Utero-Placental (Accidental) Hæmorrhage.

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PROLOGUE.

Since the commencement of July 1915 I have treated, or seen treated, and followed up, fifty cases of so-called accidental hæmorrhage and retroplacental hæmatoma.

Three of these cases have died, and have provided me with pathological material which I have carefully compared with other pathological material removed from women dying during labour from conditions other than accidental hæmorrhage.

This clinical and pathological material has stimulated me to attempt to place this condition on a satisfactory basis. The paper which has resulted consists of two parts—Part I, Clinical; Part II, Pathological.

PART I.

A CLINICAL REPORT OF FIFTY CONSECUTIVE CASES.

The ætiology of this condition is of considerable interest, and will be dealt with first. The average age-incidence in the series of fifty cases was 31.6 years, the youngest case being 19 and the eldest 43 years.

The parity incidence was as follows: —Nine (18 per cent.) were primigravidæ; 41 (82 per cent.) multiparæ. The average parity of the multiparæ was 5.7 pregnancies, with a maximum parity of 15 pregnancies. The abortion incidence was 1 abortion to 9 labours at term, as against a normal abortion incidence of 1 to 4 or 5 labours at term.

With regard to the causative factors, it will, I think, be best to take up separately the causes given by authors and eliminate them, when possible, singly. *Traumatism*, on careful questioning, played no part in any one of the cases. Neither did it in any of the 17 cases recorded by Whitridge Williams (1915).[35] It was thought to be the cause in a small percentage of Essen-Möller's (1913)[12] 29 cases, and in 5 of Gaston's (1906)[15] 70 cases. Frequently the patient attributed the hæmorrhage to an accident,

but the nature of the accident and the interval between the accident and the onset of symptoms always excluded it as the causative factor.

Ruplure of the sinus circularis of the placenta is thought to be an occasional causative factor by Essen-Möller.[12]

Fright, morbus cordis, fibroid tumours and diseases of the blood played no part in any of the cases.

Shortness of the umbilical cord was not the causative factor in any one of the 50 cases. The average length of the umbilical cord in 32 cases in which it was measured was 21.3 in.; the longest being 34 in.; the shortest 13 in. In one case the cord is mentioned as having been wound three times round the child, but there was no tension on the cord, this child being born alive. Shortness of the cord was given as the cause in 12 of the 70 cases recorded by Gaston.[15] It appears to me impossible for shortness of the cord to cause separation of the placenta unless there is traction on the fœtus. Obviously the resistance by soft parts is such that the fœtus not only ceases to travel forwards at the time the uterine contraction ceases, but is actually forced backwards by the recoil of the muscular pelvic floor. During the pain the uterus with the placenta is travelling in the same direction and at the same pace as the fœtus is travelling. Therefore there can be no pull on the cord either during or at the termination of a pain, while the child remains within the genital tract.

Endometritis is, of course, an impossible ætiological factor to exclude, but if, as is generally stated, endometritis is a cause of abortion, the low abortion incidence would appear to negative this theory.

Syphilis. The Wassermann reaction was positive in only one of 15 cases in which it was applied. It will therefore be seen that syphilis played no part at any rate in these cases. It is mentioned as playing a part in one af Gaston's [15] 70 cases.

One condition, and one condition only, was pre-eminently associated with accidental hæmorrhage, *i.e.*, *albuminuria.* This was present in 42 (84 per cent.) of the 50 cases. The amount varied from a fine cloud to 15 parts per 1,000 (Esbach). It was usually, however, in considerable quantity. Hyaline and granular casts were present in 13 (26 per cent.) of the cases. Chronic nephritis was clinically present in only 3 (6 per cent.) of the cases. In the others, the albuminuria was apparently of toxæmic origin, *i.e.*, it cleared up after delivery, the average duration of albuminuria, excluding the chronic renal cases being 11.3 days. Toxæmic symptoms, *i.e.*, œdema, headache, vomiting, etc., were associated in 17 (34 per cent.) of the cases, eclampsia with one. This association of albuminuria cannot be looked on as an accidental

occurrence, and in the absence of other caustive factors, one naturally looks to the cause of the albuminuria as the cause of the hæmorrhage. The association of a toxæmic condition in a higher percentage of multiparæ than primigravidæ is, I know, without precedent, and I have no satisfactory explanation of this phenomenon. Winter, in 1885,[36] first directed attention to the presence of albuminuria in these cases, since which time nephritis or albuminuria has been regarded as the most constantly associated factor. It was associated with the condition in 64.7 per cent. of 17 cases (nephritis once) recorded by Whitridge Williams[35]; in 37 per cent. of 29 cases, Essen-Möller[12]; in 34.25 per cent. of cases recorded by Aschkanazy (1902)[1]; in 50 per cent. of cases, Muus (1903)[26]; in 42.86 per cent. of 70 cases, Gaston[15]; in 36.9 per cent. of 58 cases, Bar and Kervily (1906)[3] in 51.9 per cent. of 158 cases, Dorman (1913)[10]; in 50.75 per cent. of 67 cases, Hartmann (1907).[17]

The frequency of the condition in association with eclampsia has also been noticed, cases being recorded by Bar and Kervily,[3] Couvelaire (1912),[8] Essen-Möller,[12] Zarate (1915),[38] Seotz (1903)[30] and Young (1914)[37] amongst others. These figures, although in none of the records is the percentage as high as in my series, still further emphasize the association between albuminuria of toxæmic origin and utero-placental hæmorrhage.

The examination of the pathological material which I have obtained shows areas of necrosis with hæmorrhage in the uterine wall, and I formulate the following theory for the hæmorrhage: (1) Focal necrosis with hæmorrhage, in the musculature and decidua of the uterine wall; (2) resulting separation of an area of placenta opening the mouths of uterine sinuses; (3) extensive retro-placental hæmorrhage.

The onset of the hæmorrhage is usually towards the end of pregnancy. In this series there were between the twenty-third and twenty-eighth week, 3 cases; between the twenty-ninth and thirty-second week, 8 cases; between the thirty-third and thirty-sixth week, 11 cases; between the thirty-seventh and fortieth week, 24 cases.

The cases can be divided clinically into three groups: (*a*) Those in which the blood entirely escapes—*external cases;* (*b*) those in which the blood is entirely retained *in utero—internal cases;* (*c*) those in which there is free escape of blood with retention of a considerable amount *in utero—combined cases.*

Group (*a*): *External Cases.* These cases are infrequent. Among the series of fifty cases there were only seven belonging to this group. The hæmorrhage in these is never of great severity and is usually slight. The only symptom is bleeding, which comes on without cause. Repeated losses, as met with in placenta

prævia, do not occur. In the absence of labour pains there is no pain, but the bleeding is likely to bring on labour. On abdominal examination nothing abnormal is, as a rule, revealed, *i.e.*, the abdominal findings of placenta prævia are absent. The fœtal heart cannot be heard in about half the cases; its absence will assist the diagnosis, since in placenta prævia the child is usually alive at this stage. On vaginal examination nothing abnormal is revealed. If the cervix will admit the finger the placenta will not be felt. The presence of albumin in the urine will go far to confirm the diagnosis, but in the slight cases it is often absent. It was present in only two of the cases in this series. The diagnosis between this type of case and a lateral placenta prævia can usually only be made after the expulsion of the membranes and placenta, and then only when the bag of membranes is complete.

The Treatment. If the loss has been slight and has ceased, and if the child is alive, rest with a hypodermic injection of morphia will often result in the pregnancy continuing to term. The patient should be dieted and the bowels made to act two or three times daily for the remainder of her pregnancy with a view to elimination of toxin. If the loss is considerable, labour pains will always be present. The membranes should then be ruptured, a tight binder applied and a hypodermic injection of 1 cc. of pituitrin administered. If it is essential that no further loss should ensue, that is, if the condition of the patient is really serious, the vagina should be plugged with gauze in addition. With regard to plugging, I have had no case in which puerperal sepsis has followed its use. It has always been a routine that the vagina should be dried and swabbed out with iodine before plugging with dry sterile gauze. I feel, however, after my examination of pathological material that with plugging there is a risk of spontaneous rupture of the uterus through a necrosed area. This rupture actually happened in one case of combined hæmorrhage (*vide* Case 27) in which plugging was used.

Results. If treatment is carried out along these lines bleeding will cease, labour will progress rapidly, and delivery will be spontaneous, the placenta and membranes as a rule following the baby at once. In this series of cases the results to the mothers have been uniformly good; the progress of labour having been satisfactory from the onset of treatment. Half the children were stillborn, but the life of the child is not under the control of the medical man, that being dependent entirely on the initial separation of the placenta.

(*b*) *Internal Cases.* These cases have been sub-divided into (1) clinical cases of internal hæmorrhage; (2) retroplacental hæmatomata.

(1) Cases of this class are also infrequent, occurring 11 times among the series of fifty cases. The hæmorrhage in these cases is always considerable, and this is undoubtedly the reason why the blood does not appear externally. The explanations for its non-appearance hitherto given are: (1) that the cervix is not dilated; (2) that the membranes are adherent around the internal os; (3) that the presenting part obstructs the canal; (4) that the blood ruptures into the amniotic sac. These are all unsatisfactory. With regard to the first of these explanations, it is well known that in threatened abortion there is bleeding in the absence of dilatation. Why not in this condition? To give as a definition of threatened abortion bleeding without dilatation of the cervix, and to give as a reason for internal hæmorrhage the absence of dilatation of the cervix, is a paradox which should have no place in obstetrical literature. As a point of fact, dilatation up to two fingers is often present in internal cases.

With regard to the second explanation—the adhesion of membranes—one has only to watch the delivery of a case of internal hæmorrhage to see that this plays no part. The placenta and membranes follow directly on the baby, and I have seen no case of combined or internal hæmorrhage in which this sequence of events did not occur. With regard to the third explanation; in the absence of rupture of the membranes it is obviously impossible for the presenting part to block the cervical canal. With regard to the fourth explanation, it appears to me to be mechanically impossible for the blood to rupture into the amniotic sac. For it to do so a sharp salient of membranes must project into the amniotic sac. The tension in the amniotic sac, normally not inconsiderable, will be raised at once by retroplacental hæmorrhage, and as fluid under pressure resists pressure equally in all directions, the formation of this salient will be rendered impossible.

The reason why the blood is retained is that the uterus is paralyzed. One assumes a sudden considerable hæmorrhage which forcibly distends the uterus which, thus taken unawares, becomes over-distended with resulting paralysis. The degree of paralysis varies from one in which, although the power of contraction is lost, the power of resisting further distension is retained—*i.e.*, muscular tone is present; to one in which all power is lost, the uterus becoming distended to the maximum stretching point of muscle; even this is occasionally overcome, the case then terminating in uterine rupture (*vide* Cases 4, 27 and 33).

Symptoms. The onset is sudden, with a severe or intensely severe pain in the abdomen. This is referred to by the patient as "indescribably bad," as "a tight band round the waist," or as a "bursting pain." The pain persists and there is no alleviation.

The patient's mental state is often so affected by its severity that she is incapable of giving a coherent history, but rolls about the bed complaining constantly of the agonizing pain. It was a marked symptom in 54 per cent. of the cases; but may be completely or almost completely absent. Vomiting is frequently present. It was present in 45 per cent. of the cases. It may be slight or persistent. The recognition of pallor by the patient or by her friends is a common symptom, and is helpful to the clinician. Fainting attacks, sometimes described as fits, are not infrequent. They were present in 45 per cent. of the cases.

An examination of the patient reveals anæmia, usually extreme. The face is blanched, the face and hands cold and clammy. The pulse varies from a normal good pulse to a running feeble pulse of 120 or more. The temperature is as a rule subnormal, the respiration rate is usually raised. The abdominal examination is characteristic, so much so that once palpated it is, I believe, difficult to fail to recognize a future case. The uterus is large; it may be definitely larger than it should be for the date of pregnancy. It is tense; this hardness is characteristic and resembles no other uterine condition. It is frequently, although not by any means always, tender; the tenderness may be localized to a definite area of the uterus. The fœtal parts cannot be recognized on palpation as a result of the hardness of the uterus. The fœtal heart cannot be heard. There are no uterine contractions. On vaginal examination the cervix may admit one or more fingers or may be undilated. The bag of membranes, if felt, is tense all the time, but not bulging. The urine will be found to contain albumin, which was present in every case in this series. The amount was never less than a cloud, and in only four cases less than four parts per 1,000 (Esbach). Granular and hyaline casts were frequently present.

Treatment. Attempts to stimulate the uterus are not only useless, but also tend to increase shock and should therefore not be encouraged. Attempts at delivery per vaginam will as a rule be fatal, and should play no part in the treatment of this condition. Rupture of the membranes is usually fatal, as it relieves the one thing that is keeping the bleeding in check, *i.e.*, the intra-uterine pressure, which has become greater than the patient's blood pressure. The treatment that I have adopted is to employ rest, and to watch carefully (1) the size of the uterus, which should be outlined with pencil; (2) the pulse; (3) the patient's general condition. The patient is put in bed and kept warm. If her pain is intensely severe she is given a hypodermic injection of morphia, $\frac{1}{4}$ grain. The fact that morphia masks symptoms must of course be taken into consideration.

If her condition improves she should be left, the pulse being taken and charted hourly, and the height of the fundus carefully noted. After a few hours' rest, the patient will recover from her shock, the uterus will recover from its paralysis, and contractions will start (vide Cases 35 and 43). The onset of contractions produces external loss, which should be looked on as a favourable sign, indicative of expulsive pains. The blood at first expelled is often watery, *i.e.*, serum stained with blood. This, I believe, is the origin of the theory that the blood has ruptured into the amniotic sac; the nurse in attendance has on more than one occasion informed me that the patient's membranes have ruptured, and that "bloody liquor" was coming away; examination has shown, however, an intact bag of membranes. When contractions are definitely present and there is external loss, the case can be treated as one of severe external hæmorrhage, *i.e.*, the membranes can be ruptured, the vagina plugged, a binder and perineal pad applied, and 1 c.c. of pituitrin administered hypodermically, or the case can still be left to nature. The former was done in one case with good result, the latter in five cases with excellent results.[1] The child will be expelled, stillborn, and will be at once followed by the flattened placenta, membranes and one or more pounds of clot.

If the patient's condition becomes worse, *i.e.*, if the pulse rises, the uterus gets larger, or her general condition becomes worse, the case can only be treated by laparotomy. Cæsarean section should be performed, and after removal of the fœtus, placenta, membranes and clot, vigorous attempts should be made to make the uterus contract (vide Case 33). This is usually possible with powerful hand and hot towel stimulation. If satisfactory contractions are produced, the uterus should be sutured. If it remains limp and flaccid, in spite of stimulation, it must be removed (vide Cases 37 and 41), preferably by subtotal hysterectomy. Good judgment is required in defining just when stimulation should be asserted to have failed. In Case 37 it was carried out for too long a period, the patient being allowed to lose more blood than she could afford to. After Cæsarean section and suture of the uterus it should be carefully examined for external partial ruptures (vide Cases 4 and 33). The presence of these will be shown by free intraperitoneal blood. These ruptures are due to the extreme tension, possibly coupled with necrosis, rupturing the peritoneum and the outer muscle layers, which contain more fibrous tissue than do the inner layers, and are also, of course, more stretched than are the inner layers. These tears should be sutured. The uterus should then

1. Since writing this paper I have seen four other cases treated by the latter method with equally good results.

be examined as a matter of interest for subperitoneal hæmorrhages, which are usually present, as are also intramuscular hæmorrhages, which may be noted at the time of Cæsarean section. The cases in which the uterus was sutured after Cæsarean section (Cases 3, 5 and 33) have all recovered, and Cases 3 and 4 have each had a live baby at term since, without untoward symptoms. The cases in which hysterectomy has been necessary (Cases 37 and 41) have died shortly after operation from the combined shock and loss of blood.

(2) *Retroplacental Hæmatoma.* By this term I mean hæmorrhages which are entirely behind the placenta, and are, as a rule, associated either with no external loss or only with a show. There were seven cases of this type in the series of fifty cases. They are clinically unrecognizable, but should always be suspected when, in a case of toxæmic albuminuria or chronic nephritis, intra-uterine death of the fœtus has occurred. They appear to be of particularly frequent occurrence in cases of chronic nephritis, the three chronic nephritis cases in this series all being in this class. There may be a history of a sudden short attack of pain, but more often there is a complete absence of symptoms. The condition is not of necessity associated with death of the fœtus, which will be dependent on the area of placenta separated. Albuminuria was present in all cases in this series.

Combined Cases. These cases are by far the most numerous. In the series of fifty there were twenty-five cases which came under this heading. Albuminuria was present in twenty-two of the cases, and was definitely absent in only two. Hyaline and granular casts were frequently present. The cases can be divided into two subgroups: (*a*) Cases of internal hæmorrhage in which recovery of the uterus has occurred almost at once, or at any rate before the patient is seen, so that when first seen she is losing blood; (*b*) Cases which have started with external hæmorrhage, but in which a sudden severe hæmorrhage has caused paralysis of the uterus, with consequent cessation of loss.

(*a*) These cases are by far the most frequent. They show a combination both in symptoms and signs of internal and external hæmorrhage. There are, as a rule, abdominal pains, vomiting, anæmia, and often fainting attacks, followed, either at once, or after a short interval, by bleeding. In some cases these symptoms, with the exception of bleeding, are not present, and only the characteristic uterus indicates the presence of internal hæmorrhage. The treatment is on the lines of an internal hæmorrhage after external bleeding has started, *i.e.*, either the case is left to nature or the membranes are ruptured, a tight binder is applied and a hypodermic injection of 1 c.c. of pituitrin administered, or the above

are associated with vaginal plugging. The results to the mothers have been with one exception excellent; one case dying of uterine rupture (vide Case 27). The children have been with three exceptions stillborn.

(*b*) These cases are rare. It is obviously improbable that the uterus will become distended after labour has once started. They are recognized by the characteristic signs of internal hæmorrhage following on external hæmorrhage. The treatment is that of internal hæmorrhage, that is, the treatment of the state present. The results should be satisfactory to the mothers.

Briefly summarized the cases have been as follows:—

	Cases.	Incidence of Albuminuria.	Maternal Mortality.	Fœtal Mortality.
External bleeding	7 (14%)	2 (28%)	0	4[1] (56%)
Internal bleeding	11 (22%)	11 (100%)	2 (18%)	11 (100%)
Combined bleeding	25 (50%)	22 (88%)	1 (4%)	22 (88%)
Retro-placental hæmatoma ...	7 (14%)	7 (100%)	0	3 (42%)
Total	50	42 (84%)	3 (6%)	40 (80%)

To Mr. Comyns Berkeley and Mr. Eardley Holland my best thanks are due for permission to publish the cases treated at the City of London Maternity Hospital. To Dr. Russell Andrews my best thanks are also due for permission to make use of, and publish, certain cases occurring at the London Hospital. Dr. Russell Andrews was extremely kind in allowing me to see and treat cases of utero-placental hæmorrhage in the London Hospital which were under his care, and for this and other kindnesses I am greatly indebted to him.

Results of Treatment.

	(1) None.				(2) Membranes ruptured; tight binder; pituitrin.			
	Mother.		Child.		Mother.		Child.	
External bleeding	4	G (4)	G (3)	SB (1)	—	—	—	—
Internal bleeding	5	G (5)	—	SB (5)	1	G (1)	—	SB (1)
Combined bleeding ...	10	G (10)	G (3)	SB (7)	5	G (5)	—	SB (5)
Retro-placental hæmatoma...	6	G (6)	G (3)	SB (3)	—	—	—	—

	(3) Membranes ruptured; tight binder; pituitrin; plugging of vagina.				(4) Forceps delivery.			
	Mother.		Child.		Mother.		Child.	
External bleeding	1	G (1)	—	SB (1)	—	—	—	—
Internal bleeding	—	—	—	—	—	—	—	—
Combined bleeding ...	8	G(7) D(1)	—	SB (8)	1	D (1)	—	SB (1)
Retro-placental hæmatoma...	—	—	—	—	—	—	—	—

1. Two died after delivery.

	(5) Leg brought down.				(6) Cæsarean section.				(7) Cæsarean hysterectomy.			
	Mother.		Child.		Mother.		Child.		Mother.		Child.	
External bleeding	2	G(2)	—	D(2)	—	—	—	—	—	—	—	—
Internal bleeding	—	—	—	—	3	G(3)	—	SB(3)	2	D(2)	—	SB(2)
Combined bleeding	1	G(1)	—	SB(1)	—	—	—	—	—	—	—	—
Retro-peritoneal hæmatoma	—	—	—	—	1[1]	G(1)	G(1)	—	—	—	—	—

PART II.

THE PATHOLOGY OF UTERO-PLACENTAL (ACCIDENTAL) HÆMORRHAGE.

(From the Pathological Institute of the London Hospital. Prof. Hubert M. Turnbull.)

REVIEW OF LITERATURE.

The pathological work carried out by numerous investigators has been considerable. The uterine musculature, blood vessels and decidua, and the placenta have come under notice and, further, studies of the other organs at necropsy have been made. It seems best to take up these structures separately, and briefly to record the conditions noted by different investigators. The uterine musculature has been particularly studied by Couvellaire (1911),[9] Essen-Möller,[12] Whitridge Williams,[35] Zarate,[38] Seitz,[30] and von Weiss (1897),[34] The decidua by Seitz,[30] von Weiss,[34] Schickele (1904),[29] Zarate,[38] and Biancardi (1905).[5] The uterine blood vessels by Whitridge Williams,[35] Seitz,[30] von Weiss,[34] and Essen-Möller.[12] The placenta by Young,[37] Biancardi,[5] Seitz,[30] and von Weiss.[34] The organs at necropsy by Couvellaire,[8] Bar and Kervily,[3] Hartmann,[17] and von Weiss.[34]

The Uterine Musculature.

Macroscopically, peritoneal ruptures and external, superficial ruptures of the myometrium have been noted by von Weiss,[34] Kouwer (1908,[23] Hicks (1861),[18] Engström (1911), Lieven (1914),[24] Zweifel (1912),[32] and Shaw (1914),[31] and have been given special attention by Knauer (1903),[22] who attributed them to sudden severe distension, and by Fraipont (1914),[14] who attributed them to intramuscular hæmorrhages, diminished elasticity and friability of the uterine wall.

Inter- and intra-fascicular hæmorrhages in the uterine musculature. *"Apoplexie utero-placentaire."* (Couvellaire (1912.)[8]

1. Operation performed for eclampsia.

These have been noted by numerous authors, particularly by Couvellaire,[8] who first accurately described the condition. Secondly by Zarate,[38] Fabre and Bourret 1912),[13] Zweifel,[32] Berggren (1912),[4] Spaeth (1913),[28] Jettner (1913),[19] Fraipont,[14] Clifford (1914),[7] King (1914),[21] Young,[37] Brandt (1914),[6] Shaw,[31] Knauer,[22] Seitz,[30] Targett (1905),[33] Le Lorier (1906),[25] Bar and Kervily,[3] Keyworth (1909),[20] Munro Kerr,[27] and Whitridge Williams.[35] Hæmorrhages into the broad ligaments and Fallopian tubes have also been described by Couvellaire,[8] Whitridge Williams,[35] and Berggren.[4] Whitridge Williams described an extensive hæmorrhage into the right ovary, and Couvellaire described, and illustrated in colour, one case in which there were numerous punctiform and streaky hæmorrhages into both ovaries. Microscopically the hæmorrhage into the uterine wall is described by all authors somewhat as follows:—

The muscle bundles are separated and split into little bundles by streaks of red blood cells which dissect the individual bundles and, indeed, often separate cell from cell. These hæmorrhages are sometimes more numerous near the peritoneum, sometimes near the decidua. They are usually more numerous in the region of the placental site. Zarate[38] recorded one case which appears to me to be of great importance in that it gives evidence of the presence of focal hæmorrhages and necroses which preceded the accidental hæmorrhage. In a case of accidental hæmorrhage associated with eclampsia which came to necropsy he found both recent and old hæmorrhages in the uterine musculature. To quote from his paper:—"The antiquity of some of the hæmorrhages is shown by their differential colouration with eosin. The attention is attracted by the great quantity of connective tissue surrounding degenerated muscle bundles. This degenerated tissue, associated with an infiltration by round cells, is most noticeable in the areas in which older hæmorrhages are." This means, I take it, that there have been an infiltration and replacement by fibrous tissue of degenerated muscle in the region of the older hæmorrhages.

Œdema of the inter-fascicular connective tissue has been described as frequently present by many of the authors especially by Couvellaire[9] and Whitridge Williams.[35]

Leucocytic infiltration of the musculature is noted particularly by von Weiss,[34] who held that it was the cause of the condition. In his specimens the inner layers of the uterine musculature showed the most marked leucocytic infiltration, that in the outer layers being of the nature of a perivascular infiltration. It is important to add that in 4 out of the 5 cases in which the uterus was examined it had been plugged during life with iodoform gauze. This of itself would give rise to leucocytic infiltration. The case that was

not plugged showed the least leucocytic infiltration. Two of his cases showed an increase in the connective tissue of the uterine wall. Leucocytic infiltration of the musculature of slight degree around the older hæmorrhages is also recorded by Zarate[38] in one case, to a slight extent beneath the decidua in one case by Seitz,[30] and in one case to a slight extent with a perivascular distribution by Couvellaire.[8] It is recorded as being absent by Berggren,[4] Essen-Möller,[12] Couvellaire,[8] and Whitridge Williams.[35]

Degeneration of the individual muscle fibres is recorded by Seitz[30] in two cases, by Hartmann[17] and by Zarate[38] in one case, who said "some of the muscle fibres were entirely degenerated. Others showed the characteristic vacuolation of fatty degeneration." Degeneration was not noted by Couvellaire,[8] Essen-Möller,[12] Whitridge Williams[35] or Young.[37]

The Uterine Blood Vessels.

Changes in the blood vessels were noted by Whitridge Williams[35] and Seitz.[30] They were found in the smaller blood vessels by Whitridge Williams in two cases, who thus described them: "In some of the smaller arteries the intima was swollen and projected into the lumen. It was conspicuous on account of the diffuse blue staining which it assumed. In such areas the finer architecture of the vessel wall was disturbed and peculiar defects were seen which appeared as clear circular or elliptical zones, while in other places irregular blue granules were formed which stained very intensely with hæmatoxylin. Throughout these areas nuclear fragmentation and atypical arrangement of cells was observed. In several places defects were present in the intima of the larger arterioles which may have been connected with the hæmorrhagic areas in their vicinity. In the walls of many of the larger veins free hæmorrhage had occurred, just beneath the endothelium, which communicated freely with the hæmorrhagic effusions between the muscle fibres. Similar changes did not exist in the main blood vessels." "In conclusion," he said, "careful study of the arterial lesions in my specimen has led me to believe that they are toxic in origin and are due to the action of some substance circulating in the blood which possibly also produces changes in the smallest arterioles and capillaries which permit the escape of blood into the tissues just as is observed in certain varieties of snake poisoning."

Seitz found changes in the blood vessels beneath the placenta of the nature of a marked proliferation of the vascular endothelium with narrowing of the lumen.

The vascular changes described by Seitz, and the changes in

the vessels, apart from hæmorrhage, which were described and figured by Whitridge Williams, appear to me to be identical with those which normally take place in the sub-placental vessels at or about term, and which have been described in full by Goodall (1910).[39] Lacerations of the walls of some of the blood sinuses were noted by Couvellaire.[8] By many authors the blood vessels are described as being normal, noticeably by Couvellaire,[8] Berggren,[4] Essen-Möller,[12] Fabre and Bourret.[13]

The Decidua.

Hæmorrhage into the decidua has been noted by all observers. Such hæmorrhage is a natural association of labour.

Leucocytic infiltration of the decidua has been noted by many observers, especially by von Weiss,[34] Seitz,[30] who recorded its presence in one case but not in another, and Biancardi,[5] who described it as more or less well marked in two of nine cases. Von Weiss in several of his cases found extreme leucocytic infiltration of the uterine but not of the placental decidua. This infiltration was conspicuous only in the cases in which the uterus had been packed with iodoform gauze and was present in all such cases. There can be little doubt that the packing was responsible for the infiltration. Inflammatory decidual changes were not present in the cases described by Couvellaire,[8] Essen-Möller,[12] Whitridge Williams,[35] Gottschalk (1897)[16] and Zarate.[38] No micro-organisms were found by any observer.

Degenerative changes in the decidua were observed by Seitz,[30] who considered the retro-placental hæmorrhage to be a phenomenon secondary to decidual necrosis or inflammation. He stated that "when the hæmatoma is intra-placental the decidua is still necrosed. Such necrosis cannot be due to compression by the hæmatoma. We have found the cause of the pathological changes in the placenta to reside in the inflammation and degeneration of the decidua basalis." He considered the degeneration of the decidua, which he found most marked in the decidua basalis, to be due to vascular changes or chemico-toxic influences. Von Weiss[34] described extensive decidual necrosis in one case, focal decidual necrosis in two cases, and no necrosis in two cases. The necrosis when present was always most marked in the decidua basalis. Decidual degeneration was also ascribed by Schickele,[29] who attributed it to a lack of blood supply to the decidua by which means a slow necrosis was produced. This lack of blood supply he considered to be in part the result of uterine contractions, in part the result of thrombosis of the sub-placental sinuses. He said that "owing to the lack of knowledge of the changes in the blood

vessels towards the end of pregnancy one was not able to attribute the decidual changes (necrosis) to pathologic changes in the blood vessels, but there is a definite poverty of blood vessels in the region of the placental site. There is seen a defective nutrition of the decidua and a slow necrosis."

Biancardi,[5] in 9 placentæ from cases of accidental hæmorrhage, found in the decidua hæmorrhagic foci in various stages of retrogression, deposits of fibrin, distension and thrombosis of blood vessels, and thickening and degeneration of the decidual elements, varying from cloudy swelling to complete necrosis. "These changes are," said he, "always more marked in the region of placental detachment." He considered the decidual degeneration to be the cause of the placental changes which he described in detail. He admitted, however, that, apart from hæmorrhages and leucocytic infiltration, these decidual changes are found in the decidua of the normal placenta. He attributed the decidual degeneration to deciduo-ovular toxic products or fœtal toxins.

Gaston [15] found that Nitabuch's fibrin layer was thickened and the decidual cells were blurred and contained poorly stained nuclei. He considered that decidual hæmorrhage was the primary cause of placental separation. Gottschalk [16] found "extensive decidual degeneration in excess of the normal with focal areas of complete necrosis. Emboli, some recent some old, in the decidual blood vessels." He considered that the thrombosis was secondary to necrosis of the vessel walls, and that this in turn was secondary to defective decidual blood supply. The hæmorrhages he attributed to local increase of blood pressure, the result of occlusion of capillaries. He was of opinion that "the kidney of pregnancy" and retro-placental hæmorrhage were secondary to decidual necrosis, this in turn being due to defective blood supply. The renal changes he considered to be set up by liberated albuminuric poisons.

Placental Changes.

The changes in the placenta have deen described by many authors. They consist purely of the various stages of infarction. These changes have been carefully studied by James Young.[37] Apart from stages of infarction, which are mentioned by all authors, little has been described. Essen-Möller[12] stated that the degenerative changes found in the placenta are not characteristic of uteroplacental apoplexy but may occur in other conditions as well. Gaston[15] found thick vessels in the villi but no peri- or endarteritis. Seitz[30] found round-celled infiltration of those villi which lay in contact with the degenerated decidua, and considered this to be "inflammatio per contiguitatem." He also noted fibrous hyper-

trophy of von Franque (concentric stenosis) of the vessels in villous stems near infarcts, and considered such hypertrophy as secondary to vascular stenosis and occlusion in the infarcted area. He further found syncytial hyper-activity in one case. This latter condition was also noted by von Weiss[34] in two cases. The paucity of changes described in the placenta, apart from those due to infarction, is noteworthy, when one considers the numerous changes described by various authors in the other structures discussed.

Changes in other Organs.

Excluding those cases in which the condition was associated with eclampsia there are not many recorded cases in which changes in the organs are described. Bar and Kervily[3] recorded a case which is of such importance that I quote it at length.

A vi-gravida; 30 weeks pregnant. A typical case of internal hæmorrhage treated by de Ribes' bag. Patient became worse. Cæsarean section performed. Uterus contracted well. Sutured. Death. Necropsy: the liver was pale and yellowish suggesting an early fatty change. The centres of the lobules appeared as yellow dots. There were no hæmorrhages. Microscopically in the portal zones the liver cells were normal. In the central zones the liver cells were shrunken and often ragged. The cytoplasm was pale, the nuclei (sometimes free, sometimes only surrounded by a little shrunken cytoplasm) were shrunken and pale or hyperchromatic. The area affected was the inner half of the lobule. Sections stained with osmic acid showed fine fatty granules in the cells throughout the liver but more numerous in the cells around the central veins. The kidneys were large and pale. There was granular degeneration of the cytoplasm of some of the cells of the convoluted tubules. There were casts in the tubules of the loop of Henle.

Bar and Kervily arrived at the following conclusions from this case that "*if researches show that hepatic lesions similar to these* are characteristic of utero-placental apoplexy it will be proved that retro-placental hæmorrhages are not merely vaguely allied to nephritis but are directly allied to a complex state which one of us, Bar calls eclampism."

Von Weiss[34] found degeneration of the renal parenchyma in three cases. In one the epithelium was swollen; there were hæmorrhages and granular discoloured hyperæmic foci devoid of nuclei. In one there were swelling and focal necrosis of the epithelium of the convoluted tubules. In one there were swelling and granular opacity of the epithelium of the convoluted tubules, the lumina being frequently occupied by shed cells; there were also focal areas of destruction of cells and disappearance of nuclei. The

same author found liver changes in two cases. In one the liver cells were swollen and discoloured; in the other the cells were large and swollen. In this case he also recorded sub-endocardial ecchymoses.

Knaeur[22] found sub-endocardial ecchymoses in the left ventricle in one case, sub-acute nephritis with hæmorrhage in the second case, and chronic nephritis with atrophy of the kidneys and hæmorrhages into the liver in the third case. Hartmann[17] found fatty infiltration in the liver and, in one case, small blood extravasations in the liver without necrosis or thrombosis.

PERSONAL OBSERVATIONS.

The material at my disposal consisted of three uteri removed for accidental hæmorrhage from Cases 27, 37 and 41 of my paper upon the clinical phenomena, of the kidney and liver of Case 27, the kidney of Case 37 and of the kidney, liver, myocardium, diaphragm and spleen of Case 41. In one of these uteri there was spontaneous rupture. Further, I have taken as controls five uteri removed, at or near term, before or after death: (1) A uterus removed by operation from a case of rupture, the result of obstructed labour due to an impacted breech presentation with extension of the legs. (2) A uterus removed by operation from a case of rupture due to obstructed labour. (3) A uterus from a case of placenta prævia in which, during the performance of bi-polar version, the placenta was widely separated. A leg was then brought down. The uterus was at the time in a state of atony produced by chloroform. There resulted, therefore, extensive intra-uterine hæmorrhage, which caused the death of the patient in less than a quarter of an hour. The uterus was removed immediately after death and showed no macroscopical change. (4) A uterus removed by operation from a case of rupture. In this case there was no absolute obstruction although there was considerable delay and slow advance due to a minor degree of contraction of the pelvic outlet. There was, further, a very considerable degree of albuminuria, which disappeared after removal of the uterus. (5) A uterus removed post mortem from a case of rupture extending from a traumatic laceration produced by a manual dilatation of the cervix.

These two series of cases have been divided into two groups, (A) cases of accidental hæmorrhage, (B) control cases.

In the following description of the macroscopic and microscopic changes, the macroscopic reports of the control cases are omitted, because in these no macroscopic change was observed, except hæmorrhage and œdema along the edge of the rupture.

GROUP A.

Cases of Accidental Hæmorrhage.

Case A1. S.D. 1170, 1917 (Case 27 of Appendix, Part I).

MACROSCOPICAL EXAMINATION.

A uterus 16 cm. long by 12 cm. from side to side by 8.5 cm. from before backwards. Its inferior limit is, on the posterior surface, a horizontal line of operative incision 8 cm. long. The rest of the inferior limit is represented by a ragged tear with thinned edge. This tear passes through the lower uterine segment and is horizontal save to the left where there projects downwards a triangular tongue of tissue measuring 7 cm. in length and 3 cm. across its base; the apex of this tongue is part of the cervical lip. There is subperitoneal blood infiltration extending upwards from the edge of the tear for a distance of 5 cm. upon the anterior wall. A similar infiltration is present throughout both broad ligaments and extends inwards beneath the uterine peritoneum of the posterior surface for a distance of 5.5 cm. on the left side, and 4 cm. on the right side. There are faint, irregular subperitoneal hæmorrhages (up to 1 cm. diameter) scattered over the anterior wall of the upper body and over the fundus uteri. The muscularis varies from a thickness of 5 cm. at the fundus to 2.5 cm. at the lower limit. A central vertical section reveals:—In the muscularis of the fundus, at a depth of 1 cm. from the peritoneal coat, an irregular hæmorrhagic area 2.5 cm. long by 0.3 cm. deep; in the anterior wall a hæmorrhagic area 8 cm. from above downwards by 2.5 cm. deep. The latter hæmorrhagic area involves the total depth of the wall save for the outer 2 cm. and the inner 0.5 cm.; it is slightly raised above the surrounding cut surface and crimson in colour, finely mottled towards its inner margin with translucent grey; it is considerably softer than the surrounding muscularis; it is limited internally by a very definite wavy band of more swollen, less hæmorrhagic, pinkish grey, soft muscularis. Two cm. above the horizontal tear there is a small, irregular, ill-defined hæmorrhage (0.9×0.3 cm.). Parallel vertical sections show even more extensive hæmorrhage than in the areas described. The placental site is situated upon the anterior wall; it measures 6×5.3 cm. and lies internal to the hæmorrhagic area in the anterior wall. The lower margin of the placental site is 6 cm. above the edge of the tear. The tubes and ovaries are normal. There is a corpus luteum (1.4×1 cm.) with glossy fibrous centre (0.6 cm. diameter) in the left ovary.

A piece of liver (5.5×3×0.8 cm.). It is clay coloured. The parenchyma is swollen; the lobular pattern distinct. The periphery of each lobule is outlined by a pale brown rim.

Two portions of kidney (5.5×2.7×1.2 cm.). The capsule is thin, and the stripped surface perfectly smooth. The cortex, 0.8 cm. deep, is swollen, clay-coloured and flecked with very small, faint, paler dots. The cortical pattern is indistinct. The medulla is 1.2 cm. deep.

MICROSCOPICAL EXAMINATION.

Portions were embedded in paraffin and after cutting were stained with

1. The letters S.D. and P.M. and the numbers following them refer to the Ledgers of the Pathological Institute of the London Hospital. S.D. Surgical Department P.M. Post-mortem Department.

Ehrlich's hæmatoxylin and eosin, with Weigert's iron hæmatoxylin and van Gieson's mixture, and with Weigert's fuchsin and neutral red. Other portions were cut upon the freezing microtome, and sections were stained with Sudan III and hæmatoxylin. Sections (*a*), (*b*), and (*c*) were taken from the anterior wall of the uterus, (*d*) from a small hæmorrhagic area at the fundus, (*e*) from the placental site, (*f*) from an area free from hæmorrhage, (*g*) from the edge of the tear in the uterus, (*h*) from liver, (*i*) from kidney.

Section (*a*). *Anterior wall of uterus.* The surface is formed by a narrow zone of decidua vera. Throughout this the cells are dissociated to a greater or less degree and many are shrivelled and devoid of nuclei. This dissociation in places appears to be due to œdema, clear spaces separating the cells; elsewhere the interstices between the cells contain red blood corpuscles. Necrosis is greatest in the areas of hæmorrhage. In the inner sixth of the myometrium the individual muscle fibres cannot be distinguished, but the bundles are represented by a vacuolated matrix in which lie a very few tortuous narrow hyper- and hypochromatic nuclei. The veins are engorged and there are a few areas of hæmorrhage. In the remainder of the inner third a few portions of the muscular bundles show similar degeneration and necrosis. Here, in the majority of the muscle bundles, the cells are not shrivelled but are swollen; they are seldom clearly defined, and absence of nuclei is conspicuous. There are many hæmorrhages; these hæmorrhages are for the most part in the fibrous intermuscular septa. The degeneration and necrosis do not bear a fixed relation to the hæmorrhages. In the middle and outer third the changes in the muscle are very similar; defined cells are more numerous but the majority of these are greatly vacuolated; others are hyaline. There are hæmorrhages in the middle and outer third; they are more numerous in the middle third, but here are scanty in comparison with those in the internal third. In the outer sixth the bundles are widely separated by œdema.

There is an infiltration with leucocytes throughout the decidua, the infiltration being greatest where hæmorrhage is absent. There is considerable infiltration throughout the myometrium. It is chiefly perivascular, lying near veins and capillaries. The majority of the infiltrating cells have a round of oval nucleus and a basophil cytoplasm which is frequently vacuolated; there are also a very few neutrophil leucocytes.

In sections stained with Sudan III fat granules are demonstrated in the endothelium of a few capillaries and in a very few of the infiltrating cells, but not in the muscle fibres. There is a musculo-elastic hypertrophy of the intima of the larger arteries.

Section (*b*). *Anterior wall of uterus.* This section includes the decidua vera. The majority of the muscle bundles in the inner third of the myometrium show degeneration and necrosis similar to those already described. In the central third of the myometrium the fibres in a few bundles are ill-defined and vacuolated or shrivelled, but few have lost their nuclei. In the outer third are very few fibres which are not sharply defined and apparently healthy. There are hæmorrhages in the decidua and several hæmorrhages in the inner third of the myometrium. In the central third there are a few areas of hæmorrhage, and in the outer third are a very few small hæmorrhages. The hæmorrhages are found in areas of degenerate muscle, but there is much muscular degeneration independent

of hæmorrhage. In the outer third, greatest in the outer sixth, is an œdematous separation of the muscular bundles. Cellular infiltration, similar in character and distribution to that seen in the last section, is present. The decidual veins are partly closed by intimal proliferation. Fat is confined to a very few endothelial and free granule-cells.

Section (c). Anterior wall of uterus. This section also includes the decidua vera. Muscular degeneration and necrosis are more extensive than in either of the two preceding sections. They again increase in amount from without inwards, almost the whole of the inner third of the myometrium being affected. Hæmorrhages are confined to the decidua and inner third of the myometrium. Œdematous separation of muscle bundles is again conspicuous in the outer third. The cellular infiltration is also present. There are numerous polygonal and small, narrow, spindle cells loaded with fat in the interstitial tissue of the inner third of the myometrium; similar cells are scattered throughout the outer two-thirds. A very few of these spindle cells are of larger size, and may be atrophied muscle fibres.

Section (d). Fundus of uterus. A portion of myometrium from fundus uteri. In the inner third are extensive necrosis and degeneration of the myometrium together with slight œdema. In the central third the degeneration is slightly less and the œdema is greater. In the inner half of the outer third separation of the muscle bundles by œdema is very conspicuous, the muscle fibres here are less degenerated but are for the most part very narrow, pale and ill-defined. In the outer half of the outer third there is little evidence of œdema or of muscular degeneration. There are a few hæmorrhages into the interstitial tissue of the middle third. In the outer third, in both its outer and inner halves, there are numerous large hæmorrhages. Slight cellular infiltration is again present.

Section (e). Placental site in uterus. In the decidua basalis wide venous spaces are filled by red blood and clot. There is much hæmorrhage into the decidual tissue. In the inner third of the myometrium degeneration and necrosis of the muscle are extensive. A conspicuous feature is the different degree to which different fibres in one and the same bundle are affected. Thus, there are many bundles in which groups of fibres are swollen, hyaline and nucleated, whilst the remaining fibres are represented by a vacuolated area almost or completely devoid of nuclei (Fig. I). The nuclei in the hyaline fibres are pyknotic. Vacuolated hyaline fibres are also present. In the central and outer third similar degeneration and necrosis are present but are of much less degree. There are no hæmorrhages. Infiltration with neutrophil leucocytes is very great in the decidua and is conspicuous in the inner muscularis. Towards the serosa the infiltration decreases, and lymphocytes become much more numerous than leucocytes.

There are many round and polygonal cells loaded with fat in the decidua. In the myometrium there is a considerable number of fat granule cells in the interstitial tissue. Narrow spindle cells containing fat also lie between and parallel to the fibres in many bundles of muscle. In transversely cut muscle bundles the majority of these cells are triangular or stellate and lie between muscle fibres. They belong to the interstitial tissues and some at any rate represent the endothelium of capillaries.

Section (f). Non-hæmorrhagic uterus. In this section degeneration of the myometrium is much less obvious. In many bundles, however, and

more especially in portions of bundles, the muscle fibres are greatly vacuolated. The vacuolation is associated with karyolysis. Hyaline degeneration is very rare. The degeneration is slightly greater in the internal and central thirds. There is no hæmorrhage save in the decidua. The decidua is infiltrated with neutrophil leucocytes, lymphocytes, and plasma cells. In the myometrium there is a slight perivascular infiltration with lymphocytes, larger epithelioid cells and plasma cells. The cytoplasm of some of the epithelioid cells is vacuolated.

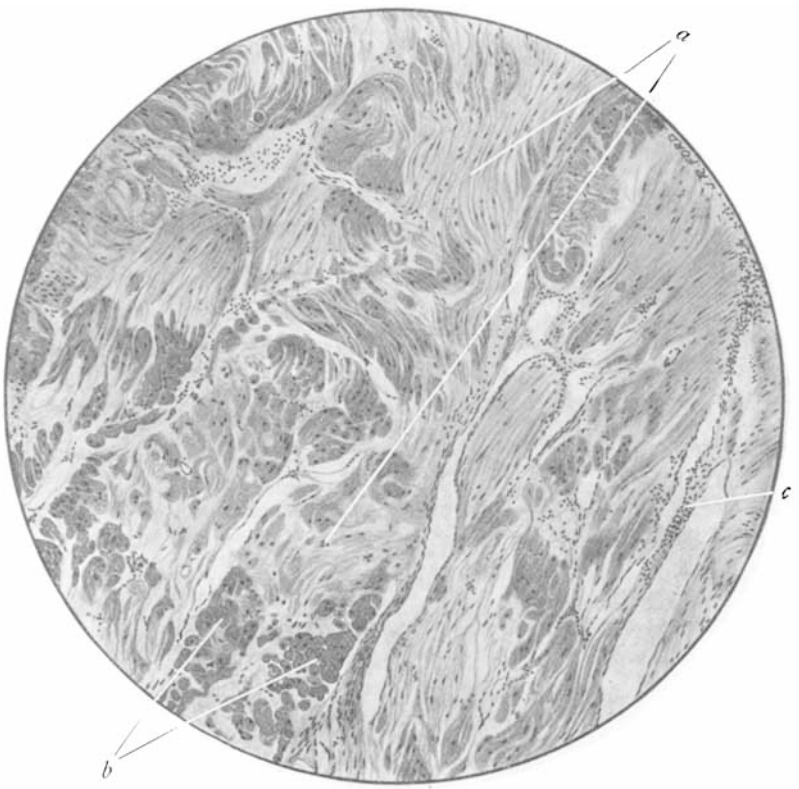

FIG. I.

S.D. 1170/1917.

Section of inner third of myometrium showing (*a*) extensive areas of degeneration and necrosis; (*b*) scattered focal islets of healthy muscle fibres, and (*c*) perivascular leucocytic infiltration.

Section (*g*). *Edge of tear in uterus.* A section taken at right angles to the long axis of the tear. It includes endometrial and peritoneal surfaces for about 2 cm. to the side of the line of cleavage. The decidua drops towards the line of cleavage so that the line of cleavage measures in depth one-quarter of the depth of the uterine wall at the other end of the section. For four millimetres from the line of cleavage the whole wall is represented by a few small islands of degenerate and necrosed muscle; narrow dissociated muscle fibres, and widely dissociated capillaries lying in a sea of blood. In places there are many leucocytes in the hæmorrhage and to a less extent in the fragments of tissue. The hæmorrhage extends beneath

the decidua throughout the section, affecting rather less than the inner half of the myometrium. It decreases in extent as it passes from the line of cleavage. As it decreases the fibres in the muscular bundles become less and less dissociated, but there is very little diminution in the degeneration and necrosis of the muscle. In the outer half of the myometrium hæmorrhages are absent; the bundles are separated by œdema; there is very little degeneration of muscle fibres and necrosis is confined to some of the more internal bundles.

Section (*h*). *Kidney.* Œdema is indicated by separation of the tubules from one another and from their basement membrane. The cells of the first convoluted tubules and ascending limbs of the loops of Henle vary in size and are frequently ill-defined and vacuolated or fragmented. Numerous nuclei are very faintly stained and shrivelled; pyknotic nuclei are less common; cells without a nucleus are frequent. In many first convoluted tubules the cells are almost completely necrosed. The cells of the second convoluted tubules are fatty and usually dissociated. There are small granules of fat in the epithelium of some of the collecting tubules. In the discharging tubules the cells show perinuclear vacuolation and frequently complete disintegration. There is no cellular infiltration.

Section (*i*). *Liver.* Small sparse fatty granules are present in the hepatic cells and are most numerous in those of the centres of the lobules.

Case A2. S.D. 1135, 1918 (Case 37 of Appendix, Part I).

Macroscopic examination.

A uterus amputated through the upper part of the cervix and hardened in formaldehyde. It measures 15×10×5 cm. The wall is 2 cm. thick at the fundus, and narrows to 1.2 cm. thick at the lower extremity. The peritoneal surface is smooth and pale, save in the following areas. At the lower extremity of the anterior surface is a patch of bright red hæmorrhage (3 cm. diameter) in the subserosa. Around the left cornu the serosa covers a bright red and purple patch (4.5 cm. diameter). On the right cornu is a blue purple area (2 cm. diameter), and a purple area (1 cm. diameter) lies immediately behind the reflection of the right broad ligament near its centre. The inner surface is covered by a velvety, bright red membrane save for an area (12×8 cm.) on the posterior aspect; here the surface is ragged and deep red; this is apparently the placental site. The cut surface here is pure white with scattered, red specks. The cut surface in the upper part of the anterior wall is, in the inner 1.5 cm., bright red and ragged. An incision through the left cornu passes into the placental site. Here on the cut surface there is, immediately beneath the serosa, a layer (0.5 cm. deep) in which white myometrial strands are separated by wide spaces filled by fluid red blood; subjacent to this is a layer (0.8 cm. deep) of white tissue in which are scattered narrow red lines (0.2 cm. long); internal to this is 1 cm. of hæmorrhagic red tissue. An incision through the right cornu shows some narrow clefts, filled with blood, immediately below the serosa; in the remainder of the cut surface, white bundles are separated by bright red lines. In the fundus, in the centre of the right half, the inner two-fifths of the wall are hæmorrhagic and bright red. In other portions of the wall the cut surface shows small grey areas, separated by white bands, and no red areas. Serial transverse sections show scattered red flecks in the wall beneath the placental site. Other parts show scattered red flecks.

Accompanying specimen is a piece of kidney (6×2×2 cm.). Cortex almost white, medulla very pale purple. Labyrinths somewhat opaque and white.

MICROSCOPICAL EXAMINATION.

The following portions were taken for microscopic sections :—(*a*) pure white area in anterior wall of uterus; (*b*) hæmorrhagic area in upper part of anterior wall; (*c*) left cornu (placental site); (*d*) hæmorrhagic area in left cornu; (*e*) kidney. The sections were stained by the same methods as in Case A1.

Section (*a*). *Non-hæmorrhagic anterior wall of uterus.* The decidua consists of a narrow superficial zone of spongy tissue, containing vascular and glandular spaces. There is no cellular degeneration. There is considerable infiltration with lymphocytes and less numerous neutrophil leucocytes, the infiltration being especially periglandular. There are no hæmorrhages and no changes in the walls of the blood spaces.

In a few places in the inner third of the myometrium there are a few red blood cells, immediately outside the vessels. There is widespread but slight perivascular infiltration with lymphocytes, larger mononuclear cells and an occasional neutrophil leucocyte. The infiltration affects the whole wall, but is greatest immediately below the decidua. Certain groups of muscle fibres are narrower than others and are vacuolated. The nuclei in these groups are faintly stained or are absent. These changes are most extensive in the inner quarter. There is intimal proliferation in a few large veins. A section stained with Sudan III shows fine granules of fat filling the cytoplasm of free granule-cells in the fibrous tissue. The muscle cells are free.

Section (*b*). *Hæmorrhagic anterior wall of uterus.* The inner one-sixth is feebly stained and has a homogeneous appearance. No decidua is recognizable. The muscle cells are either shrivelled or swollen, and few contain nuclei. Where the muscle is shrivelled, the interstitial tissue is swollen and is not stained red with van Gieson. There is a little hæmorrhage in this zone. In the subjacent three-sixths there is extensive diffuse hæmorrhage, and also œdematous swelling of trabeculæ (Fig. 11). The muscle cells in the inner part of this zone show very similar changes to those just described, but on passing deeper more nuclei are visible, and ultimately there is very little degeneration or necrosis. In the next sixth the muscle bundles are very widely separated by an extreme swelling of the fibrous stroma which is due to impregnation with an albuminous coagulated fluid. The outer sixth appears normal. Leucocytic infiltration is slightly less than in section (*a*). The walls of the arteries and veins in the inner necrotic area are swollen and œdematous; the muscle fibres of the media are shrunken and the majority are without nuclei. Here, and in all other sections, the changes in the walls of the vessels resemble those in the surrounding tissue, but are, if anything, less severe, not more severe. Fat is limited to free cells in the fibrous stroma. Such fatty cells are few, except in the outermost third.

Section (*c*). *Left cornu of uterus, with placental site.* In the inner fifth, which includes a part of the placental site, there is extensive hæmorrhage throughout the spongiosa. This hæmorrhage is, in places, extreme. Where the hæmorrhage is very great, the tissue is entirely destroyed; where great, the decidual cells and, deeper, the muscle fibres, are separated and extensively necrosed. Where the tissue is less dissected by blood,

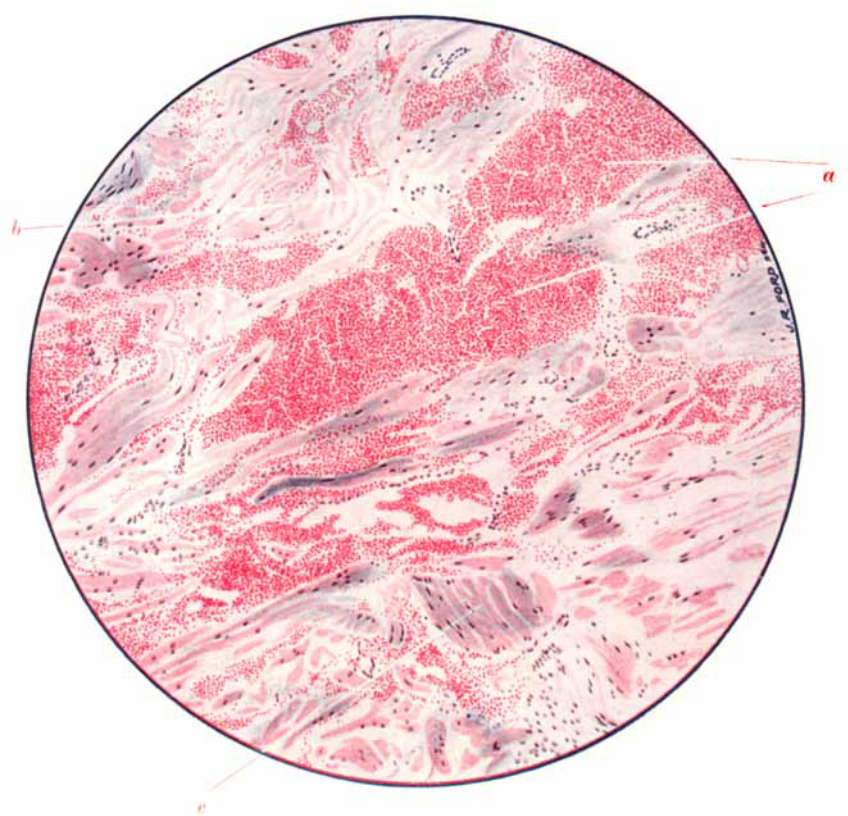

FIG. II. S. D., 1135/18. Section of myometrium.

Shows (*a*) an extensive haemorrhage; (*b*) degeneration necroses of muscle fibres; (*c*) healthy muscle fibres.

the muscle fibres are less deeply stained and less sharply defined than in the subjacent two-fifths. There is little loss of nuclei. In the next two-fifths hæmorrhage is considerable, but not nearly so extensive as in section (*b*). There is very little evidence of degeneration and necrosis. In nearly the whole of the outer two-fifths there is separation of the muscle bundles by albuminous fluid. This is not seen in the extreme outer part, but here the muscle fibres are feebly stained, ill-defined, often vacuolated and frequently devoid of nuclei. They are less degenerate in the œdematous area. Leucocytic infiltration is greater in the inner two-fifths than in previous sections. In the spongiosa neutrophil leucocytes are numerous and beneath this plasma cells are present in addition to lymphocytes and occasional leucocytes. In the spongiosa the blood spaces are filled by red clot; beneath this zone several arteries are greatly narrowed by intimal proliferation. Fat is again confined to round and spindle connective tissue cells. These cells are most numerous where the myometrium is least altered.

Section (*d*). *Hæmorrhagic left cornu of uterus.* In the outer zone of longitudinal fibres the myometrium appears normal. Internal to this is a broad zone in which muscle bundles are widely separated by hæmorrhage and œdema. Throughout the remainder of the wall are numerous large focal hæmorrhages. Areas of necrosis occupy large portions of the inner third of the wall. Perivascular leucocytic infiltration is less than in previous sections. There are a few fat granule cells in the interstitial tissue.

Section (*e*). *Kidney.* There is a conspicuous cloudy swelling of the epithelium of the first convoluted tubules. In several of these tubules there is severe dropsical degeneration. There is a little cloudy swelling of the epithelium of the ascending loops of Henle. A multinuclear giant cell is seen in the lining of one convoluted tubule. There is slight fatty degeneration of the epithelium of the second convoluted and collecting tubules. Albuminous casts are present in a few ascending limbs of the loops of Henle, and second convoluted, collecting and discharging tubules. The epithelial lining of the tubules in the medulla is separated by œdema. There is no active inflammatory reaction.

CASE A3. S.D. 189/19 (Case 41 of Appendix, Part I).

MACROSCOPICAL EXAMINATION.

A uterus amputated through the upper part of the cervix and hardened in Kaiserling No. 1. It measures 17×16×4.5 cm. There is subserous hæmorrhage about both cornua over an area 4 cm. in diameter. Fainter, purple, smaller areas of subserous hæmorrhage are numerous over the posterior surface and there are a few on the anterior surface. On section, the lining of the posterior wall is grey, granular and nodular; on the anterior wall it is rough, ragged and hæmorrhagic. The wall is 2.4 cm. thick in the lower part. There are hæmorrhages in the subserosa which extend for a depth of only 1 cm. into the muscularis. The muscularis elsewhere shows white strands enclosing very small, grey areas and grey, narrow lines. There are also in the muscularis a number of groups, varying in number in different sections, of pin-point, red flecks; they are most numerous in the upper part of the body and much more numerous anteriorly than posteriorly. In the anterior wall of the body near the mucosa are a few ill-defined red areas up to 0.5 cm. in diameter.

NECROPSY.

A complete post mortem examination was performed on this case. The following is an extract of the findings :—*P.M.* 78. 1919. *Anæmia. Operation : Hysterectomy for internal accidental hæmorrhage. Toxæmia of pregnancy.* Severe parenchymatous degeneration of liver (14 lbs. 13 ozs.). Numerous faint, pin-head, opaque, yellow necroses throughout liver especially in right lobe. Anæmia of myocardium and of all organs and tissues. Severe parenchymatous degeneration of kidneys (11 ozs.). Slight myeloid transformation of spleen.

MICROSCOPICAL EXAMINATION.

Sections were stained by the methods enumerated above. The following sections were taken from the material obtained by operation : (*a*) from posterior wall of uterus, 2 cm. from lower end, including a few pink points; (*b*) from the right cornu, with hæmorrhage extending for 0.8 cm. beneath serosa; (*c*) from anterior wall, 4 cm. from fundus, to show red area near mucosa.

Section (*a*). *Posterior wall of uterus.* The inner surface is covered by a narrow layer of decidua. In large areas of the inner and central thirds of the myometrium the muscle fibres are vacuolated and ill-defined. Where these changes are greatest the bundles in longitudinal section are represented by an almost homogeneous matrix, composed of indistinct parallel wavy fibrils traversed by parallel ill-defined clefts, and furnished with few nuclei. In cross section such bundles are represented by a meshwork of homogeneous cytoplasm containing a few nuclei and enclosing large vacuolar spaces. There is considerable perivascular infiltration with lymphocytes and larger basophil cells throughout the myometrium, in the decidua there is a similar infiltration in which a few leucocytes are present.

Section (*b*). *Right cornu of uterus.* There is very little muscular degeneration. Infiltration of the myometrium is greater and contains a few leucocytes. The muscle bundles in the outer third of the wall are widely separated by œdema, coagulated albumen and blood. In the outer sixth the bundles appear to float in blood.

Section (*c*). *Anterior wall of uterus.* There are many areas of vacuolar degeneration and necrosis of the muscle in the inner third of the myometrium, and one focal area of necrosis in the central third. In the decidua and inner third of the myometrium hæmorrhages are numerous; in the outer third there are two hæmorrhages and œdema is conspicuous. There is infiltration similar to that in the previous section. There is fat in a few endothelial cells.

The following sections were taken post mortem :—(*d*) from the upper part of the cervix uteri; (*e*), (*f*) and (*g*) from the liver; (*h*) from the kidney, (*i*) from the myocardium, (k) from the diaphragm, (*l*) and (*m*) from the spleen.

Section (*d*). *Upper part of cervix uteri.* There are a few muscular bundles in which the individual fibres have fused so as to form a homogeneous matrix in which nuclei are almost completely absent. More numerous are small areas occupied by a widely meshed net of delicate

collagen fibrils, enclosing large clear spaces. Many of such areas lie within muscular bundles, groups of muscle fibres lying in their periphery and projecting in places into their centtres. They do not, therefore, merely represent œdema of the interstitial tissue, but an œdematous destruction of muscle. Both the above forms of necrosis are found chiefly in the immediate neighbourhood of a large hæmorrhage beneath the mucosa. Isolated areas of œdematous necrosis are found, however, at a distance from the hæmorrhage. In the deeper layers of the muscularis and in the submucosa there is a considerable infiltration which contains many neutrophil leucocytes. In the intermediate muscularis infiltration is very slight, and neutrophil leucocytes are rare.

Sections (*e*), (*f*) and (*g*). *Liver.* There are grey thrombi in a few large hepatic veins. The cells in the outer third of the lobules are small, dissociated, sharply defined and frequently furnished with two nuclei. In the central two-thirds the cells are swollen, arranged in columns, and can seldom be differentiated from one another; many of their nuclei show karyolysis. Large vacuoles lie in the columns chiefly in their periphery. In sections stained by Sudan III there is much fat in the cells of the central columns; the dissociated peripheral cells seldom contain fat. Proliferation of interstitial tissue and infiltration of portal systems are absent. The capillaries contain many neutrophil leucocytes.

Section (*h*). *Kidney.* The cells of the first convoluted tubules and the ascending limbs of the loops of Henle are swollen, faintly stained and distinctly granular. In many the nuclei are very faintly stained, in others no nucleus is visible. There is a little fat in the cells of the second convoluted tubules and some of the collecting tubules. The cells lining the discharging tubules show extreme perinuclear vacuolation and frequently complete disintegration. There are hyaline casts in several of the discharging tubules.

Section (*i*). *Myocardium.* There is slight œdema of the interstitial tissue. Cross striation is distinct in the muscle fibres. There is lipochrome in the sarcoplasm at the poles of the nuclei, but the fibres contain no fat. There are many leucocytes in the capillaries.

Section (*k*). *Diaphragm.* There is extensive hæmorrhage into the lipomatous tissue beneath the pleura. There are small areas of infiltration with lymphocytes and epithelioid cells in the peritoneal fibrous tissue. Cross striation is distinct in the muscle.

Sections (*l*) and (*m*). *Spleen.* The capillary veins are usually narrow. The reticular strands are somewhat swollen, their fibrils are fragmented, and many of the nuclei of the cells of the reticulum are very faintly stained or are absent. There are very few free cells in the reticular strands. Such as are present are chiefly plasma cells and eosinophil leucocytes.

In the capillary veins and in the peripheral zones of the Malpighian bodies, there are numerous free cells. The great majority of these are neutrophil leucocytes of which many have round nuclei. With these is a considerable number of large mononuclear leucocytes and endothelial cells. Eosinophil leucocytes are relatively rare. There are several neutrophil myelocytes, a few normoblasts and one or two eosinophil myelocytes. No megakarocyte was observed.

GROUP B.

Control Cases.

Case Bi. S.D. 1198. 1918. *Rupture of uterus due to obstructed labour.*

MICROSCOPICAL EXAMINATION.

Section (*a*). *Posterior wall of uterus, above tear.* There is considerable patchy infiltration containing many neutrophil leucocytes in the decidua, and very slight perivascular infiltration in the myometrium. There are no hæmorrhages and there is no œdema in the myometrium. The muscle fibres are large and sharply defined (Fig. 3). The only evidence of

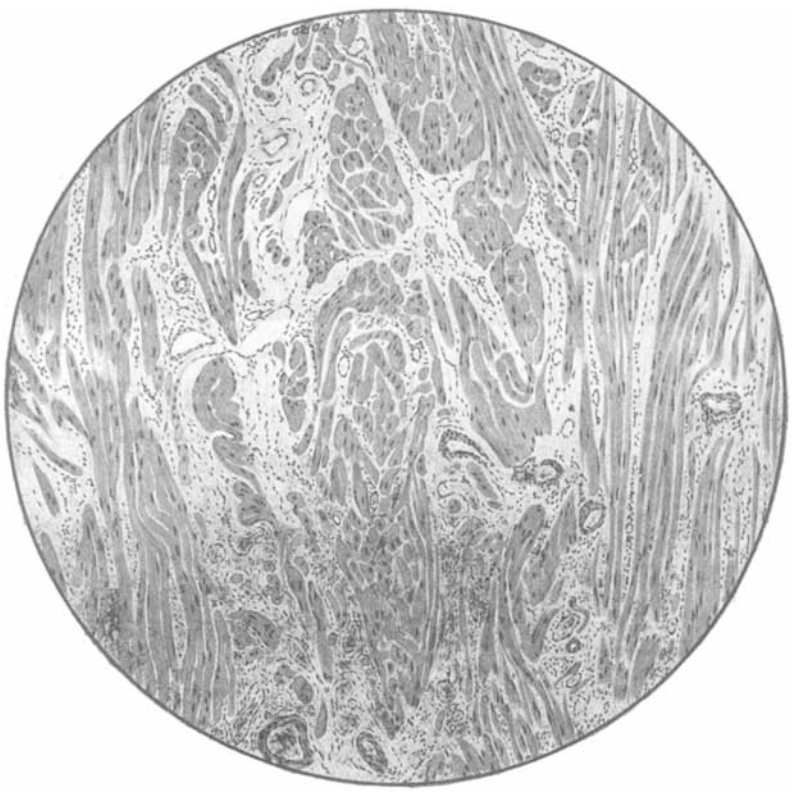

Fig. 111.

S.D. 1198/18.
Section of myometrium of upper segment showing normal muscular bundles.

degeneration is a vacuolation which is seen in a few fibres, especially in bundles cut transversely. The lumina of the veins and of several arteries are narrowed by a proliferation of the fibrous, elastic and muscular elements of the intima. In sections stained with Sudan III there are a few small granules of fat in a very few spindle and round cells in the interstitial tissue about vessels.

Sections (*b*) and (*c*). *At right angles to tear in upper uterine segment.* In both sections the torn edge is only about one-third the length of the total thickness of the uterine wall, the decidual surface curving outwards

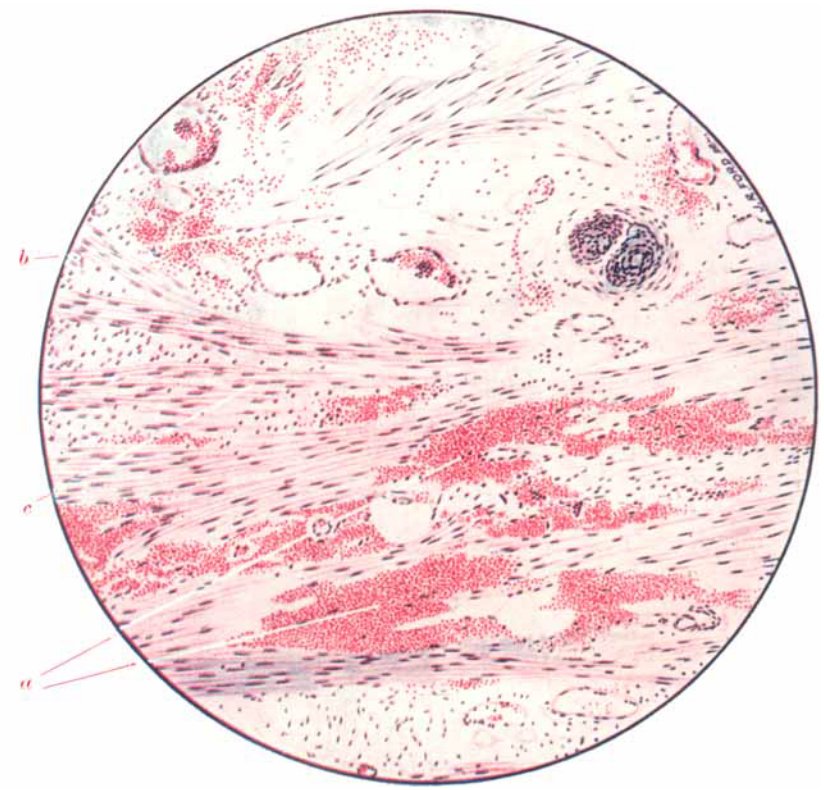

FIG. IV. S. D., 1198/18. Section of stretched lower uterine segments.

Shows (*a*) hæmorrhage; (*b*) œdema; (*c*) attenuation of otherwise normal muscle fibres.

towards the line of cleavage. The tear follows the course of large fibrous intermuscular septa, and in (*b*) has opened a large vein. Thus, the uterine wall, before rupture, appears to have thinned at a point where the interstitial tissue was relatively abundant and the muscularis relatively scanty. There is hæmorrhage in the decidua, in intermuscular septa and in the subserosa for a moderate distance from the line of cleavage. In many of the muscular bundles, actually isolated by the hæmorrhage, the fibres are pale and ill-defined whilst their nuclei are shrunken or absent. These bundles are infiltrated with numerous leucocytes. Apart from a slight interstitial œdema the remainder of the myometrium resembles that in section (*a*). In sections stained by Sudan III fat is only found in a very few interstitial cells. The muscular necrosis in these sections is closely associated with the hæmorrhage in the margin of the line of cleavage and appears to be secondary thereto.

Section (*d*). *At right angles to tear in lower uterine segment.* The line of cleavage is only slightly shorter than the total depth of the wall, the mucosa curving out for but a short distance. The line of cleavage is remarkably straight. Leucocytic infiltration is present throughout, and is greatest at and near the line of cleavage. There is extreme œdema of the inner three-fifths of the myometrium throughout the section. External to this, the œdema is relatively slight. Extensive hæmorrhages are present throughout and are more abundant towards the limit of the section furthest (2 cm.) from the line of cleavage than in the margin of the line of cleavage. They are most abundant in the inner, extremely œdematous zone. The muscle fibres in the inner three-fifths of the myometrium are widely separated by the œdema and are represented by sinuous threads; loss of nuclei is not appreciable (Fig. 4). In sections stained with Sudan III fat granules are present in a few interstitial cells, and at the poles of the nuclei of some of the attenuated muscle fibres.

The abnormalities in this section are not confined to the margin of the tear nor are they focal in the remainder of the tissue. They are evenly distributed throughout. Compared with the cervix removed post mortem in Case 3 of accidental hæmorrhage (p. 92) the abnormalities are much more general and diffuse. In the case of accidental hæmorrhage there were focal areas of œdema in which the muscle bundles had almost entirely disappeared. In this case there is almost universal dissociation of the muscle fibres by œdema with little evidence of necrosis.

CASE B2. S.D., 732. 1919. *Rupture of uterus in obstructed labour.*

MICROSCOPICAL EXAMINATION.

Section (*a*). *Edge of the laceration in the lower uterine segment.* There is hæmorrhage on, in, and in a few places, at a little distance from, the line of fission. The muscle bundles throughout are separated by œdema and there is a little cellular infiltration of the interstitial tissues similar to that in preceding cases. There are a few small hæmorrhagic areas in the decidua at a distance from the line of cleavage. The fibres of the myometrium appear almost without exception to be longer and more sharply defined than in the previous case. Within the areas of hæmorrhage the fibres, in some of the bundles, are ill-defined and are occasionally slightly vacuolated. There appears also to be some loss of nuclei. Nowhere, however, are there the conspicuous degenerations and necroses

which were widely distributed throughout the myometrium in the cases of accidental hæmorrhage.

Section (*b*). *Edge of laceration in dilated lower uterine segment.* The line of cleavage is less than two-thirds of the depth of the uterine wall, the decidua and subjacent myometrium curving out towards the line of cleavage. At, and for some distance from, the edge of the laceration hæmorrhages are numerous through the myometrium. In the subserosa great hæmorrhage extends for a long distance from the edge of the rupture. There are very numerous hæmorrhages in the submucosa and subjacent myometrium to a depth rather less than the inner half of the wall. External to this there are scattered small hæmorrhagic areas. There is great œdema throughout. Perivascular infiltration, containing many neutrophil leucocytes is also present throughout. For a considerable distance from the edge of the rupture the muscle fibres are represented by widely separated, very narrow, often sinuous fibrils containing spindle nuclei. Throughout the remainder of the wall the muscularis has for the greater part undergone a similar change. Bundles of stout fibres are present, however, in the outer half of the wall, and occasional bundles showing only slight dissociation are present at the sides of some of the larger vessels. In the more solid bundles a considerable number of the fibres have undergone hyaline degeneration and there is a deficiency of nuclei. The changes in the cervix resemble those in Case B1, and differ in the same manner from those in the cervix in Case A3 of accidental hæmorrhage.

CASE B3. S.D., 1277b. 1919. *Placenta prævia and intra-uterine hæmorrhage.*

MICROSCOPICAL EXAMINATION.

Section of wall of upper uterine segment. There is considerable infiltration of the decidua; among the infiltrating cells is a moderate number of leucocytes. In the remainder of the wall there is a slight perivascular infiltration in which leucocytes are scanty. Compared with the cases of accidental hæmorrhage the muscle fibres are stout and show very little variation in size and, with the exception of a narrow zone immediately beneath the mucosa, they are much more sharply differentiated. A vacuolar degeneration of different type is present. In the transversely cut bundles of the subserous zone perinuclear vacuolation is very conspicuous; many bundles appear to be almost entirely composed of a mosaic of rounded spaces containing central nuclei. Occasional fibres showing a similar vacuolation, but usually of less degree, are seen throughout the rest of the myometrium.

CASE B4. S.D., 1419. 1917. *Rupture of uterus; slight obstruction; albuminuria.*

MICROSCOPICAL EXAMINATION.

Sections (*a*) and (*b*). *Placental site on anterior wall of uterus.* Vacuolation, loss of definition, and loss of nuclei is very widely spread throughout the inner half of the myometrium. They are less in degree and distribution in the outer half. There are no hæmorrhages. There is considerable œdema of the outer half of the myometrium. In sections stained by Sudan III there are fat granules in a very few interstitial cells in the outer half of the myometrium.

Sections (*c*) and (*d*). *Posterior wall of uterus.* Resemble sections (*a*) and (*b*) save that in the outer longitudinal layer the muscle fibres are shrunken and vacuolated.

Section (*e*). *Lower uterine segment at right angles to serous and subserous prolongation of tear.* The uterine wall tapers towards the lower or cervical end. Vacuolation and loss of differentiation of the muscle fibres is present throughout the inner half of the myometrium. In the outer half, in the margins of the tear, the bundles are isolated by hæmorrhage; the fibres show similar changes, or are hyaline, and there is extensive loss of nuclei. On both sides of the tear there is great œdema of the outer half of the myometrium. On the uterine side the muscular bundles are separated by the œdema and there is conspicuous karyolysis. On the cervical side the fibres in the bundles are dissociated and are seen as sinuous threads usually furnished with nuclei. On this side there are hæmorrhage and great infiltration with leucocytes. In sections stained with Sudan III fat is present not only in interstitial cells but at the poles of the nuclei of a few attenuated muscle fibres.

CASE B5. S.D., 930. 1919. *Rupture of uterus produced by manual dilatation of cervix.*

MICROSCOPICAL EXAMINATION.

Section (*a*). *Uterine wall at placental site.* There is a slight dissociation of the muscle fibres such as occurs when specimens have been kept for some time before fixation. The muscle fibres in the inner sixth of the myometrium are less deeply stained, and in general slightly smaller than those in the outer five-sixths. In this inner zone the fibres in longitudinal section are frequently split by narrow longitudinal clefts, and the individual fibres cannot be differentiated. There are no hæmorrhages. There is an infiltration similar to that in all previous cases.

In sections stained with Sudan III granules of fat are present in a considerable number of cells of the interstitial tissue, chiefly in spindle cells.

Section (*b*). *Uterine wall opposite placental site.* In the inner quarter of the myometrium the fibres show a slightly greater degree of the degeneration present in the inner sixth of section (*a*). In a few fibres the nuclei are shrunken, or are only just recognizable; a few fibres are without nuclei.

SUMMARY AND COMPARISON OF MICROSCOPIC FINDINGS.

In the uterine wall of the three cases of accidental hæmorrhage the following histological changes were found:—An infiltration of the decidua with cells among which neutrophil leucocytes were numerous; a slight perivascular infiltration of the myometrium, which decreased in amount from within outwards, and in which leucocytes were scanty; a conspcuous leucocytic infiltration in the margins of the rupture (Case A1); fat granules in occasional interstitial cells and endothelial cells, and, in A1, apparently also in degenerate muscle fibres; a conspicuous œdema, usually albuminous, in the outer part of the myometrium; widespread, conspicuous, focal areas of degeneration and necrosis in the myometrium of the upper uterine segment, especially in its inner half;

widely-spread areas of hæmorrhage in the myometrium of the upper uterine segment; an absence of invariable coincidence between the areas of necrosis and of hæmorrhage, so that the necrosis could not be considered to be secondary to the hæmorrhage; in A3 in which examination was made of the lower uterine segment, a large focal area of necrosis, hæmorrhage and œdema in the inner portion of the myometrium.

In the uterine wall of the five cases taken as controls the following changes which had been found in the three cases of accidental hæmorrhage were again found in all cases; the cellular infiltration of the decidua and myometrium; the conspicuous leucocytic infiltration in the margins of ruptures; fat granules in occasional interstitial cells and endothelial cells, throughout the myometrium.

In B1, a case of rupture of the uterus in obstructed labour, and in B4, rupture of the uterus following trivial obstruction to labour, fatty granules were also found at the poles of the nuclei of a few muscle fibres in the neighbourhood of the ruptures.

In other respects the control cases differed from those of accidental hæmorrhage: œdema in the upper uterine segment of the control cases was confined to the neighbourhood of ruptures; but was found throughout such sections as were taken of the stretched lower uterine segment (Cases B1, B2, rupture of uterus due to obstruction, and B4, rupture of uterus associated with trivial obstruction).

In B1, a case of rupture of the uterus due to obstructed labour, necrosis was confined to muscle fibres enclosed within the hæmorrhage at the margin of the rupture. The muscle fibres in the lower uterine segment were greatly elongated and attentuated, but necrosis was not observed. In the upper uterine segment, the margin of the rupture being excluded, the only evidence of degeneration was vacuolation of a few muscular fibres. In B2, the second case of rupture due to obstruction, sections were taken from the lower uterine segment alone, including the rupture. There were, again, degeneration and some necrosis of fibres included within areas of hæmorrhage, and great elongation and attentuation of the muscle fibres throughout. In B3, the case of distension of the uterus by hæmorrhage due to a placenta prævia, the upper uterine segment was alone examined; degeneration was confined to a perinuclear vacuolation of the fibres in the longitudinal layer of muscle beneath the serosa. In B4 the case of rupture of the uterus with trivial, obstruction, widely-spread degeneration and necrosis of the muscle were present in the inner half of the myometrium of the upper and lower segments. This necrosis was less severe than in the cases of accidental hæmorrhage. In the lower segment there was also the attenuation of muscle fibres seen in B1 and B2. In B5, the case of rupture of the uterus during manual dilatation of

the cervix, degeneration and necrosis were again present in the inner portion of the myometrium in both portions of the upper segment examined. The necrosis was less severe than in B4.

Hæmorrhage in the upper uterine segments of the control cases was confined to the margin of a rupture (B1). In the lower uterine segment hæmorrhage was found in the margin of ruptures (Cases B1, B2 and B4) and a diffuse hæmorrhage was also found in the two cases in which the rupture was due to gross obstruction (B1 and B2).

Where in the upper segment of the control cases there were both hæmorrhages and necroses, the necroses were found within areas of hæmorrhage at the margins of ruptures; the necrosis appeared to be the result of isolation of muscular bundles by hæmorrhage.

Comparison of the cases of accidental hæmorrhage (A) with the control cases (B) shows, therefore, that certain changes were present in all the uteri examined. These changes were: infiltration of the decidua with cells among which neutrophil leucocytes were numerous, a perivascular infiltration throughout the myometrium which decreased in amount from within outwards and in which leucocytes were scanty; fat granules in a few cells of the interstitial tissue, in a few endothelial cells and in a very few muscular fibres.

Inasmuch as these changes were present not only in the cases of accidental hæmorrhage but in all the examples of various conditions taken as controls, they appear to be changes common to all uteri about full time. They are certainly not peculiar to cases of accidental hæmorrhage.

Further, hæmorrhage and a conspicuous leucocytic infiltration were present in the margins of all ruptures, not only in the case of accidental hæmorrhage but in the control cases.

This hæmorrhage and leucocytic infiltration can only be considered to be the result of rupture.

In the cases of accidental hæmorrhage focal hæmorrhage was widespread throughout the myometrium of the upper uterine segment. In the control cases there was no hæmorrhage in the myometrium of the upper uterine segment, except in the margins of ruptures. In the lower uterine segment, however, of two of the control cases (B1 and B2) diffuse hæmorrhage was associated with attenuation and elongation of the muscular fibres. The two control cases were cases of rupture of the uterus due to obstruction to labour.

Diffuse hæmorrhage was found, therefore, in the stretched, lower uterine segment of the two cases of obstruction to labour. There can be little doubt that these hæmorrhages were the direct result of mechanical stretching of the lower uterine segment. The upper segment of these uteri was doubtless protected from stretching and

consequent hæmorrhage by active contraction and retraction. These observations suggest that the hæmorrhages in the upper segment of the three cases of accidental hæmorrhage may have been due to stretching, by internal hæmorrhage, of upper segments which were not protected by active contraction. This conclusion is excluded, however, first by the absence of microscopic evidence of stretching of the muscular fibres, and secondly by the control case B3. In Case B3 there was, owing to separation of a placenta prævia during the bringing down of a leg, a greater distension of the uterus, by internal hæmorrhage, than in any of the cases of accidental hæmorrhage. Further, contraction and retraction of the upper segment in this case were excluded, the uterus being in a state of atony produced by chloroform. Yet, in this case, B3, there was no hæmorrhage in the upper uterine segment.

It can be concluded, therefore, that diffuse hæmorrhage in the lower segment of the cases of obstructed labour was due to mechanical stretching, but the widespread hæmorrhage in the upper uterine segment of the cases of accidental hæmorrhage cannot be ascribed to a similar mechanical cause.

For similar reasons the great œdema, for the most part albuminous, which was present in the outer zones of the myometrium throughout the uteri in the cases of accidental hæmorrhage cannot be ascribed to mechanical stretching, although great œdema was present in the stretched lower segment of the control Cases B1 and B2 in which the uterus ruptured owing to definite obstruction to labour, and B4 in which the uterus ruptured in the presence of trivial obstruction to labour. Neither can the œdema found in the cases of accidental hæmorrhage be considered of similar origin to that found in the upper uterine segment of control cases. In the control cases it was only found in the margins of ruptures; in the cases of accidental hæmorrhage it was present in all sections, including those of the two cases in which there was no rupture.

The following changes were, therefore, peculiar to the cases of accidental hæmorrhage:—a severe œdema and a widespread focal hæmorrhage in the myometrium which were independent of stretching and of rupture.

Finally, in all three cases of accidental hæmorrhage, focal areas of degeneration and necrosis were widespread throughout the myometrium, especially in the inner zones. This degeneration and necrosis were associated with hæmorrhage, but they were not due to the hæmorrhages because they did not occur invariably in areas in which hæmorrhage was present. They differed, therefore, from the degeneration and necrosis found in control cases within the hæmorrhages at the borders of ruptures. The degenerations and necroses were even more conspicuous than the widespread hæmorrhage and œdema which have been shown to be peculiar to the

cases of accidental hæmorrhage. But these degenerations and necroses were not peculiar to the cases of accidental hæmorrhage. They were found in less degree in Case B4, in which rupture of the uterus was associated with trivial obstruction, and in still less degree in Case B5, in which the uterus ruptured during manual dilatation. On the other hand, they were not found in the two cases of rupture due to obstructed labour (B1 and B2) nor in the case of distension of the uterus by hæmorrhage in placenta prævia (B3).

From comparison of the conditions within all the uteri examined and of the mechanical conditions to which these uteri were exposed, it is not possible to find an explanation of the incidence of these necroses. An explanation may be found when other features in the cases are taken into consideration.

In all three cases of accidental hæmorrhage, toxæmia was indicated by the presence of albuminuria during life, and was proved by such histological examinations of the kidney and liver as were made; that is of the kidney in all three cases and of the liver in two cases. In the kidneys there was a severe albuminous and a slight fatty degeneration. In one liver there was a conspicuous fatty degeneration; in the other there was fatty degeneration in the centres of the lobules, whilst in the periphery of the lobules there were changes indicative of a slow destruction and regeneration of parenchyma.

In the control Case B4 there was also evidence of a toxæmia of pregnancy; a severe albuminuria was present, and disappeared soon after the removal of the ovum and uterus.

In the absence of any other explanation of the degeneration and necrosis in the myometrium of the three cases of accidental hæmorrhage and of Case B4, the fact that they were associated with a toxic degeneration of other organs leaves little doubt that the changes in the myometrium were the result of a toxæmia of pregnancy. The absence of evidence of toxic degeneration of the kidney as shown by albuminuria, in Case B5 does not nullify this conclusion. The degeneration and necrosis in Case B5 were of less degree than in the other four cases. They may well have been due to a toxæmia of slighter intensity.

The degeneration and necrosis of the myometrium evidently rendered the uterus liable to rupture. In one of the three cases of accidental hæmorrhage, the uterus ruptured in the absence of any abnormal obstruction to labour. In Case B4 the uterus ruptured in the presence of an obstruction insufficient to cause rupture of a normal uterus. It is possible that the uterus in Case B5 ruptured under a strain which a normal uterus would have withstood; unfortunately no information could be obtained concerning the strain to which the uterus was subjected by the manual dilatation.

Name.	Age.	Parity.	Degree of Albuminuria.	Toxæmic Symptoms.	Period of Gestation.
1. M.K.	36	VIII. Ch. 8, misc. 0	——	——	31½ weeks
2. E.B.	21	0	In quantity	?	?
3. L.K.	28	III. Ch. 3, misc. 0	In quantity	?	?
4. A.A.	33	IV. Ch. 3, misc. 1	4/1,000 (Esbach), hyaline and granular casts	——	Term
5. F.J.	28	III. Ch. 3, misc. 0	——	——	Term
6. M.W.	33	V. Ch. 4, misc. 1	In quantity	——	34 weeks
7. A.N.	32	II. Ch. 2, misc. 0	Fine cloud	——	Term
8. F.W.	19	0	In quantity, no casts	——	32 weeks
9. C.P.	43	XII. Ch. 12, misc. 0	——	——	38 weeks
10. L.S.	34	III. Ch.2, misc. 1	In quantity	– –	29 weeks
11. A.H.	40	VIII Ch. 8, misc. 0	?	Swelling of feet and legs 5 weeks	Term
12. A.S.	25	I. Ch. 1, misc. 0	In quantity	Swelling of legs and hands 3 weeks	Term
13. G.M.	29	II. Ch. 2, misc. 0	In quantity	——	37 weeks
14. J.F.	24	0	In quantity	Swelling of legs, face and abdominal wall 14 days, headache and excitability during labour	37 weeks
15. E.H.	42	XI. Ch. 9, misc. 2	In quantity	——	38 weeks
16. L.R.	31	V. Ch. 3, misc. 2	1.8/1,000 (Esbach)	Swelling of legs, feet and face 4 weeks	33½ weeks
17. M.S.	42	VII. Ch. 7, misc. 0	3/1,000 (Esbach), granular casts	——	36 weeks
18. R.S.	39	IX. Ch. 7, misc. 2	0.75/1,0000 (Esbach), no casts	——	38 weeks
19. A.H.	36	XII. Ch. 11, misc. 1	Fine cloud	——	38 weeks
20. B.C.	34	I. Ch. 1, misc. 0	Fine cloud, granular casts	——	31 weeks
21. M.S.	19	0	Fine cloud	——	?
22. A.J.	32	III. Ch. 2, misc. 1	In quantity	Headache 1 month	?
23. P.R.	26	I. Ch. 1, misc. 0	Fine cloud	——	38 weeks
24. E.B.	24	II. Ch. 2, misc. 0	2/1,000 (Esbach), hyaline and granular casts	——	35 weeks
25. E.D.	25	III. Ch. 3, misc. 0	Thin cloud, no casts	Swelling of legs 3 weeks, headache throughout	Term
26. S.L.	38	IV. Ch. 4, misc. 0	——	——	37 weeks?
27. L.J.	41	IX. Ch. 9, misc. 0	In quantity, granular casts	——	Term
28. E.C.	39	IX. Ch. 8, misc. 1	Fine cloud	——	35 weeks?
29. E.D.	30	I. Ch. 1, Misc. 0	In quantity, no casts	——	38½ weeks

Abdominal Pain.	Type.	Day of puerperium on which urine became free from albumen.	Treatment.	Result.	
				Mother.	Child.
—	External	—	Membranes ruptured, leg brought down	G.	D.
?	Combined, internal>external	—	Forceps delivery, pituitrin	D.	S.B.
?	Internal	10th day	Cæsarean section	G.	S.B.
Present	Internal	17th day	Cæsarean section	G.	S.B
—	External	—	None	G.	G.
Present	Combined, int.>ext.	9th day	Membranes ruptured, vagina plugged, pituitrin	G.	S.B.
—	Retro-placental hæmatoma	14th day	None	G.	G.
Present	Combined, int.>ext.	4th day	Membranes ruptured, vagina plugged, pituitrin	G.	S.B.
—	External	—	External version, membranes ruptured foot brought down	G.	D.
—	External	4th day	None	G.	S.B.
—	Combined, int.>ext.	—	None	G.	S.B.
Present at 30th week	Retro-placental hæmatoma at 26th and 30th week	Chronic nephritis	None	G.	G.
—	Combined, ext.>int.	13th day	None	G.	G.
—	Internal	9th week	None	G.	S.B.
Present	Combined, int.>ext.	11th day	Membranes ruptured, vagina plugged, pituitrin	G.	S.B.
Present	Combined, int.>ext.	7th day	Membranes ruptured, vagina plugged, pituitrin	G.	S.B.
—	Combined, int.>ext.	8th day	None	G.	S.B.
—	Internal	10th day	None	G.	S.B.
—	Combined, ext.>int.	13th day	Membranes ruptured, tight binder	G.	S.B.
Present	Combined, int.>ext.	9th day	None	G.	S.B.
—	Internal	8th day	None	G.	S.B.
Present	Combined, int.>ext.	6th day	Pituitrin	G.	S.B.
Present	Combined, int.>ext.	3rd day	Membranes ruptured, vagina plugged, pituitrin	G.	S.B.
Present	Combined, int.>ext.	10th day	Membranes ruptured, vagina plugged, pituitrin	G.	S.B.
—	Internal	6th day	None	G.	S.B.
—	Combined, ext.>int.	—	Pituitrin, tight binder	G.	S.B.
Present	Combined, int.>ext.	—	Membranes ruptured, vagina plugged, pituitrin, rupture of uterus, hysterectomy	D.	S.B.
Present	Combined, int.>ext.	4th day	Membranes ruptured, tight binder, pituitrin	G.	S.B.
Present	Combined, int.>ext.	6th day	None	G.	S.B.

Name.	Age.	Parity.	Degree of Albuminuria.	Toxæmic Symptoms.	Period of Gestation
30. R.O.	31	IV. Ch. 2, misc. 2 : 1st misc., 2nd 6/12 mac., 3rd misc., 4th 8/12 mac.	Fine cloud	General malaise	23 weeks
31. M.B.	42	XIII. Ch. 12, misc. 1	Fine cloud	——	31 weeks
32. G.C.	21	0	Fine cloud, granular casts	Vomiting 14 days, jaundice post-partum	34 weeks
33. R.P.	32	VI. Ch. 5, misc. 1	6/1,000 (Esbach), no casts	Swelling of legs 2 days	Term
34. S.K.	24	0	2/1,000 (Esbach), hyaline and granular casts	Swelling of face 19 days, legs 7 days, headache and vomiting throughout pregnancy	31 weeks
35. L.H.	39	VIII. Ch. 8, misc. 0	1/1,000 Esbach	Swelling of legs 3 weeks	28 weeks
36. K.W.	29	0	In quantity	——	38 weeks
37. J.F.	40	XIII. Ch. 12, misc. 1	4/1,000 (Esbach), hyaline and granular casts	——	32 weeks
38. A.C.	32	IX. Ch. 10, twins (2), misc. 1.	2/1,000 (Esbach), hyaline and granular casts	——	$33\frac{1}{2}$ weeks
39. A.H.	32	IV. 1st, 2nd and 3rd 36 weeks mac., 4th 36 weeks induction	Fine cloud	Headache and vomiting throughout	24 weks
40. S.S.	29	VIII. Ch. 6, misc. 2	——	——	37 weeks
41. C.W.	37	VII. Ch. 7, misc. 0	12/1,000 (Esbach), hyaline and granular casts	Headache 2 days	Term
42. M.E.	28	V. Ch. 4, misc. 1 : 1st misc. 5/12, 2nd S.B. 8/12, 3rd term, alive, 4th S.B. 7/12, 5th term, alive	6/1,000 (Esbach), hyaline and granular casts	Headache 1 day ante-partum, persistent vomiting, headache and drowsiness for 48 hours post-partum	35 weeks
43. E.H.	35	IX. Ch. 8, misc. 1	Cloud, hyaline and granular casts	Headache 1 month	38 weeks
44. E.R.	28	II. Ch. 2, misc. 0	Fine cloud	——	34 weeks
45. E.G.	29	I. Ch. 1, misc. 0	——	——	Term
46. S.K.	27	II. Ch. 2, misc. 0	——	——	34 weeks
47. E.E.	34	III. Ch. 3, misc. 0	0.5/1,000 (Esbach)	——	32 weeks
48. C.W.	39	XV. Ch. 13, misc. 0	1/1,000 (Esbach), granular casts	——	38 weeks
49. I.Y.	21	0	Fine cloud, pus cells, colon bacilli	Pyelitis of pregnancy, swelling of legs 1 month	Term
50. L.C.	29	0	15/1,000 (Esbach)	Eclampsia.	34 weeks

Vomiting occurred in Nos. 4, 6, 8, 12, 15, 23, 24, 27, 28, 29, 31, 33, 34, 37, 38, 41, 42, 43, 44, 50.

There was pronounced anæmia in Nos. 2, 3, 4, 6, 8, 9, 15, 16, 19, 22, 23, 24, 26, 27, 28, 29, 31, 33, 34, 35, 36, 37, 38, 41, 42, 43, 47, 48.

Fainting occurred in Nos. 2, 3, 4, 6, 15, 27, 28, 31, 33, 34, 35, 36, 37, 41, 42, 43, 47.

Abdominal Pain.	Type.	Day of puerperium on which urine became free from albumen.	Treatment.	Result. Mother.	Child.
—	Retro-placental hæmatoma	Chronic nephritis	None	G.	S.B.
Present	Combined, ext.>int.	5th day	Membranes ruptured, vagina plugged, pituitrin	G.	S.B.
—	External	5th day	Membranes ruptured, pituitrin	G.	S.B.
Present	Internal	4th day	Cæsarean section	G.	S.B.
Present	Retro-placental hæmatoma	10th week	None	G.	S.B.
Present	Internal	7th day	None	G.	S.B.
—	Combined, int.>ext.	4th day	None	G.	S.B.
Present	Internal	—	Cæsarean section, hysterectomy	D.	S.B.
—	Combined, ext.>int.	8th day	Membranes ruptured, foot brought down, pituitrin	G.	S.B.
—	Retro-placental hæmatoma	Chronic nephritis	None	G.	S.B.
—	External	—	None	G.	G.
Present	Internal	—	Cæsarean section, hysterectomy	D.	S B
—	Combined, int.>ext.	Trace 14th day	Membranes ruptured, tight binder	G.	S.B.
Present	Internal	10th day	Watched, later membranes ruptured, tight binder, pituitrin	G.	S.B.
—	Combined, ext.>int.	2nd day	Membranes ruptured, tight binder, pituitrin	G.	S.B.
—	External	—	None	G.	G.
—	Combined, ext.>int.	—	None	G.	S.B.
—	Combined, ext.>int.	—	None	G.	G.
—	Combined, ext.>int.	6th day	None	G.	G.
Present	Retro-placental hæmatoma	2nd day	None	G	G.
—	Retro-placental hæmatoma	6 months	Cæsarean section for eclampsia	G.	G.

Placental infarcts were found in Nos. 11, 12, 14, 16, 17, 23, 25, 30, 34, 39, 43, 44, 49, 50.
The umbilical cord measured 20 inches or more in length in all but nine cases, viz., 13 ins. in No. 44; 14 ins. in No. 34; 16 ins. in No. 1; 17 ins. in No. 9; 18 ins. in Nos. 6, 10, 18, 30; and 19 ins. in No. 14.
The Wassermann reaction was tested in 15 cases and was found positive in one only—No. 42.

Conclusions.

1. The changes characteristic of, and peculiar to, cases of accidental hæmorrhage are widespread necrosis, hæmorrhage and œdema in the myometrium.

2. These changes are not due to mechanical injury.

3. The necrosis is closely associated with the hæmorrhages but is not due to the hæmorrhages. The necrosis, hæmorrhage and œdema are due to the same cause.

4. The cause is a toxæmia of pregnancy, which damages the kidneys and liver as well as the uterus.

5. A less degree of this toxæmia can cause necrosis of the myometrium without hæmorrhage and œdema (Case B4).

6. A still less degree can cause a slight degree of necrosis without hæmorrhage and œdema, and without an albuminuria giving evidence of poisoning of the kidney (Case B5).

7. The necrosis of the myometrium renders the uterus liable to rupture.

8. In the uteri affected by this necrosis evidence of fatty degeneration is extremely slight, and is no greater than that in normal uteri at full time.

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