recorded in Chart XXIX.

Date .	12/7/40. 1.45 p.m.	4.30 p.m.	13/7/40	16/7/40.
Haem^{t}.		25%	19.5%	19.5%
Sodium	296	305	321	308
W.B. Cl.			628	603
Urea.	18	35	29	29

delivered 10 a.m. 2.30 p.m. 8cc.Eschatin.
3 p.m. placenta removed.

CASE 214. Clinical description given in text.

Date.	5/2/40. 10 a.m.	5/2/40. 4 p.m.	6/2/40. 2.30 p.m.
Haem^{t}.	34%	37%	28%
Sodium	296	317	331
Plasma Cl.	638	640	
Uric Acid.	10.4		
Urea.	37	47	78

delivered 11.55 p.m.

CASE 215. Clinical description given in text and blood pressure readings recorded in Chart XXIX.

Date.	17/7/40	18/7/40. 10 a.m.	18/7/40. 11.25 a.m.	18/7/40. 10 a.m.
Haem^{t}.	41.5%	39%	40%	26%
Sodium.	304	293	293	308
W.B. Cl.	498	551	568	-
Urea.	12	14	12	-

delivered 10.25 a.m.

CASE 216. Clinical description given in text and blood pressure readings recorded in Chart XXIX.

	25/4/40.								
Date.	10 a.m.	1 p.m.	3 p.m.	3.30 p.m.	4.40 p.m.	6.30 p.m.	26/4/40	27/4/40	29/4/40.
Haem^{t}.	42%	40%	40%	40%	38.5%	39%	31%	29%	30%
Sodium.	324	319	323	322	316	309	319	326	321
W.B. Cl.	498	586	507	660	-	-	-	550	533
Urea.	34	36		36	42	43		34	25

delivered 2.30 p.m.

CASE 217. Clinical description given in text.

	12/2/40.					13/2/40.		14/2/40.		
Date.	10.15 a.m.	12.30 p.m.	1.30 p.m.	2.45 p.m.	4.45 p.m.	10 a.m.	4.30 p.m.	10 a.m.	4.30 p.m.	P.M.
Haemt.	43%	42%	33%	38%	37%	34%	33%	36%	34%	-
Sodium.	340	315	303	289	309	293	296	306		332
Plasma Cl.	664	625	590	603	615	598				
W.B.Cl.	507	498	463	498	498	540	551	516		
Urea.	45	51	49	56	53	-	110	144		199

delivered 1 p.m.

placenta expressed 9.30 p.m.

CASE 218. Clinical description given in text and blood pressure readings recorded in Chart XXIX.