A THESIS ON
ACCIDENTAL ANTE PARTUM HAEMORRHAGE,
ITS AETIOLOGY, PATHOLOGY, AND TREATMENT.

by

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ACCIDENTAL ANTE PARTUM HAEMORRHAGE,
ITS AETIOLOGY, PATHOLOGY, AND TREATMENT.

This thesis consists of two parts. The first part is a critical survey of the views and teaching of recognised authorities on the subject of Accidental Ante Partum Haemorrhage. The second is a detailed record of one hundred consecutive cases of that condition occurring at the Royal Maternity and Simpson Memorial Hospital, Edinburgh, during the period January 1st 1923 and February 22nd 1927.

The record of cases is followed by an analysis, and the conclusions drawn from it are given.
SECTION I. A.

ACCIDENTAL ANTE PARTUM HAEMORRHAGE.

DEFINITION.

Strictly speaking Ante Partum Haemorrhage is bleeding occurring at any period of pregnancy up to the time of the birth of the child. In practice the phrase is used to denote bleeding in cases after the child has become viable - after the 28th week of pregnancy - and before the child has been born. The source of haemorrhage in the great majority of cases is the placental site. It is true that slight haemorrhages may occur from such conditions as carcinoma of the cervix, fibroid tumours of the uterus, and even profuse haemorrhage may result from rupture of a varicose vein in the vulva or vaginal wall. However these latter conditions are rare. Cervical cancer is known to inhibit pregnancy, and fibroid tumours are a frequent cause of sterility. For the purpose of this paper one proposes to regard Ante Partum Haemorrhage as "Bleeding, after the 28th week, from the placental site".

Cases of Ante Partum Haemorrhage are one of two types depending upon the position of the placenta from whose site the bleeding originates.
These types are:

(1) Inevitable Ante Partum Haemorrhage or Placenta Praevia.

(2) Accidental Ante Partum Haemorrhage

In the first type the placenta is placed partly or completely in the lower uterine segment. In the second type the position of the placenta is normal in the upper uterine segment.

**VARIETIES OF ACCIDENTAL ANTE PARTUM HAEMORRHAGE.**

The bleeding from the placental site is due to a premature separation of the placenta. This separation may be partial or complete. The blood may be retained within the uterus, or it may pass into the vagina and so escape. Sometimes there is a combination of the two processes - some blood remaining in the uterus and some escaping. According to the behaviour of the blood Accidental Haemorrhage is one of three types:

(1) Concealed.

(2) Revealed.

(3) Mixed.

In practically all cases more or less blood escapes per vaginam. Totally concealed cases are very rare.

Some authorities subdivide cases of concealed accidental haemorrhage still further, according to/
to the position of the blood remaining in the uterus.

Concealed Accidental Haemorrhage.

(a) Retro Placental
(b) Retro Membranous
(c) Intra Membranous.

Some deny the occurrence of the Intra Membranous type.

However the varieties of accidental Ante Partum Haemorrhage will be discussed more fully later in the paper.
The fact that Accidental Haemorrhage resulted from the separation of a normally placed placenta was first noted by Louise Bourgeois in 1609.

Rigby in 1776 differentiated the condition from Placenta Praevia and called it Accidental Haemorrhage. This term has been objected to on the ground that it might be understood as haemorrhage the result of an accident.

R.W. Holmes of Chicago in 1901 proposed to call the condition "Ablatio Placentae". In Placenta Praevia the placenta from its position in the lower uterine segment will inevitably separate in the process of stretching of that part of the uterus in labour. The bleeding from the site of a normally placed placenta will only occur if certain aetiological factors are present. Hence Rigby's definition. The term "Accidental" is still the more commonly used.

Goodell in 1875 collected details of 106 cases of Concealed Accidental Haemorrhage and pointed out the seriousness of the condition.

Winter in 1885 first directed attention to the presence of albuminuria in cases of accidental haemorrhage.

Holmes in 1901 collected 200 cases of concealed accidental/
accidental haemorrhage.

Observations were later carried out by Dorman, Conclough, and Essen-Müller, over a large series of cases.

In 1914 Dr James Young of Edinburgh carried out work on the aetiology of eclampsia and albuminuria and their relation to accidental haemorrhage.

Whitridge Williams made a study of 18 cases of Accidental Haemorrhage which occurred in the last 2000 patients of his hospital practice prior to July 1915.

In 1921 Gordon Ley produced a valuable and comprehensive paper based on the study of 50 consecutive cases occurring in the City of London Maternity Hospital and in the London Hospital.

In 1926 F.J. Browne read a valuable paper to the Edinburgh Obstetrical Society on his experimental investigation into the aetiology of Accidental Haemorrhage and Placental Infarction.
SECTION I. B.

AETIOLOGY.

FREQUENCY OF THE CONDITION.

Dorman found separation of the placenta on an average of one in 115 labours. Conclough one in 207 labours) Over a large series Essen-Moller one in 216 "

These figures give an average of 1 case in 179 labours or 0.55%.

Concealed Haemorrhage occurred in less than 1 case of 10 of these or less than 0.05%.

This gives a total percentage of 0.60% of labours.

Williams in his series of 2000 consecutive labours noted separation of the placenta in 0.85%

and only 1 case of Concealed Accidental Haemorrhage in 15,000 labours.

Gordon Ley does not give the number of labours occurring in the two hospitals during the period in which he collected the 50 cases.

During/
During the period 1923-28 inclusive, 168 cases of Accidental Ante Partum Haemorrhage occurred at the Edinburgh Royal Maternity Hospital. During the same period there were 10,119 deliveries in the hospital. This gives an incidence of 1 case in 59 deliveries or 1.6%.

Patients "treated" by the hospital numbered 24,234. Thus the average incidence was 1 case in 143 patients or 0.7% of all cases treated by the hospital. Those treated include those delivered in hospital, those who attended at the Ante Natal department but who were not delivered in hospital, and those treated in their own homes.

Ley found in his 50 cases that 9 (18%) were primigravidae and 41 (82%) multiparae.

The figures for the Edinburgh Royal Maternity Hospital were:

<table>
<thead>
<tr>
<th>Year</th>
<th>Cases</th>
<th>Primigravidae</th>
<th>Multiparae</th>
</tr>
</thead>
<tbody>
<tr>
<td>1923</td>
<td>17</td>
<td>5 (29%)</td>
<td>12 (71%)</td>
</tr>
<tr>
<td>1924</td>
<td>23</td>
<td>5 (21%)</td>
<td>18 (79%)</td>
</tr>
<tr>
<td>1925</td>
<td>20</td>
<td>6 (30%)</td>
<td>14 (70%)</td>
</tr>
<tr>
<td>1926</td>
<td>33</td>
<td>7 (17%)</td>
<td>26 (83%)</td>
</tr>
<tr>
<td>1927</td>
<td>44</td>
<td>8 (18%)</td>
<td>36 (82%)</td>
</tr>
<tr>
<td>1928</td>
<td>31</td>
<td>5 (16%)</td>
<td>26 (84%)</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>168</strong></td>
<td><strong>36 Primigravidae 21.4%</strong></td>
<td><strong>132 Multiparae 78.5%</strong></td>
</tr>
</tbody>
</table>
Professor R.W. Johnstone in his Text Book of Midwifery says "The condition (Accidental Haemorrhage) is four times commoner in Multiparae than in Primiparae".

Holmes found in his series of cases 19.2% to be Primiparae.

In Ley's series the average parity of the Multiparae was 5.7, and a maximum parity of 15.

Of the 134 Multiparae in the Edinburgh Royal Maternity Hospital the average parity was 4.2, and the maximum parity 17.

An interesting fact in Ley's cases was an abortion incidence of 1 abortion to 9 labours at term. It will be shown later that one of the theories of premature separation of the placenta is that there is an endometritis present. If this theory is true then one would have expected a high abortion rate.
THE FACTOR OF TRAUMA.

Eden says "With regard to accidental haemorrhage, instances are rare in which it can be attributed solely to direct injury, e.g. a kick on the abdomen or a fall; but there is no doubt that such injuries may cause separation of a healthy placenta from its normal attachments."

Professor Johnstone says "It is probable, however, that these factors (of trauma) have no influence unless there is some underlying weakness in the placental attachments. On the other hand, if such weakness or disease of the uterine wall is present, they may precipitate the disaster by causing sudden alterations in the blood pressure, or exciting contractions of the uterus".

R.W. Holmes of Chicago noted trauma as a possible factor in 67 cases.

Whitridge Williams says he found no history of trauma in any of his eighteen cases. Neither did Ley in his 50 cases.

In the record of the 100 consecutive cases (Royal Maternity Hospital) a history of possible trauma was mentioned in 7 cases. However the "cases" were not taken with this particular point of investigation in view. Possibly had they been so, more cases would have been found in which trauma may have played a part. However/
However there is room for considerable error. A patient is always ready to blame her trouble on some fall or blow. This is especially so if a leading question is asked.

It seems probable that the influence of trauma in the causation of Accidental Haemorrhage is small. It is recognised that abdominal trauma may cause abortion by damage to the placental site, even in a healthy uterus. In accidental haemorrhage the uterus is definitely diseased — as will be described later. A fortiori will the placenta tend to separate as the result of trauma. As a definite history of trauma is obtained in some cases the association is probably accessory and coincidental.

ABNORMAL SHORTNESS OF THE UMBILICAL CORD.

In 1892 Pinard and Varnier described a case of Accidental Haemorrhage in which they assigned the cause to traction on the placenta by an abnormally short umbilical cord.

Others have given this as a cause, especially Gardiner who stressed the point.

However it is rarely that one finds a cord of abnormal shortness. In the series of cases examined the average length of the cord was 20.2".
Gordon Ley does not believe that shortness of the cord can cause traction upon the placenta. He points out that during labour pains the uterus with the placenta contracts down upon the child. Thus the placenta is travelling in the same direction as and with the child. At the end of the pain the uterus relaxes, but at the same time the resisting soft parts of the perineum and the muscles of the pelvic floor cause the child to be pressed back towards the fundus. Thus during labour before the birth of the child there can be no traction upon the cord. It seems possible that if forceps were used wrongly between pains that a short cord might pull upon the placenta.

Thus shortness of the cord is a rare aetiological factor, if it is a factor at all.

**ENDOMETRITIS.**

The frequency of Accidental Haemorrhage increases directly with the number of pregnancies. For this reason endometritis has been suggested as an underlying cause. Yet Ley's finding of a low abortion incidence is against this theory.

Probably an endometritis is present in some cases but like trauma it is accessory and coincidental.
OTHER FACTORS.

Syphilis, rupture of the circular sinus of the placenta, blood diseases, irregular uterine contraction, general disease - all have been given as possible causes.

However there is no doubt that the underlying cause of the condition is Toxaemia.

This factor will now be discussed.

THE INFLUENCE OF A TOXIC STATE.

It has been definitely established that in a large percentage of cases of Accidental Haemorrhage an albuminuria is present. As already mentioned, Winter in 1885 drew attention to this fact. This physical sign is more frequently associated than any other. Different research workers found the frequency of the association to vary in the different series of cases observed. Ley found for example a frequency of 84% (albuminuria) in his fifty cases. Whitridge Williams gave 64.7% as the figure. Essenn-Möller found the frequency in his cases to be only 34.25%.

Another association has been observed by different authorities. This is the frequency of eclampsia occurring along with Accidental Haemorrhage. The/
The aetiology of eclampsia and albuminuria and their relation to accidental haemorrhage was fully discussed in a paper by Dr James Young in 1914. At that time his view was that accidental haemorrhage could not be caused by toxaemia. He says "It is impossible to look upon the toxaemia as the cause of the bleeding. For in a large number of the cases there is no trace of a toxic state." However most authorities are now agreed that the condition is due to a toxaemia, especially since the work of F.J. Browne has been published.

Williams believes that the albuminuria is either an accidental complication, or else a manifestation of a toxaemic process. He does not believe that chronic nephritis is at all a common aetiological factor. Others have regarded chronic nephritis as the common cause of accidental haemorrhage. They argue that a damaged kidney will not allow the unwanted products of metabolism to pass from the body. In the same way toxins from whatever source will be held up. They have pointed out the frequent occurrence of red infarcts of the placenta in chronic nephritis, and suggest that if similar haemorrhages occurred in the decidua a separation of the placenta would probably result.

He recognises the occasional association of eclampsia and accidental haemorrhage but points out that/
that the toxaemia of the latter condition is not of the typical pre-eclamptic type. He examined a number of uteri from cases of concealed accidental haemorrhage. Clinically there had been no albuminuria or pre-eclamptic symptoms. He found degenerative changes in the intima of the smaller uterine arteries. He explained this condition as being due to the circulation of a toxin. The degenerative changes of the vessels resulted in haemorrhage and the development of a condition of "uterine-placental apoplexy."

In a series of 17 cases Williams found albuminuria in 11 cases, but a chronic nephritis in one case only.

In Ley's cases the urine was examined for some time following labour. It was found that the average duration of the albuminuria was 11.3 days. This was excluding those cases (6%) which were found to have a chronic nephritis. He also found that 34% of his patients had toxaemic symptoms, i.e. oedema, headache and vomiting, and that one had eclampsia.

Now as albuminuria is present in so large a proportion of cases of accidental haemorrhage it is reasonable to look upon the cause of both conditions as being the same.

Is Williams wrong in his belief that Chronic Nephritis (Parenchymatous) is rarely associated? Is the condition a simple acute nephritis in which metabolic/
metabolic products are held up and disorganise the uterine muscle and placental site? The albuminuria is unlikely to be physiological as it occurs in so many cases and to such a degree.

Most authorities are agreed that the essential basis of the condition is a toxaemia of pregnancy. They disagree as to the source of the toxin, and also in regard to the process of placental separation and the accompanying haemorrhage.

Dr Young suggests that the important factor is a primary thrombosis in the veins of the Broad Ligament and uterine wall. This results in an engorgement of the uterus and decidua and a consequent rupturing of veins. The placenta is partly raised by the haemorrhage and parts of the tissue become necrotic. The products of necrosis are absorbed into the circulation by the portion of placenta that remains attached to the uterine wall. The toxic products act upon the tissues throughout the body - especially upon the kidney and liver. There are two objections to this theory. The first is that no cause has been found for the thrombosis, and the second is that because of the free anastomosis of veins from the uterus, the thrombosis would have to be a massive one.

In 1925 Dr Young suggested that the condition might be due to an infection of the placenta, but Dr/
Dr F.J. Browne however has found that cultures from the placental infarcts were always sterile.

Ley explains the haemorrhage by saying that it is due to a focal necrosis with bleeding in the muscle of the uterine wall and decidua. As a result of this he says the placenta separates and opens the mouth of the uterine sinuses. He considers the focal necrosis and haemorrhage to be the result of toxaemia.

Morse thought that torsion of the uterus might be the cause of the haemorrhage and utero-placental apoplexy. This view is not held by others. Williams in doing abdominal section for cases of concealed accidental haemorrhage found no torsion of the uterus. Polak reports one case. Browne has found a degree of torsion but there was no haemorrhage.

An extremely valuable paper based upon the experimental investigation into the aetiology of Accidental Haemorrhage and Placental Infarction was given before the Edinburgh Obstetrical Society in March 1926 by Dr F.J. Browne. 3

In 1923 Dr F.J. Browne and Dr W.R. Logan were working in Edinburgh on the aetiology of intra-natal infection of the new-born. A 20 day pregnant rabbit had been given an intra-venous injection of B. Pyocyaneus
in saline solution. Six hours later the rabbit was found to be bleeding from the vagina. The rabbit was killed, and the abdomen opened. On opening one of the pregnant uterine horns a newly dead foetus was found. The placenta was almost completely separated from the uterine wall by a large blood clot. The uterine wall in relation to the sac was examined microscopically. There was found a definite oedema separating the muscle bundles. In the decidua were areas of haemorrhage. These were in close relation to small ruptured blood vessels. The walls of these vessels showed degeneration.

A kidney was examined. It showed a very definite acute parenchymatous nephritis.

The liver showed nothing save a round cell infiltration.

Here then was a case of Accidental Haemorrhage in which it was known that a possible factor of toxaemia was present. There was a nephritis present also, but it was not known whether this was the result of the toxaemia.

The experiments were carried on using solutions of B. Pyocyaneus and other organisms and other substances, e.g. histamine. A number of rabbits were used. Some died, some aborted, but none showed accidental haemorrhage.
It seemed that a factor was missing. Brown thought that it might be Nephritis. He therefore set to work to produce Nephritis in the rabbits.

The possibility of Nephritis playing a part was brought to his mind by the fact that in many of his cases he could obtain a history of Nephritis, or albuminuria in previous pregnancies.

He took 34 rabbits and was able to produce Nephritis by giving intravenous injections of sodium oxalate.

He proved this by finding a high concentration of blood urea. Also several rabbits died and the kidneys showed Nephritis.

He then attempted to produce Accidental Haemorrhage. The rabbits were mated. On the 20th day of pregnancy - which was the equivalent of the 6th month in the human, the rabbit was given an injection of sodium oxalate. A Nephritis had previously been produced by oxalates. A slight haemorrhage occurred the following day. More oxalate was given but this time combined with an emulsion of B. Pyocyaneus. Further haemorrhages resulted.

Five other rabbits were treated by the same methods. He produced accidental haemorrhage in each of the six rabbits. He also produced haemorrhages in the uterine wall and placental infarction.
An interesting fact was that in some of the rabbits although a Nephritis was present, as shown by a high blood urea, there was no albumen in the urine. This points to the fact that some of the patients with accidental haemorrhage and no albuminuria may still have a Nephritis. This finding points out a possible error in Dr Young's deduction that "it is impossible to look upon the toxaemia as the cause of the bleeding. For in a large number of the cases, there is no trace of a toxic state."

As a result of these experiments Browne concluded that an acute Nephritis may be a predisposing cause of accidental haemorrhage, and that although a chronic nephritis may be present it is necessary for there to be an acute exacerbation. He also found that organisms of the coliform group gave the best results with these experiments.

He formulates the following theory as the sequence of events in Accidental Haemorrhage.

The patient has either a chronic Nephritis or an acute Nephritis. Then from some source bacterial toxins are liberated. The toxins act on the kidney and cause an acute Nephritis or an exacerbation. As a result of this, kidney function is upset. The unwanted products of metabolism are retained within the body. These toxins (Endogenous) act upon the uterus, the blood vessels of the uterine muscles, and upon the placenta, and result in premature separation of the placenta and haemorrhage.
It is more common to find toxaemia in Primiparae than Multiparae. Yet in Accidental Haemorrhage we have a toxic state occurring more frequently in Multiparae than Primiparae.

Miller and Whitaker have shown that the antibacterial power of the blood is the same in both Multiparae and Primiparae.

In view of Browne's findings it is reasonable to suppose that the strain of pregnancies has damaged the kidney. The bacteria cannot be eliminated. The toxaemia _ endogenous and exogenous _ becomes intense. A vicious circle is set up. Also as Miller and Whitaker further showed, the antibacterial activity of the blood is depressed in cases of albuminuria.

The patient therefore has little chance of resisting the invasion.

In the following section the pathology of Accidental Haemorrhage will be described, and discussed in relation to the various theories of causation that have been given.
A considerable amount of research work has been carried out on the Pathology of Accidental Haemorrhage. The uterine muscle, the blood vessels, the decidua, the placenta, and tissues and organs throughout the body—all have been studied in large numbers of cases.

**THE UTERINE MUSCLE AND BLOOD VESSELS.**

The most obvious and the most characteristic lesion of the uterus is an intra-muscular haemorrhage. In severe cases of accidental haemorrhage the uterus and sometimes the tubes and ovaries take on a purplish colour and resemble very much in colour a twisted ovarian cyst. In several severe cases Williams was forced to do hysterectomy, because of the danger of post-partum haemorrhage resulting from the failure of the uterus to contract down after the delivery of the child. He carried out an investigation to find the cause of these intra-muscular haemorrhages.

On microscopic examination he found that some of the smaller vessels of the uterine muscle showed degeneration of their walls, and that in relation to these vessels were areas of haemorrhage. The same condition was/
was present in the decidua but it was not so marked. The larger vessels showed no such change. From these findings he explains the process of premature separation of the placenta. He suggests that the degenerative changes in the blood vessels of the uterine wall and decidua are caused by a circulating toxin. Blood then escapes into the decidua basalis causing it to split in such a way that a thin layer attaches itself to the placenta and a thicker layer to the uterine wall. Bleeding continues from the exposed vessels of the placental site. The blood collects behind the placenta. It presses upon areas of the placenta. In slight cases there may be no symptoms of this bleeding and the only sign is seen after the birth of the placenta where there may be small depressions in the maternal side, filled with blood clot. If the haemorrhage is more marked it passes, in the majority of cases, round the membranes and out through the cervix and so to the vagina. Sometimes the blood remains entirely within the uterus - concealed haemorrhage.

Williams gives four reasons for the blood remaining within the uterus.

1. The blood may be retained behind the placenta because the margins of the placenta may remain adherent to the uterine wall.

2. The membranes may be adherent at the os.
(3) The blood may perforate through into the amniotic sac.

(4) The blood may be unable to escape because the presenting part fills the lower uterine segment.

These views are now not generally held.

In regard to the reasons (1) and (2) Ley and John Hewitt have noted in cases of Accidental haemorrhage that the placenta and membranes follow without any delay the birth of the child. There is no evidence of the membranes being adherent.

Neither Ley nor Hewitt has seen a case where the blood has escaped into the amniotic sac. Ley says it is mechanically impossible as the pressure of the blood would cause an increase of pressure in all directions of the amniotic fluid, and this would resist the dipping in of the membranes into the amniotic sac. Personally one would think that the increased pressure on both sides of the membrane might result in rupture from the stretching of an abnormally thin membrane.

In regard to the last reason Ley says there can be no obstruction until the membranes have ruptured.

John Hewitt of Glasgow points out that the blood may not reach the lower uterine segment but may pass up to the fundus of the uterus. Here none of the given factors need come into play.
was present in the decidua but it was not so marked. The larger vessels showed no such change. From these findings he explains the process of premature separation of the placenta. He suggests that the degenerative changes in the blood vessels of the uterine wall and decidua are caused by a circulating toxin. Blood then escapes into the decidua basalis causing it to split in such a way that a thin layer attaches itself to the placenta and a thicker layer to the uterine wall. Bleeding continues from the exposed vessels of the placental site. The blood collects behind the placenta. It presses upon areas of the placenta. In slight cases there may be no symptoms of this bleeding and the only sign is seen after the birth of the placenta where there may be small depressions in the maternal side, filled with blood clot. If the haemorrhage is more marked it passes, in the majority of cases, round the membranes and out through the cervix and so to the vagina. Sometimes the blood remains entirely within the uterus - concealed haemorrhage.

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(2) The membranes may be adherent at the os.

(3) /
especially in those vessels in the muscle tissue below the placental site. He does not mention degeneration of the vessel walls, and he gives the cause of the haemorrhage - as mentioned earlier in this paper - to thrombosis of the uterine veins with resulting "apoplexy of the uterine vessels".

**LEUCOCYTIC INFILTRATION.**

This has been noted by many. This infiltration is especially marked in the region of the placental site.

One observer - Von Weiss - found a very marked leucocytosis in five cases; but of these cases four had been treated with iodoform gauze packing. According to Ley this would have been sufficient to have caused the infiltration.

**OEDEMA.**

Most authorities have described an oedema of the uterine muscle.
PERITONEAL CHANGES.

Occasionally when a post mortem examination has been carried out on a patient who has died from Accidental Haemorrhage, free blood has been found in the peritoneal cavity. On examination the uterus and the peritoneum covering it have shown longitudinal fissures. These are superficial but sufficiently deep to cause a fatal haemorrhage. Their cause is not known. It is probably a result of the degenerative changes in the muscle—the weakened muscle being unable to resist the increased pressure of intra-muscular haemorrhage.

PLACENTAL CHANGES.

All authorities have noted placental infarcts in cases of Accidental Haemorrhage. These are due to interference with the maternal blood supply of the part. When there has been haemorrhage into the decidua basalis, the placenta above the region of the haemorrhage will be raised up and so will be deprived of its blood supply. This part of the placenta shows areas of infarction. These infarcts vary with the age of the clot and the amount of separation that has occurred. Over old clots the infarcts are yellow or white.
Most authorities have noted degenerative changes in the decidua, especially in the decidua basalis. These changes have been described as varying from cloudy swelling to extensive necrosis. In relation to some of these areas of degeneration are regions of haemorrhage. Ley points out that the degeneration and necrosis cannot be due to the haemorrhage because they do not occur invariably in areas in which haemorrhage is present. He believes the degenerative changes to be due to circulating toxins.

A number of authorities have put the cause down as defective nutrition of the decidua.

Schickele - as quoted by Ley - speaks of a defective nutrition of the decidua leading to a slow necrosis. The poor blood supply he says is due to uterine contractions and also thrombosis of the sub-placental sinuses. He says that there is also a poverty of blood vessels in the region of the placental site.

Gottschalk - also quoted by Ley - found in addition to the degeneration and necrosis, emboli in the decidual blood vessels. He says that a defective blood supply causes a necrosis of the vessel walls leading to thrombosis.

Young noted "enormous expansion of the decidual vessels into thin walled sinuses, and here and there haemorrhages."
Of recent work on the pathology of Accidental Haemorrhage the most valuable is that of Gordon Ley (See Bibliography). The material at his disposal was as follows:

**Case A₁** Ruptured uterus from case of Accidental Haemorrhage. Kidney and liver from same case.

**Case A₂** Uterus and Kidney (Accidental Haemorrhage)

**Case A₃** Uterus, kidney, liver, myocardium, diaphragm and spleen (Accidental Haemorrhage)

Then as controls he had 5 uteri

**Case B₁** Uterus removed because of rupture.

**Case B₂** Uterus - obstructed labour - impacted breech.

**Case B₃** Uterus with placenta praevia (Haemorrhage and death).

**Case B₄** Uterus ruptured - no definite obstruction but delay. Albuminuria which cleared up, i.e. toxic.

**Case B₅** Uterus ruptured. Traumatic laceration by manual dilatation of cervix.

The following is an epitome of his findings.

In the cases of Accidental Haemorrhage he found

1. The decidua to be infiltrated with cells especially neutrophil leucocytes.
2. The myometrium to show a slight perivascular infiltration. This decreased from within outwards.
Leucocytes were scanty.

(3) In $A_1$ - the ruptured uterus - there was a definite leucocytic infiltration in the margins of the rupture.

(4) Fat Granules in some interstitial cells, and in A some degenerate muscle fibres.

(5) The myometrium to show a definite oedema in its outer part.

(6) A widespread, conspicuous focal area of degeneration and necrosis in the myometrium of the upper uterine segment.

(7) Widely spread areas of haemorrhage in the myometrium of the upper uterine segment.

He then examined the controls.

He found:—

(1) Cellular infiltration of the decidua and myometrium.

(2) Conspicuous infiltration of margins of the ruptures.

(3) Fat granules.

In $B_1$ and $B_4$

Few fat granules found. The oedema was confined to the region of the ruptures, but was present throughout the lower uterine segments of $B_1$, $B_2$ and $B_4$. 
In B₁ (obstructed labour)
Necrosis was confined to muscle fibres enclosed within haemorrhages at the margins of the ruptures.

In B₂ (obstructed labour)
A slight degree of necrosis in areas of haemorrhages.

In B₃ (Placenta Praevia)
Practically no degeneration.

In B₄ (Ruptured uterus with practically no obstruction)
Widely spread degeneration and necrosis in the inner half of the muscle of the upper and lower segments.
The necrosis was less marked than in the cases of Accidental Haemorrhage.

In B₅ (Ruptured Uterus)
Degeneration and necrosis.

Thus from this work he found that a severe oedema and a widespread focal haemorrhage in the myometrium were peculiar to the cases of Accidental Haemorrhage. Focal areas of degeneration and necrosis were widely spread throughout the myometrium. The degeneration and necrosis were more conspicuous than the haemorrhage and the oedema.

In all these cases of Accidental Haemorrhage there was an albuminuria during life. This showed that/
that there was a toxaemia.

Also on examination of the kidney from these cases and the liver in two cases - degenerative changes were found.

Accidental haemorrhage can be classified as already explained, under three clinical groups.

1. External haemorrhage
2. Concealed
3. Combined external and concealed haemorrhage.

This condition was the most common of all the cases of accidental haemorrhage occurring at the Royal Maternity Hospital during the six years 1926-31. Professor Johnston in his text book stated that this form is the most common. Gordon Lay in his series of 36 cases, on the other hand, found it to be present in only seven cases. Simple external haemorrhage without retention of some blood is probably not so common as the figures of the analysis would show. It is probable that in a number of cases blood clot accompanied or followed soon after the expulsion of the placenta, showing the ease to be a combined condition, and that these facts have not been noted.

The majority of cases of external haemorrhage are not severe. The patient has probably had an uneventful pregnancy.
SECTION I. D.

CLINICAL PICTURE.

Cases of Accidental Haemorrhage can be classified, as already explained, under three clinical groups.

(1) External haemorrhage

(2) Concealed "

(3) Combined external and Concealed haemorrhage.

EXTERNAL HAEMORRHAGE.

This condition was found to be the most common of all the cases of Accidental Haemorrhage occurring at the Royal Maternity Hospital during the six years 1923-28. Professor Johnstone in his text book states that this form is the most common. Gordon Ley in his series of 50 cases, on the other hand, found it to be present in only seven cases. Simple External haemorrhage without retention of some blood is probably not so common as the figures of the analysis appear to show. It is probable that in a number of cases blood clot accompanied or followed soon after the expulsion of the placenta, showing the case to be a combined condition, and that these facts have not been noted.

The majority of cases of external haemorrhage are not severe. The patient has probably had an uneventful pregnancy/
pregnancy without any signs of toxaemia. Possibly there has been a slight albuminuria. Then about the 35th week of pregnancy she has had a loss of blood. This comes on for no apparent reason. There is no pain associated unless the bleeding brings on labour pains. Usually the bleeding does not recur. With suitable treatment the patient goes to term, and, in those cases where the placental separation has not been great delivers a living child.

On examination of the abdomen nothing abnormal is found. The head, if the presentation is a vertex, is well down in the pelvis. The foetal heart will probably be heard. On vaginal examination, if the os is dilated, nothing abnormal is felt. The blood pressure is normal and so too the temperature. The pulse may be a little increased in rate.

Sometimes the bleeding recurs and is severe. In this case there is usually a retention of blood within the uterus as well. The clinical picture is very similar to that which is described under Concealed Haemorrhage. In some cases the external bleeding is great, while the retained bleeding is slight in amount. The patient, although she will show the signs of loss of blood and some degree of shock, she will not show, however, as do the cases of Concealed Haemorrhage, the large, hard, tender uterus, nor will she complain of the severe abdominal pain which is such a marked feature of the 'Concealed' cases.
CONCEALED HAEMORRHAGE.

This is a severe and serious condition. Practically every patient will have had during pregnancy an albuminuria. Probably the first complaint will be a sudden attack of abdominal pain. Possibly there will be a slight vaginal haemorrhage. In the minority of cases there will be no external bleeding.

On examination, if the patient is seen early the pulse rate will be increased to about 100. The temperature will be normal. As the bleeding continues into the uterus the patient will show definite signs of loss of blood and shock. The pulse rate will increase to 100 or 120 or more, and it will become irregular and thready. Respiration increases in rate and becomes shallow, and in very severe cases there is air hunger. The temperature is subnormal. The patient complains of extremely violent abdominal pain over the uterus. On examination of the abdomen the uterus is very hard and tender. The size of the uterus may be greater than one would expect from the stage of pregnancy. The size may increase even in a few minutes due to the bleeding into the uterine cavity and the resulting distension. Foetal parts cannot be felt because the uterus is in tetany. The uterus does not contract. No foetal heart is heard because the child is dead.
On vaginal examination the os is usually closed. If it is patulous the membranes will be felt bulging. If the patient's condition improves it will be due to the return of uterine contractions. This will be shown by the escape of blood which has been forced from the uterus.

It has been pointed out by Hewitt and Cameron that shock plays a bigger part than the severity of the haemorrhage. They point out that in many fatal cases of concealed haemorrhage the loss of blood is trivial as compared with the loss of blood in other obstetrical cases that survive.

COMBINED HAEMORRHAGE.

The difficulty of a true classification of cases of Accidental Haemorrhage will be seen from an examination of the case records. A patient with what is apparently External Accidental Haemorrhage will come under observation. This might possibly be a case of Concealed Accidental Haemorrhage in which the uterus has regained its power of rhythmic contraction. The patient will show, according to the amount of blood lost and the amount of shock, those symptoms and signs that have been described. There will be almost certainly a history of albuminuria. Bourne in "Recent Advances in Obstetrics and Gynaecology/
Gynaecology" speaks of cases which have started as External haemorrhage, but then have had a severe intra-uterine haemorrhage with resulting distension of the uterus, and cessation of the external loss. These cases are rare.
SECTION I. E.

DIFFERENTIAL DIAGNOSIS.

EXTERNAL ACCIDENTAL ANTE-PARTUM HAEMORRHAGE.

A patient in the last three months of pregnancy comes under observation with a history of vaginal bleeding. The important fact to know is whether this is a case of accidental haemorrhage, or whether it is bleeding resulting from placenta praevia. The treatment for each of these conditions is very different. In some cases of accidental haemorrhage one can take an expectant attitude, but if the case is one of placenta praevia then palliative treatment is absolutely contraindicated.

The only certain way of diagnosing placenta praevia is actually to feel the placenta on vaginal examination. This is only possible when the os is open. If the bleeding has been at all marked the os will be sufficiently dilated to allow this. If the os is closed it is sometimes possible to feel the placenta through the fornices. This is difficult unless one has had considerable experience. If on vaginal examination no placenta is felt then the condition is diagnosed as accidental haemorrhage.

A placenta situated in the lower uterine segment
will prevent the foetal head from becoming engaged before the beginning of labour. This sometimes results in a mal presentation. If on abdominal examination the head is engaged the condition is probably accidental haemorrhage.

In the majority of cases of Accidental Haemorrhage there will be a history of albuminuria. In placenta praevia albuminuria is the exception. However not all cases of accidental haemorrhage show albumen, and a few cases of placenta praevia are complicated by its presence.

In the majority of cases of accidental haemorrhage the foetus is dead; but in placenta praevia it is alive at the commencement of labour.

The haemorrhage of accidental haemorrhage is continuous and is blood stained serum. In placenta praevia the blood comes in gushes and is fresh and clotted, and it is apt to cease and later to recur.

According to the amount of blood lost so will the symptoms and signs of anaemia and shock vary. These have been described in the previous section.

As mentioned at the beginning of this paper there are other conditions that may result in vaginal bleeding during the last three months of pregnancy. Bleeding may result from carcinoma of the cervix, from a fibroid polypus, from varicose veins in the vulva, vagina or cervix, from a simple cervical erosion/
erosion, from haemorrhoids, or even from an excessive "show". Careful examination will eliminate these possible causes.

The bleeding will probably be due to the separation of a normally placed placenta, or to placenta praevia. The differential diagnosis is made in the way described.

CONCEALED HAEMORRHAGE.

The diagnosis of a severe case of concealed accidental haemorrhage is not difficult. The symptoms and signs of the condition have been described in the previous section.

The rupture of an advanced ectopic pregnancy may give all the symptoms and signs of internal haemorrhage, but the uterus will not show the hardness of the tetanic uterus of concealed haemorrhage.

In complete rupture of the uterus the foetal parts will be felt through the abdominal wall.

A generalised peritonitis resulting from a perforation of a gastric or duodenal ulcer may resemble superficially a concealed haemorrhage, but on examination tenderness will be found in the iliac fossae, a sign that is absent in the latter condition.
The history too will help to clear up the question of diagnosis.

Many cases of mild concealed haemorrhage give no clinical symptoms or signs until the placenta is born. Some small retro-placental blood clots will then be passed.

**COMBINED CASES.**

The diagnosis is based upon the facts that have been given under the headings of External, and Concealed Haemorrhage.
SECTION I. F.

PROGNOSIS.

Figures given by different authorities:

Out of 129 consecutive cases of Accidental Haemorrhage occurring at Queen Charlotte’s Hospital

Maternal mortality 4%
Infantile " 58%

Prof. Johnstone’s figures:

Accidental Haemorrhage in general

Maternal mortality 5-10%
Infantile " about 60%

Concealed Haemorrhage alone.

Maternal mortality 50%
Infantile " 90%

The prognosis depends a great deal upon the method of treatment chosen and upon the skill with which it is carried out. Little can be done to save the life of the child. It seems however that by following a more conservative treatment than has been followed in the past a lower maternal mortality is obtained. Treatment will be discussed in the following section.
SECTION I. G.

TREATMENT.

PROPHYLAXIS.

Efficient ante and post natal care and treatment will help to prevent the occurrence of accidental haemorrhage.

It is true that a number of cases of accidental haemorrhage show no albuminuria and no evidence of toxaemia during pregnancy. In these cases the first evidence of the condition is a sudden vaginal bleeding accompanied or not by abdominal pain.

However the serious and severe cases of Concealed haemorrhage in practically every case show evidence of a toxaemic state. Now all patients who become pregnant should undergo a thorough physical examination. All septic foci should be dealt with. Full instructions in the hygiene of pregnancy should be given.

As soon as albuminuria, or other toxaemic signs or symptoms are discovered vigorous eliminative treatment should be carried out. The patient should be given hot baths, hot packs, castor oil, and enemata. The diet should be cut down. Under this treatment in the majority of cases the condition will clear up. If the albuminuria does not clear up within a fortnight the/
the labour should be induced rather than run risk of permanent damage to the kidneys supposing the case be allowed to go to term. If the patient escapes eclampsia the kidneys may nevertheless be seriously damaged. Thus should the patient become pregnant again still further strain will be thrown upon them. If a septic focus then develops the patient will have both factors—according to Prof. Browne's theory—necessary to cause an accidental haemorrhage.

Thus it is seen that thorough ante natal examination and treatment will do much to eliminate possible factors in the causation of Accidental haemorrhage.
TREATMENT OF ACCIDENTAL HAEMORRHAGE.

EXTERNAL HAEMORRHAGE.

Many of these cases are admitted to hospital with a history of a sudden attack of vaginal bleeding. The loss is probably small. On examination the bleeding has either stopped or else there is a slight oozing. The patient's general condition is good. Here the only treatment required is rest. The patient should be put to bed. Morphia should be given. Then by giving a suitable diet, and plenty of fluids, and by keeping the bowels open, the patient is placed into the best possible physical condition. Should there be any albumen in the urine this will probably clear up under the treatment.

Sometimes the bleeding is more severe. In these cases labour pains are usually present. Active measures have to be taken to induce labour and so control the bleeding. The cervix is usually about two fingers dilated. Two methods of treatment are suitable at this stage. One can either plug the vagina or else rupture the membranes. The patient is usually shocked and as packing the vagina definitely increases shock, some prefer simply to rupture the membranes.

Vaginal/
Vaginal plugging is the method advocated by Tweedy and the Dublin School.

The vagina is first douched and then very carefully swabbed with iodine. Then small swabs of cotton wool are squeezed out of lysol solution and then packed into the vagina. This is done systematically. First the fornices are tightly packed, and then more and more swabs are introduced until the whole vagina is tightly packed. Then a vulvar pad is applied with a T bandage. A tight abdominal binder is applied as well. The uterus is further stimulated to contract by an hypodermic injection of pituitary.

Tweedy states that the packing controls the bleeding by direct pressure upon the uterine arteries. Many obstetricians - amongst whom may be mentioned FitzGibbon of Dublin and Cameron of Glasgow - have pointed out that although the packing may exert some pressure on the uterine arteries it cannot possibly control the bleeding from the ovarian arteries.

The fact remains that the bleeding is frequently stopped. Probably the packing, in preventing the blood in escaping from the uterus causes the intra uterine pressure to increase until it is equal to the maternal blood pressure. The bleeding then stops. The packing stimulates the uterus to contract. When the contractions are strong the packing is removed.

Another disadvantage of packing is the possibility of/
of septic infection. This is probably more liable to occur outside hospital. Gordon Ley says that he has not had a case of sepsis following vaginal packing for accidental haemorrhage. Cases in which Caesarean section has been performed following vaginal plugging certainly show a higher sepsis rate than those in which Caesarean section alone has been performed. While advocating vaginal plugging to control the bleeding, the Dublin school stresses the importance of treating the accompanying shock. If the patient's condition is serious they give an hypodermic injection of ether, and then brandy. Intramuscularly they give 1 cc. of infundibulum extract. They bandage the patient's legs and arms to drive the blood from the peripheral circulation. The foot of the bed is raised. Then two pints of Sodium bicarbonate (one drachm to the pint) are given intravenously at 100°-115°F. If the condition is not so urgent it can be given into the breasts. They watch the patient carefully. After six hours they remove the plug. If bleeding recurs they replug the vagina.

If one decides against plugging the membranes can be ruptured and a tight abdominal binder applied and pituitary given. The escape of liquor allows the uterus to contract down upon the child, and so the bleeding/
bleeding is controlled.

Sometimes bleeding continues. It is better to do Caesarean section in these cases rather than to attempt accouchement forcée. The results of the latter treatment are bad. The patient already shocked is unable to withstand such drastic operative measures.

CONCEALED HAEMORRHAGE.

Hewitt and Cameron, both of Glasgow, gave their views on the treatment of this condition to the Edinburgh Obstetrical Society on January 9th 1926.

They maintain that in this condition shock plays a bigger part in causing the high mortality than does the loss of blood. They point out that in other obstetrical conditions such as abortion and placenta praevia the patient frequently loses a greater amount of blood than do the cases of concealed haemorrhage, and yet survive. They therefore attempt to deal first with the patient's shocked condition. They put the patient to bed and give her morphia. The uterus is in a state of tetany. This condition they say will tend to control the haemorrhage more effectively than would any measures that one might take. They watch the patient and in many cases the condition improves.

Bleeding/
Bleeding becomes less and the patient recovers from shock. Then they give intramuscular injections of 0.5 cc. of pituitary every \( \frac{1}{2} \) hour until labour pains begin. Once the shock has passed off the chief danger is over.

If, as in some cases, the patient's condition does not improve under rest and morphia they advise Caesarean section. They say that in all their cases the uterus has responded to treatment and contracted down after the section has been performed. Most authorities are agreed that after section one sometimes finds an uterus that remains absolutely flaccid in spite of all efforts to make it contract. And for this they advise hysterectomy.

Prof. Kynoch at a meeting of the Ed. Obstet. Society on the 13th of Jan. 1926 described a case of his which rather gave support to the views of Hewitt and Cameron that the uterus will contract down on sufficient stimulation being given. He had a case of severe concealed haemorrhage. On opening the abdomen he found the uterus haemorrhagic and showing purplish patches. He performed Caesarean section. The uterus was flaccid. He stimulated it with hot towels and with an injection of pituitary. After ten minutes the uterus contracted down. He thus was able to leave the uterus in situ.

He quoted statistics of the mortality rate following/
following the two operations

Caesarean Section alone  -  11%
Caesarean Section with Hysterectomy  46%

He was probably quoting from a paper by Eardley Holland which appeared in the Journal of Obstetrics and Gynaecology 1921, Vol. 28. The title of this paper was - "The Results of a Collective Investigation in Caesarean Section performed in Great Britain and Ireland from the year 1911 to 1920 inclusive".

The writer, Eardley Holland, collected details of 66 cases.

These cases were classified as follows

8 mild accidental haemorrhage

\[
\begin{align*}
66 & \quad 49 \quad \text{"Concealed".} \\
58 & \quad 9 \quad \text{"Concealed and revealed".}
\end{align*}
\]

Of the 66 cases

Maternal Mortality of 18 or 27%

Of the 58 cases

Maternal Mortality 17 or 29%

Of the 8 cases

Maternal Mortality 1 or 12%

Whole Group.
(1) **Caesarean Section**  
36 cases 4 deaths 11%  

**Caesarean + Hysterectomy**  
30 " 14 " 46.6%  

(2) "Concealed" and "Concealed and Revealed cases".  

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Cases</th>
<th>Deaths</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Caesarean Section</td>
<td>30</td>
<td>4</td>
<td>13%</td>
</tr>
<tr>
<td>Caesarean + Hysterectomy</td>
<td>28</td>
<td>13</td>
<td>46%</td>
</tr>
</tbody>
</table>

The mortality in severe cases of concealed haemorrhage will always be high whatever operative treatment is carried out. Eardley Holland says that according to the records of the two groups of cases - those upon whom Caesarean section was performed and those where Caesarean section was combined with hysterectomy, the degree of severity of the condition before operation was about the same.

From these figures then it is seen that the mortality following the **combined operation** is about four times as great as that of simple Caesarean section.

It has been found that in about 4% of cases in which a simple Caesarean section for one or other indication has been performed, rupture of the uterine scar is apt to occur in a subsequent pregnancy. How much more likely would this accident be liable to occur in cases where the uterus has been the seat of multiple areas of necrosis and haemorrhage.

One/
One has found one record of a case of rupture of the uterus following Caesarean section for concealed haemorrhage. This is reported in the Proceedings of the Royal Society of Medicine 1920 Vol.XIV Obstet. and Gyn. Section p.74. The details of the first operation are few. Eleven months after the operation, when the patient was seven months pregnant the uterus ruptured. The rupture was along the scar of the previous operation.

However in operating on a case of accidental haemorrhage all one's efforts must go towards saving the mother’s life. The question of probabilities in a future pregnancy does not arise.

The Dublin School is very much against drastic operative treatment of concealed accidental haemorrhage cases. Bethel Solomons, the present Master of Rotunda Hospital in an article "Caesarean Hysterectomy" appearing in the British Medical Journal March 29th 1930 says:

"We have arrived at the far more successful results which follow the simple methods at present in use at the Rotunda, and we are of the absolute and dogmatic opinion that while there may be a minute place for Caesarean section there is no place for Caesarean hysterectomy in the treatment of accidental haemorrhage."

There/
Their method of treatment is much the same as described under external haemorrhage. They rupture the membranes, plug the vagina and give pituitary.

The following are the results obtained at the Rotunda Hospital from 1910–11 to 1918–19 (Series I.) plugging was the principal treatment. There were 51 cases with 8 deaths:

- 16 cases plugged
- 4 " Caesarean section 1 death
- 3 " Hysterectomy 2 deaths
- 28 " Palliative measures 3 "

In the extern practice there were

33 cases with 6 deaths (4 cases were plugged)

The treatment was mostly palliative.

Results:

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palliative treatment</td>
<td>10.7%</td>
</tr>
<tr>
<td>Plugging</td>
<td>12.5%</td>
</tr>
<tr>
<td>Caesarean Section</td>
<td>25%</td>
</tr>
<tr>
<td>Hysterectomy</td>
<td>66%</td>
</tr>
</tbody>
</table>

In the nine years of the first series of cases there were 33,000 confinements and 8 deaths.

At the end of this period Gibbon FitzGibbon decided to follow strictly palliative measures.
In a second series of cases extending over six years there were 3 deaths.

22,000 confinements and 3 deaths in 6 years or corrected to 9 years 4.5 deaths.

He had a series of 64 cases with 3 deaths. One death followed hysterectomy. He did not see this case. Probably he would have advised against the operation of hysterectomy.

Munro Kerr agrees that in severe cases of accidental haemorrhage the high maternal mortality following abdominal section makes it imperative that the more conservative treatment should be followed.
SECTION II. A.

A DETAILED RECORD OF 100 CASES OF ACCIDENTAL ANTEPARTUM HAEMORRHAGE.

The first column gives the year during which the case occurred and also its number in the hospital register.

The next column gives the number of the case in the series of one hundred cases investigated for this paper.

Then the initials of each patient are given and following these her age.

The column headed "Pregnancy and Abortion" gives first the number of previous pregnancies. Then in those cases where a history of abortion has been recorded this fact is noted, and where date alone, the stage of pregnancy at which it occurred, for example:

Case No.3 shows that the patient had had three previous/
INTRODUCTION.

As already noted the cases recorded occurred at the Royal Maternity and Simpson Memorial Hospital during the period January 1st 1923 and February 22nd 1927.

Clinical details of each case have been noted in columns.

The first column gives the year during which the case occurred and also its number in the hospital register.

The next column gives the number of the case in the series of one hundred cases investigated for this paper.

Then the initials of each patient are given and following these her age.

The column headed "Pregnancy and Abortion" gives first the number of previous pregnancies. Then in those cases where a history of abortion has been recorded this fact is noted, and where data allow, the stage of pregnancy at which it occurred.

For example:

Case No.5 shows that the patient had had three previous/
previous pregnancies. The third pregnancy ended in an abortion at 5\(\frac{1}{2}\) months.

In some cases the number of previous pregnancies is noted but no history of these pregnancies is given. In some of these cases abortions may have occurred. Where these details are lacking the letters N.N. have been used as an abbreviation for "Not Noted".

The next column gives the fact of presence or absence of albumin in the urine. In some cases no examination of the urine has been made. This is noted. In practically no case has a quantitative estimation of albumin been made. The amount of albumin present is noted as a "trace" or as +, ++, or +++.

The next column deals with the factor of trauma.

Then the full length of the umbilical cord in each case is given in inches. In some cases the cord has not been measured.

Then signs and symptoms of toxaemia are recorded. In investigating some of the cases it has been difficult to decide whether some of the signs and symptoms are the evidence of a pathological or a physiological state. For example: many pregnant women have oedema of the legs and feet towards term. The difficulty is to decide at what stage is this oedema/
oedema evidence of a toxic state.

In this column all evidence of a possible toxic state has been noted, e.g. excessive morning sickness, constipation, severe or frequent headaches, visual disturbances, and oedema. All the facts have been considered and then a decision has been made in each case as to whether or not the patient was in a state of toxæmia.

The next column gives the week of pregnancy during which the bleeding occurred.

Then the presence or absence of abdominal pain in relation to the bleeding is noted.

The type of bleeding is then given. The abbreviations:

Ext. for external
C. for concealed
and E. & C. for combined haemorrhage being used.

The column headed "Treatment" requires no explanation.

The next column gives the effect of the pregnancy and its accompanying pathological condition upon the mother and upon the child. The following abbreviations are used.
L. for living
S.B. for still-born
and D. for died.

Where the child has not been delivered in hospital this is noted.

The final column is an epitome of each case. All particulars of importance or of interest that have not been given in the previous columns are noted. For example where post-mortem examinations have been made the findings are recorded. In many cases the placenta is described. In most cases the history of the labour is given.
<table>
<thead>
<tr>
<th>No.</th>
<th>Case No.</th>
<th>Name</th>
<th>Age</th>
<th>Pregnancy &amp; Abortion</th>
<th>Abdomal Pain Type</th>
<th>Treatment</th>
<th>Result</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>147</td>
<td>2 F.V.</td>
<td>22</td>
<td>0</td>
<td>N.N. 21</td>
<td>External Packed.</td>
<td>L.</td>
<td>L.</td>
<td>N.I abnormal till 2 weeks before bleeding had downward pain for 1/2 hour. Bleeding on admission. Cervix I finger dilated. Much bleeding after exam; Vaginas packed. Later Membranes were ruptured, Scopolamine &amp; Morphine. Med. forceps. Placenta infarcted. Large retro-placental clot.</td>
</tr>
<tr>
<td>350</td>
<td>3 M.M.</td>
<td>25</td>
<td>1</td>
<td>N.N. 18</td>
<td>External N.I.</td>
<td>L.</td>
<td>S.B.</td>
<td>Brisk vaginal bleeding before and after admission. Uterus was tender. Patient looked ill. So an internal podalic version was performed and a leg was brought down. Large R-P clot. No F.P. on child. Mother's pulse only 85.</td>
</tr>
<tr>
<td>612</td>
<td>6 J.B.</td>
<td>22</td>
<td>1</td>
<td>N.N. 18</td>
<td>External N.I.</td>
<td>L.</td>
<td>L.</td>
<td>Patient began to bleed while asleep. Bleeding stopped. Same evening went into labour. Normal delivery. Placenta showed a small area of older blood clot.</td>
</tr>
<tr>
<td>Case Name, Age, &amp; Abortion</td>
<td>Pregnancy &amp; Abortion</td>
<td>Abdominal Pain</td>
<td>Type</td>
<td>Treatment</td>
<td>Result</td>
<td>Notes</td>
<td></td>
<td></td>
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<tr>
<td>---------------------------</td>
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<td>--------</td>
<td>-------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.D. 36</td>
<td>1-</td>
<td>Nil 40</td>
<td>Ext.</td>
<td>Spontaneous</td>
<td>L. S.B.</td>
<td>Case record missing.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>J.F. 27</td>
<td>0</td>
<td>Nil</td>
<td>E.S.O.</td>
<td>Eclamptic treatment</td>
<td>L. S.B.</td>
<td>Five history of tuberculosis. Patient had tuberculosis treatment 13 yrs. before. Also had tuberculosis inmates. Patient has felt ill throughout pregnancy. Severe ext. haemorrhage. Patient admitted. Memb. rupt. at 10.20p.m. Labour 3:25a.m. Excessive bleeding during 3rd stage. Large retro-placental clots followed the birth of the placenta.</td>
<td></td>
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</tr>
<tr>
<td>1923</td>
<td>14 M.H. 35</td>
<td>6-</td>
<td>N.N.</td>
<td>16</td>
<td>Nil</td>
<td>32</td>
<td>Nil</td>
<td>Ext. Nil req.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Reg.No.</th>
<th>Case No.</th>
<th>Name</th>
<th>Age</th>
<th>Regrettably</th>
<th>Pregnancy &amp; Abortion</th>
<th>Alb.</th>
<th>Trauma</th>
<th>Cord Ins.</th>
<th>Toxie Symptoms</th>
<th>Week of Progress</th>
<th>Abdominal Pain</th>
<th>Type</th>
<th>Treatment</th>
<th>Result</th>
<th>Mother</th>
<th>Child</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1924. 487</td>
<td>23</td>
<td>D.</td>
<td>34</td>
<td>929</td>
<td>41st at 10th week</td>
<td>N.N.</td>
<td>N.N.</td>
<td>Nil</td>
<td>33</td>
<td>During bleeding</td>
<td>Ext. Rest</td>
<td>L</td>
<td>L</td>
<td>Undelivered</td>
<td>Wassermann Neg.</td>
<td>Three mths before admission had a haemorrhage. Stayed in bed for a week. Three days before admission pain in back &amp; further loss of blood. Admitted to hospital. Treatment - Nil other than rest. No further bleeding. Patient determined to leave hospital. No record therefore of birth.</td>
<td></td>
</tr>
<tr>
<td>663</td>
<td>25</td>
<td>N.M.</td>
<td>24</td>
<td>0</td>
<td>0</td>
<td>++</td>
<td>N.N.</td>
<td>18</td>
<td>39</td>
<td>For 1 month swelling of hands &amp; Ect also headache &amp; constipation</td>
<td>Bleeding pain</td>
<td>Ext.</td>
<td>Mil req.</td>
<td>L</td>
<td>L</td>
<td>Admitted because of bleeding. Spont. delivery twelve hours later.</td>
<td></td>
</tr>
<tr>
<td>677</td>
<td>27</td>
<td>M.B.</td>
<td>36</td>
<td>5</td>
<td>5-</td>
<td>+</td>
<td>N.N.</td>
<td>26</td>
<td>33</td>
<td>Morning sickness throughout pregnancy</td>
<td>Before &amp; bleeding</td>
<td>Ext.</td>
<td>Packing</td>
<td>L</td>
<td>S.B.</td>
<td>Admitted 10-45p.m. That morning at 10a.m. abdominal pain. At 1p.m. pain and bleeding. Bleeding continued. Abdomen tender. Labour 7 p.m.</td>
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<tr>
<td>915</td>
<td>28</td>
<td>G.O.</td>
<td>35</td>
<td>2</td>
<td>N.N. N.N.</td>
<td>19</td>
<td>N.N.</td>
<td>19</td>
<td>34</td>
<td>Nil save possibly a few headaches</td>
<td>E.N.</td>
<td>Ext.</td>
<td>Mil req.</td>
<td>L</td>
<td>S.B.</td>
<td>First labour following a fall was premature. Child S.B. at 6th month. Present pregnancy: Bleeding about 2 pints at 10-30a.m. Deliver at 3-10p.m.</td>
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<tr>
<td>929</td>
<td>29</td>
<td>E.V.</td>
<td>35</td>
<td>5</td>
<td>5 last at 4½ mths.</td>
<td>+</td>
<td>N.N.</td>
<td>15½</td>
<td>27</td>
<td>Good deal of morn. sickness but nil else</td>
<td>Nil</td>
<td>Ext.</td>
<td>Mil req.</td>
<td>L</td>
<td>L</td>
<td>3rd Preg. aborted at 5th mth. Had a fall. 5th Preg. 7th mth. Plac. Praevia. Present pregnancy. Admitted 8:30 p.m. At 4 p.m. previous day red discharge with pain. No loss after admission. 7 days later spont. delivery. Memb. ruptured spont. altho' noted under treatment in R's report. Placenta with many infarcts. it was adherent, removed manually. Wassermann - Neg.</td>
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<tr>
<td>975</td>
<td>30</td>
<td>M.M.</td>
<td>23</td>
<td>0</td>
<td>++ N.N.</td>
<td>13</td>
<td>Nils</td>
<td>36</td>
<td>33</td>
<td>Nil abdom. Ext. Packing very tender</td>
<td>L</td>
<td>Ext.</td>
<td>Packing</td>
<td>S.B.</td>
<td>Three hours before admission patient had a very profuse bleeding. Abdomen tender to touch. Sameday gave birth to a macerated foetus. For two weeks following labour had severe vomiting. During pregnancy had been taking ergot as an abortifacient. The vomiting was probably due to a resulting gastritis.</td>
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<tr>
<td>Reg. No</td>
<td>Case Name, Age, No.</td>
<td>Pregnancy &amp; Abortion</td>
<td>Alb. Trauma</td>
<td>Cord in Ins.</td>
<td>Toxic Symptoms</td>
<td>Week of Freg.</td>
<td>Abdominal Pain</td>
<td>Type</td>
<td>Treatment</td>
<td>Result</td>
<td>Mother Child</td>
<td>Notes</td>
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<tr>
<td>1080</td>
<td>1011</td>
<td>32 J.Me 30</td>
<td>1 at 3rd mt. between 4th &amp; 5th child</td>
<td>N.N.</td>
<td>13 Nil</td>
<td>27</td>
<td>Nil Ext.</td>
<td>Packing Lower ut. sect.</td>
<td>L. D.</td>
<td>Slight oozing for 10 days before delivery. Labour induced with pituitary. No result so lower uterine segment packed. Delivered a living child but it died 3 hours later.</td>
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<tr>
<td>1179</td>
<td>1179</td>
<td>34 V.C. 27</td>
<td>1</td>
<td>N.N.</td>
<td>21</td>
<td>36</td>
<td>N.N. Ext.</td>
<td>Internal ver-L. seq. Foot brought down</td>
<td>L. L.</td>
<td>Treated by own doctor for albuminuria. The uterus was very hard - did not relax. It was not very tender. Shoulder presentation. Following labour patient developed a pyelitis.</td>
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<tr>
<td>1334</td>
<td>1334</td>
<td>36 M.Me 27</td>
<td>1</td>
<td>N.N.</td>
<td>22</td>
<td>38</td>
<td>Nil E.&amp;C.</td>
<td>Membranes ruptured</td>
<td>L. S.B.</td>
<td>Patients appearance, together with the increased pulse &amp; hardness of the uterus suggested &quot;concealed&quot; haemorrhage as well as external. The membranes were ruptured but very little blood escaped.</td>
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<tr>
<td>110</td>
<td>J.G. 34</td>
<td>1-</td>
<td></td>
<td>N.N.</td>
<td>18</td>
<td>Gedema of hands &amp; ankles lat, Blurred vision lat. Freq. of micturition</td>
<td>35</td>
<td>Thro' preg. slight, Nil with bleeding</td>
<td>Ext. Memb. Rupt.</td>
<td>L. L.</td>
<td></td>
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<tr>
<td>130</td>
<td>C.H. 34</td>
<td>8-</td>
<td></td>
<td>N.N.</td>
<td>12</td>
<td>Nil</td>
<td>38</td>
<td>Nil until labour</td>
<td>Ext. Memb. Rupt. &amp; packed before admission</td>
<td>L. L.</td>
<td></td>
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<tr>
<td>143</td>
<td>A.B. 22</td>
<td>1-</td>
<td></td>
<td>N.N.</td>
<td>15</td>
<td>Nil</td>
<td>36</td>
<td>For some hours prior to bleeding</td>
<td>Ext. Memb. Rupt.</td>
<td>L. S.B.</td>
<td>Large Retro-placental clots</td>
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<tr>
<td>179</td>
<td>M.R. 25</td>
<td>0</td>
<td></td>
<td>N.N.</td>
<td>18</td>
<td>Nil</td>
<td>28</td>
<td>Nil with bleeding</td>
<td>Ext. Memb. &amp; other than sedative</td>
<td>L. D.</td>
<td></td>
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<tr>
<td>45</td>
<td>R.D. 26</td>
<td>2-</td>
<td></td>
<td>N.N.</td>
<td>19</td>
<td>Slight oedema of rt. leg, slight visual disturbance</td>
<td>N.N.</td>
<td>Ext. Bipolar pediale version</td>
<td>Ext. Memb. &amp; foot brought down</td>
<td>L. L.</td>
<td>*N.B. Undelivered as Accidental Haem: Patient was treated as Accidental Haem: was discharged but later readmitted and found to be a placenta previa. When first in Hospital treated with rest and sedatives. Albumen on day of delivery,</td>
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<tr>
<td>64</td>
<td>T.R. 24</td>
<td>0</td>
<td></td>
<td>N.N.</td>
<td>16</td>
<td>For last 2 yrs, slight visual disturbance. Also very constipated</td>
<td>Rt. side for 2 mts.</td>
<td>Ext. Memb. &amp; req.</td>
<td>L. D.</td>
<td>Slight external haemorrhage. P.M. child - &quot;Prematurity and Cerebral Haemorrhage&quot;.</td>
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<tr>
<td>89</td>
<td>H.N. 32</td>
<td>6-</td>
<td></td>
<td>N.N.</td>
<td>29</td>
<td>Gedema feet &amp; leg for last 2 yrs.</td>
<td>35</td>
<td>For 2 mts. severe just before bleeding</td>
<td>Ext. Memb. Rupt.</td>
<td>L. L.</td>
<td></td>
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</table>

Notes:
- Although noted in Registrar's report as Accidental Haemorrhage the case record shows that there was a marginal placenta previa. No Wassermann taken. Case noted as "Ext. & Concealed" but no details in the case justify "Concealed".
- Bleeding said to be "free before delivery". In Registrar's report type said to be external and concealed, but in record no evidence of "concealed."
- Large Retro-placental clots. P.M. child - Prematurity and Asphyxia.
- Child premature. Lived 24 hours. Bleeding on am off for 42 days. Wassermann Neg.
- "N.B. Undelivered as Accidental Haem: Patient was treated as Accidental Haem: was discharged but later readmitted and found to be a placenta previa. When first in Hospital treated with rest and sedatives. Albumen on day of delivery, "
- Slight external haemorrhage. P.M. child - "Prematurity and Cerebral Haemorrhage".
- Frighteningly excessive vomiting 6th A.P.H. 3 days before delivery lasting one day.
- Large Retro-sclerotic clot. Patient epileptic. Frequent fits especially during last 2 mts. Has had 8 S.B. children. P.M. child - cerebral Haemorrhage, complete abortion at 6 months. Wassermann negative. Bleeding Ext. on admission. No notes as to abdominal examination.
<table>
<thead>
<tr>
<th>Reg. No.</th>
<th>Case Name, Age &amp; Abortion</th>
<th>Pregnancy &amp; Abortion</th>
<th>Alb. Trauma in Inc.</th>
<th>Cord in Inc.</th>
<th>Toxic Symptoms</th>
<th>Week of Prog.</th>
<th>Abdominal Pain</th>
<th>Type</th>
<th>Treatment</th>
<th>Result Mother Child</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1176</td>
<td>A.W. 17</td>
<td>0 - N.H.</td>
<td>37</td>
<td>Nil</td>
<td>Ext. Nil req.</td>
<td>L.</td>
<td>Not *</td>
<td>*Later re-admitted and delivered of a living child, delivered Three weeks before re-admission had one sudden vaginal haemorrhage. Nil after.</td>
<td></td>
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</tr>
<tr>
<td>1491</td>
<td>L.S. 32</td>
<td>3- Washing clothes Not delivered</td>
<td>38</td>
<td>Nil</td>
<td>Ext. Nil req.</td>
<td>L.</td>
<td>Not *</td>
<td>*Later re-admitted and delivered of a living child, delivered Three weeks before re-admission had one sudden vaginal haemorrhage. Nil after.</td>
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</tr>
<tr>
<td>1665</td>
<td>A.D. 30</td>
<td>2, 1 at 3rd month + N.H. Kept sterile</td>
<td></td>
<td>Severe during bleeding</td>
<td>34</td>
<td>E.&amp;C. Nil req.</td>
<td>L.</td>
<td>S.B.</td>
<td>Six days before delivery slight vaginal haemorrhage with fainting. Admitted same day. P 100 B.P.17/90 P.M. Macerated foetus. Asphyxia from Accidental Haemorrhage (concealed &amp; External)</td>
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</tr>
<tr>
<td>Reg.No.</td>
<td>Case Name, Age, Abortion</td>
<td>Pregnancy &amp; Trauma</td>
<td>Weeks Before Ad-Delivery</td>
<td>Cord in Inc.</td>
<td>Toxic Symptoms</td>
<td>Week of Pregnancy</td>
<td>Abdominal Pain</td>
<td>Type</td>
<td>Treatment</td>
<td>Result</td>
<td>Notes</td>
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<tr>
<td>1960</td>
<td>L.W. 27 4- - N.N. 16 Mil 30</td>
<td>With bleeding</td>
<td>Ext. Rest</td>
<td>L. L.</td>
<td>Case record missing.</td>
<td></td>
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</tr>
<tr>
<td>265</td>
<td>A.N. 31 3- - N.N. 17 Mil 36</td>
<td>N.N., Ext. Mil req.</td>
<td>L. S.B.</td>
<td>Patient was admitted moribund. P.110. Bleeding began with pains. The treatment was as given on the opposite column. Death 3 hrs. after admission. The P.M. showed a generalized septic condition. A possible focus of infection was the mouth. The teeth were all in a bad state of decay. The placental site showed a large clot. This case could be taken as an example of accidental haemorrhage being caused by toxaemia.</td>
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<tr>
<td>315</td>
<td>B.M. 26 1st at 6 1/2 mts. + N.N. 18 Mil 36</td>
<td>Mil with bleeding</td>
<td>Ext. Mil req. save rest</td>
<td>L. L.</td>
<td>This case does not appear to be really one of A.P.H. She had bleeding for 1 day after 2 mts amenorrhoea. A month later she had free vaginal haemorrhage for 3 hours followed by oozing of blood for one week. No bleeding after this. Thus all bleeding occurred before the child was viable. Admitted for labour. No life felt for 2 weeks. Spent, delivery of Macerated fetus. Premature Exits. Cause of death not evident as no placenta was sent for exam; No evidence of R.P. clots. Case is one of &quot;Threatened Abortion&quot;.</td>
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On day of admission patient had a sudden painless haemorrhage. Rested in hospital for 2 weeks. No further bleeding. Went home. Re-admitted 3 wks. later. Spontaneous delivery. Mother & child well. No notes are given as regards the condition of the placenta.
<table>
<thead>
<tr>
<th>Reg. No.</th>
<th>Case Name</th>
<th>Age</th>
<th>Pregnancy &amp; Abortion</th>
<th>Alb.</th>
<th>Trauma in Ins.</th>
<th>Cord Symptoms</th>
<th>Toxic</th>
<th>Week of Pregnancy</th>
<th>Abdominal Pain</th>
<th>Type</th>
<th>Treatment</th>
<th>Mother</th>
<th>Child</th>
<th>Notes.</th>
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</thead>
</table>

Normal pregnancy until just before admission when she had a slight vaginal bleeding. On admission fairly free from bleeding. On examination uterus was fairly firm & tender. Cervix fully dilated. Membr. ruptured. Bleeding stopped. Spont. delivery. Large R.F. clots were found at lower portion of the placenta. Puerperium Normal.


Slight bleeding on day of admission. Labour induced. Nil else to note.

Sent in at 1 a.m. because on previous evening had has bleeding. On admission on one finger dilated. No Fetal E. heard. Patient having pains. Still bleeding. At 5 a.m. clots passed. Membranes were ruptures. Hand pro- lapsed. Pushed back but returned. Bleeding stopped. Bider & pad applied. At 7:30 a.m. spont. delivery. Appearance of placenta "suggested" accidental haemorrhage.

Well until 8.30p.m. on evening of admission. Then had a painless loss of blood. Admitted at 9.15p.m. Anaesthetic. Exam. No placenta felt. G8 admitted one finger. The cervical canal and vagina were then packed with gauze. Next morning Pituitrin was given. From 10.15a.m. to 1.45 p.m. 30c. Then stopped. At 2p.m. pack removed & douch given. G8 admitted one finger. Pains every 5-10 min. However these ceased. No further pain or bleeding. Discharged three days later. Re-admitted 2 days. Labour and puerperum normal.
<table>
<thead>
<tr>
<th>Reg.No.</th>
<th>Case Name, Age &amp; Abortion</th>
<th>Pregnancy &amp; Abortion</th>
<th>Alb.</th>
<th>Trauma</th>
<th>Toxic Symptoms</th>
<th>Week of Preg.</th>
<th>Abdominal Pain</th>
<th>Type</th>
<th>Treatment</th>
<th>Result</th>
<th>Mother Child</th>
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<tbody>
<tr>
<td>444</td>
<td>C.P. 37</td>
<td>0</td>
<td>+ N.N.</td>
<td>21</td>
<td>Nil</td>
<td>36</td>
<td>Pain with bleeding</td>
<td>Ext.</td>
<td>Packing Induction</td>
<td>L. S.B.</td>
<td></td>
</tr>
<tr>
<td>76</td>
<td>E.D. 32</td>
<td>1</td>
<td>+ N.N.</td>
<td>15</td>
<td>Nil</td>
<td>32</td>
<td>Severe Pain</td>
<td>C.</td>
<td>Caesarian Section</td>
<td>L. S.B.</td>
<td></td>
</tr>
<tr>
<td>79</td>
<td>E.G. 37</td>
<td>2</td>
<td>+ N.N.</td>
<td>N.N.</td>
<td>Nil</td>
<td>36</td>
<td>Mil</td>
<td>E &amp; C.</td>
<td>Mil req.</td>
<td>L. D.</td>
<td></td>
</tr>
<tr>
<td>80</td>
<td>C.M. 27</td>
<td>4</td>
<td>+ N.H.</td>
<td>Kept sterile</td>
<td>Sickness</td>
<td>40</td>
<td>Pain with bleeding</td>
<td>C.</td>
<td>Caesarian Section</td>
<td>D. S.B.</td>
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Case Record Missing.

Week before admission had a sudden painless haemorrhage lasted few minutes. Five days later a second Haemorrhage - more severe. Admitted. No placenta felt. Rest for a week. Then another slight bleeding. Then sixteen days after admission normal labour.

All well till day of admission 10-4-26. At 5a.m. had severe & continuous abdominal pain. Admitted 11a.m. P.84, T.98°. No Ext. bleeding. Uterus tender. At 11.20a.m. severe pain & general collapse. Uterus very tender & in notes said to be "boggy" (?) At Noon Caesarian Section. Uterine wall normal in colour. On section large R.F. clots. Placenta lying free. Uterus contract-ed down well. Pituitary 3cc given. Good recovery. P.M. Child - death due to asphyxia. Placenta showed rashed fibrosis with 2 large areas where there had been R.F. haemorrhages. Several placental infarcts present.

Case record missing.

During last 4 mts. of preg. pain in region of fundus. Admitted 8.5.26 at 5.30p.m. At 10a.m. onset of labour. Slight bleeding at noon & another at 2p.m. No bleeding on admission. On Exam. tenderness over Rt. fundal area. Following day spent. delivery. Small tumour felt in fundal region. Probably subperitoneal fibroid. Still felt on discharge from hospital. Placenta - No infarct where but large R.F. clots. Child died 7 days after delivery. "Has never done well."

At 12 noon day of admission sudden haemorrhage followed by severe abdominal pain. Bleeding continued. Vagina packed at 8p.m. Not successful. Caesarian section at midnight. Uterus appeared normal. Placenta separated off in uterus. Mother died from peritonitis 16 days later. P.M. General peritonitis. Anterior wall of uterus was necrotic & much pus was present. P.M. child - asphyxia from placental infarction.
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</thead>
<tbody>
<tr>
<td>19575</td>
<td>H.P. 38</td>
<td>9-</td>
<td>+ N.N.</td>
<td>Kept sterile</td>
<td>Nil</td>
<td>Pain before E.C. Nil req. &amp; with bleeding</td>
<td>L. S.B.</td>
<td></td>
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</tr>
<tr>
<td>797</td>
<td>E.R. 32</td>
<td>4. 1 at 6 wks.</td>
<td>+ N.N.</td>
<td>Kept sterile</td>
<td>Nil</td>
<td>Pain fol-owed E.C. Caesarian Section</td>
<td>L. S.B.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>820</td>
<td>J.R. 34</td>
<td>2. 2 at 2 mts.</td>
<td>+ N.N.</td>
<td>12</td>
<td>Nil</td>
<td>Nil</td>
<td>Ext. Packing</td>
<td>L. S.B.</td>
<td></td>
<td></td>
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<tr>
<td>880</td>
<td>J.S. 22</td>
<td>2. 1 at 6 mts.</td>
<td>+ See Notes</td>
<td>20</td>
<td>Sickness</td>
<td>N.W.</td>
<td>Ext. Nil req.</td>
<td>L. S.B.</td>
<td></td>
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<tr>
<td>1904</td>
<td>W. 32</td>
<td>5.2nd at 4th mt.</td>
<td>+ N.N.</td>
<td>Not delivered</td>
<td>Nil</td>
<td>Nil</td>
<td>Ext. Rest</td>
<td>L. Under- liv- ered</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1934</td>
<td>M.A. 38</td>
<td>12-</td>
<td>- N.N.</td>
<td>Slit. oedema</td>
<td>40</td>
<td>Nil</td>
<td>Ext. Nil req.</td>
<td>L. S.B.</td>
<td></td>
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</tbody>
</table>

For some weeks continuous pain in lower abdomen, at 10.30 a.m. day of admission pain worse & then passed a little blood & clots. Two joc's of Pituitary given at 4.30 p.m. & 6 p.m. Admitted 3.30 p.m. No further haemorrhage. At 10 p.m. abdomen tender. At 1 a.m. spontaneous delivery almost precipitate. Child S.B. macerated. P.M. Astphxia from separation of placenta. Discharged 15 days after labour. Had a temperature for some days for which no cause could be found. Wassermann - Neg.


Patient woke in bed & found she was bleeding. Her doctor packed the vagina & she was admitted to hospital. Fock removed. Placenta not felt. Douched. Spontaneous delivery. Child S.B. & macerated. P.M. Greater part of placenta fibrosed. A few infarcts. Cause of death of child "Astphxia following fibrosis of the placenta".

Admitted to R.I.M. with fracture of right tibia & fibula went into labour. Slight bleeding. Admitted to Simpson. Same day delivered of a S.B. foetus. Some clots preceded the placenta. No retro-placental clots.

Fairly good health until 5 weeks before admission. Then had a considerable amount of vaginal bleeding & passed a large clot. Bleeding again a week before admission. Bleeding continued for 5 days. Two days later admitted. Seven days later slight coming again. Fourteen days later - resting in bed - no further bleeding. Patient discharged. Wassermann - Neg.

<table>
<thead>
<tr>
<th>Reg.No.</th>
<th>Case Name, Age, No.</th>
<th>Pregnancy &amp; Abortion</th>
<th>Alb. Trauma</th>
<th>Cord In Ins.</th>
<th>Toxic Symptoms</th>
<th>Week of Preg.</th>
<th>Abdominal Pain</th>
<th>Type Treatment</th>
<th>Result</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1487</td>
<td>93 B. 27 3 1 at 2 mts.</td>
<td>+ See Notes Not delivered</td>
<td>Nil</td>
<td>30 With bleeding Ext.</td>
<td>Rest, Morphia L.</td>
<td>Not delivered</td>
<td>? Pyelitis or albuminuria with last two pregnancies. For years has had precipitous micturition. Well until 4 days before admission. Then fell heavily and bruised the left buttock. Two days later severe dysuria &amp; down bearing sensation. Two days after this labour pains &amp; loss of blood. On admission P.96. T.97°, B.P.115/55. Urine 24hrs specimen 1100cc. Urea N. 520Mg%. NH3 34. Rest for 8 days. No return of bleeding. Discharged.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1923</td>
<td>95 I.N. 19 0</td>
<td>+ N.N. 33</td>
<td>Nil</td>
<td>33 No pain with bleeding Ext.</td>
<td>Rest L.</td>
<td>Not delivered</td>
<td>Slight Antepartum haemorrhage 3 days before admission. Sudden painless loss of about a tablespoon of blood. Has not been repeated. Following day much vomiting. Kept in Hospital for 5 days. No recurrence of vomiting or bleeding so discharged.</td>
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<tr>
<td>Reg.No.</td>
<td>Case Name.</td>
<td>Age.</td>
<td>&amp;</td>
<td>Alb.</td>
<td>Trauma</td>
<td>Cord</td>
<td>Toxic</td>
<td>Symptoms</td>
<td>Abdominal</td>
<td>Pain</td>
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<tr>
<td>1927</td>
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<td></td>
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<tr>
<td>68</td>
<td>96 C.</td>
<td>26</td>
<td>0</td>
<td>N.N.</td>
<td>Possibly</td>
<td>Not</td>
<td>Swelling</td>
<td>deliv.of hands &amp; feet</td>
<td>Nil</td>
<td>Ext.</td>
</tr>
<tr>
<td>242</td>
<td>99 J.M.</td>
<td>18</td>
<td>0</td>
<td>N.N.</td>
<td>Nil</td>
<td>38</td>
<td>Nil with bleeding till labour</td>
<td>Ext. Memb.,Rupt.</td>
<td>L.</td>
<td>L.</td>
</tr>
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SECTION II. B.

AN ANALYSIS OF THE RECORDS.

INTRODUCTION.

In making the following analysis the records of one hundred consecutive cases occurring at the Edinburgh Royal Maternity Hospital and classified there as Accidental Antepartum Haemorrhage have been considered. These cases occurred during the years 1923 to 1926 inclusive. Five case records from 1927 were taken to complete the series of one hundred cases.

Case records were missing in eleven cases; but some of the required details were obtained from the Registrar's report.

One case No. 65 is classified in the Registrar's report as Accidental Haemorrhage, but it is really a case of threatened abortion as all the bleeding occurred before the child was viable. The case is not considered in the analysis.

Case No. 42 is one of Accidental Haemorrhage and Marginal Placenta Praevia. It is considered in the analysis.

Case No. 46 is one of Placenta Praevia. This patient developed
developed an albuminuria, a fact that bears relation to Dr James Young's theory of placental toxaemia. Details of this case have not been considered in the analysis.

Cases 42 and 46 will be discussed later in the paper.

A difficulty in making the analysis has been the fact that in many of the case records essential details have been lacking. In others there are obvious inaccuracies. In the detailed record of cases the absence of a particular fact is noted. Inaccuracies have been corrected where the given information has been sufficient.

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Information in regard to parity was obtained in 97 cases. It was lacking in one case. No. 126.

Of these patients 52 were multiparous and 27 primiparous.

These figures may be taken as percentages.

In the final part of this paper figures of parity were given for 149 cases of Antepartum

AGE INCIDENCE.

The age of the patient was obtained in 89 cases. The total ages came to 2719 years. This gives an average age of 30.55 years.

The youngest patient was 17 years old, No. 56. The eldest, 42 years old, No. 40. Both these patients were pregnant for the first time. Nothing of note is found in their case record.

Ley in his series of 50 cases found the average age incidence to be 31.6 years. His youngest case was 19 years old, and the eldest, 43 years.

PARITY INCIDENCE.

Information in regard to Parity was obtained in 97 cases. It was lacking in one case, No. 100.

Of these patients, 72 were Multiparae and 25 Primiparae.

These figures may be taken as percentages.

In the first part of this paper figures of parity were given for 169 cases of Antepartum haemorrhage.
haemorrhage occurring at the Edinburgh Royal Maternity Hospital during the years 1923 to 1928 inclusive.

In 39 cases of 1927 and in all the cases of 1928 the details of parity were obtained from the Registrar's report and have not been checked. It appears that in these two latter years the proportion of Multiparae to Primiparae was greater than in the previous four years. The figures for the six years work out at

78.5% Multiparae
21.4% Primiparae.

ABORTION INCIDENCE.

Facts in regard to previous pregnancies were obtained in the case of 67 Multiparae. These gave a history of 258 pregnancies. Of these pregnancies 26 ended in abortion. Unfortunately there is not sufficient information to allow one to say how many of the pregnancies were full time. One can only say

26 abortions in 258 pregnancies
or a proportion of

1 abortion to 10 pregnancies. (9.9)

If one had full details in regard to previous pregnancies and so too the number of full time labours, the abortion rate would be higher, but one doubts whether/
whether it would be as low as the normal abortion rate of 1 abortion to every 4 or 5 full time labours.

**PARITY OF MULTIPARAE.**

The total number of pregnancies was obtained in 72 cases.

This was 298

The average parity of the Multiparae therefore was 4.13

The maximum parity of these cases was 15 Case No. 38

All the previous labours in this case had been full time.
ALBUMINURIA.

Albumin was present in 53 cases.

No. 5. This patient had been treated in the Ante Natal Department for albuminuria during the pregnancy but under treatment the condition cleared up. No record is given of examination of the urine on admission. Case considered as Albumin.

No. 46. Case not considered. (Placenta Praevia with albuminuria)

Albumin was absent in 29 cases.

Examination of urine not recorded, or case record missing in 16 cases.

Thus it is found that of 82 patients with Accidental Antepartum Haemorrhage 53 had an albuminuria. This gives a percentage of 64.63%.

Whitridge Williams found the percentage to be 64.7% but this was in a series of only 17 cases.
CASES ASSOCIATED WITH ECLAMPSIA.

Case No.9. This case is noted in the Registrar's report as Pre-eclamptic. Unfortunately the case record is missing.

Cases No.11 and No.51 are definitely eclamptic.

As already stated, in 16 cases no record of the examination of the urine has been made, or else the case record is missing. If any of these patients had shown evidence of eclampsia no doubt the urine would have been examined and the results recorded. One can therefore say that approximately 3% of the cases were associated with eclampsia.

ASSOCIATION OF ALBUMINURIA WITH OTHER TOXIC SYMPTOMS.

Of the 53 cases with albuminuria other toxic symptoms were present in 19 cases.

Nos. 9, 11, 21, 25, 27, 33, 41, 42, 47, 48, 51, 52, 54, 59, 60, 80, 84, 88, and 90.

Absent in 30 cases

Nos. 5, 8, 10, 15, 18, 19, 23, 26, 29, (?) 30, 34, 36, 39, 40, 44, 53, 57, 66, 72, 73, 75, 76, 79, 81, 82, 83, 85, 93, 95, 97.
Not recorded in 4 cases

3, 20, 73, 61.

Thus one finds that in 30 cases out of 98 of Accidental Antepartum haemorrhage the only evidence of there being a toxic state on clinical examination was the presence of albumin in the urine.

This fact demonstrates the importance of a routine examination of the urine in all pregnant women.

PARITY OF CASES WITH ALBUMINURIA.

Of the 53 cases with Albumin

39 were Multiparae
14 " Primiparae

As Gordon Ley has pointed out one expects to find toxaemic conditions associated more often with primiparae than with multiparae.

In this series of cases almost three times as many multiparae as primiparae show a toxaemia.
**INFLUENCE OF TRAUMA.**

Case records were missing in 11 cases

Nos. 9, 17, 20, 51, 61, 63, 74, 77, 78, 86, 100.

In 77 cases in which records were obtained trauma played a possible part in 7 cases. In 70 cases trauma was not mentioned.

Case 5.

Here during pregnancy a transverse lie was corrected several times in the Ante Natal department. Possibly trauma played a part in causing the separation of the placenta. However the patient had also been treated for albuminuria. Also she was para 3 and gave a history of "kidney trouble" following the birth of the second child. One can say that trauma was a possible factor.

Case 13.

This patient was washing clothes when the bleeding came on. Here again however other factors seem to have played a part because at the 2nd month of pregnancy the patient had a threatened abortion.

Case 58.

This patient whilst washing clothes had a sudden vaginal bleeding. She had no albumen, and no toxæmic symptoms. Her previous pregnancies had been normal. Here trauma was probably the cause of the haemorrhage.
Case 84.

This patient was admitted to hospital with a fracture of the right tibia and fibula. She went into labour almost at once, and on the same day was delivered of a still born foetus. She had albumen in the urine. However there is little doubt that trauma was the direct cause of the accidental haemorrhage.

Case 93.

This patient fell heavily and bruised her buttock. Two days later she had a severe dysuria and down-bearing sensation. Two days still later she had labour pains and lost blood. She gave a history of possible pyelitis, or albuminuria in her last two pregnancies. Trauma probably played a part but there is evidence of an underlying cause in the history of renal trouble.

Case 96.

Three weeks before admission patient was doing some washing when a little blood escaped. It is not noted whether there was an albuminuria or not. Probably there was, because of the recorded toxic symptoms. Here trauma was probably an accessory factor.
Case 97.

Three days before the bleeding this patient fell on the ice. She also was found to have albumen.

Cases 84 and 97 suggest an interesting question.

Was the presence of albumen the result of the trauma or was it evidence of a previously existing toxaemia?

In case 84 one would not think that albumen would appear so rapidly after the trauma.

In Case 97 perhaps the trauma caused a partial separation of the placenta, and from this portion toxins were absorbed into the circulation.

**The Length of the Umbilical Cord.**

Case records were missing in 8 cases.

In 19 cases the cord was kept sterile, or the length was not noted, or the patient was not delivered.

In 69 cases the cord was measured.

Total length was 1399"

Average "  " 20.2"

The Maximum length was 34"

The Minimum  "  " 13" (No. 43 and 83)

In case 43 there is not sufficient information to/
to say whether bleeding began before labour pains. In Case 83 the condition of the placenta is sufficient to account for the separation. The patient was not in labour when she had the first attack of bleeding.

We can thus say that the length of the umbilical cord plays no part in the causation of Accidental Haemorrhage.

**PERIOD OF GESTATION.**

98 cases considered.
Total in weeks of gestation periods = 3395.
Average period

\[ = 34.6 \text{ weeks} \]

**THE FREQUENCY OF ABDOMINAL PAIN WITH ACCIDENTAL HAEMORRHAGE.**

98 cases were considered.

There were no abdominal pains, other than labour pains, in 42 cases.

There were pains, other than labour pains, before, accompanying, or after bleeding in 33 cases.
No record was made in 23 cases.

Of 7 cases of Concealed haemorrhage there was severe pain in 4 cases.

In 2 cases the records were missing.

In 1 case the presence or absence of pain was not noted.

**TYPE OF ACCIDENTAL HAEMORRHAGE.**

A difficulty arises in the examination of the records at this stage. Some of the case records have been labelled with a diagnosis which is clearly incorrect. For example No. 3 is diagnosed both on the case record and in the Registrar's report as "Concealed Haemorrhage". From the history one finds that the patient had "brisk vaginal bleeding before and after admission". Other case records on examination give nothing to justify the diagnosis of the type of Accidental Haemorrhage.

In drawing up this record one has noted the type of haemorrhage according to the diagnosis on the case record, except where the diagnosis is obviously wrong. In these latter cases a diagnosis is made from the given details.

In/
In the 98 cases considered there were:
72 cases of External haemorrhage
7 " " Combined External and Internal haemorrhage
& 6 " " Concealed haemorrhage.

In the year 1926 a large number of the cases are noted as External Haemorrhage. (27). Many of them are probably "Combined". The data are insufficient to make a diagnosis. Probably the diagnosis was made from observations that have not been recorded.

THE PRESENCE OF TOXIC SYMPTOMS WITHOUT THE PRESENCE OF ALBUMEN.

Of the 29 cases without Albumen - toxic symptoms were present in 7 cases. See Nos. 24, 37, 38, 49, 87 and 89.

There were no toxic symptoms in 18 cases
There was no record of " " 4 cases

The case records of these 18 cases were closely examined.

No. 31 Had a Wassermann +++ .
No. 62 " " " ++ .

We can say that in 16 cases there was no definite evidence of toxaemia.
What possibly could have caused the accidental haemorrhage in these 18 cases?

No. 12. Had had antepartum haemorrhage with a previous pregnancy.

No. 14. Had also had antepartum haemorrhage before. Ten months before present condition she had an "enlarged ovary" removed. One can suggest in this case an endometritis.

No. 62. As well as syphilis patient had antepartum haemorrhage in a previous pregnancy.

No. 13. Had a threatened abortion at 2nd month. Trauma too probably played a part.

No. 58. Trauma probably the cause.

This leaves 14 cases in which evidence of an aetiological factor is lacking. No. 13 is still considered because the cause of the threatened abortion was probably a factor in the accidental haemorrhage. No. 14 and No. 58 are eliminated.
TREATMENT.

No treatment, was required, other than rest in bed, careful nursing, and possibly sedatives in 35 cases.

The membranes were ruptured in 28 cases.

Of these cases a binder and pad was as well used in No. 41 and 57, and 70, and a binder in No. 97.

The vagina was packed in 12 cases.

Of these:

No. 43 was packed before admission. After admission the membranes were ruptured.

No. 54 was a breech case. External version failed.

The vagina was packed after this.

No. 60 was packed before admission. On admission the pack was removed, the membranes were ruptured and the vagina repacked.

No. 72 was also given pituitary.

No. 73 was also induced with quinine and pituitary.

The membranes were ruptured, and the vagina packed in 3 cases:

No. 7. Abdominal binder as well.

84. Pituitary and blood transfusion also.

89. Quinine, pituitary and bougie induction as well.
Forceps were used in 4 cases:

No. 2. Vagina packed, membranes ruptured and medium forceps.


No. 26. Patient exhausted. Forceps used when head was on the perineum.

No. 48. A case of Concealed haemorrhage.

The patient was collapsed. Low forceps delivery. Death from shock soon after delivery.

Internal podalic version and leg brought down . . . . Nos. 3 & 4

Podalic version and leg brought down No. 31

Membranes ruptured and foot brought down No. 5

Internal Version and foot brought down Shoulder presentation . . . . No. 34

Breech. Leg brought down . . . . No. 37

Membranes ruptured. Bipolar cephalic Version . . . . No. 50

Rest and Pituitary - No. 16

Quinine and pituitary induction Nos. 69 and 92

Eclamptic treatment - No. 11

Caesarean Section. No. 9 (Pre-eclamptic) No. 76

"Concealed". No. 80 "Concealed". No. 82 "Combined".

Caesarean Hysterectomy. No. 20 "Combined". No. 78 "Concealed".
In 98 cases of Accidental haemorrhage there were 7 maternal deaths:—

No. 4. Admitted moribund. Died from shock following delivery. No P.M.

No. 20. Case of "Combined" bleeding. Caesarean hysterectomy performed. Patient died immediately after the operation. Case record missing.


No. 54. Patient had been packed. Died seven days later from septicaemia.

No. 60. Packed. Died undelivered.

No. 64. Admitted moribund. Died 3 hours after admission. P.M. showed a generalised septic condition. See Notes.

No. 80. Vaginal packing followed by Caesarean Section. Died 16 days later from peritonitis. See Notes.

Maternal Mortality 7.14%.
INFANTILE MORTALITY.

30 children lived.
11 " were not delivered in hospital.

There was a mortality of 56. Of these cases 49 were still born and 7 died soon after birth.

Particulars of these cases will be found in the detailed record.

POST-MORTEM REPORTS ON CHILDREN.

Details will be found in cases numbered 1, 7, 11, 13, 30, 32, 41, 44, 45, 47, 48, 50, 51, 53 54, 59, 73, 76, 80, 81, 82, 83, 87.

In the majority of cases death was due to asphyxia, or to prematurity, or to cerebral haemorrhage. No.87 is an interesting case. The child had a spina bifida. See Notes.
A blood test was carried out in 12 cases. In No. 8 the mother's blood was negative but the husband's blood was +++. See Case record. In No. 31 the blood was triple positive. Here syphilis was probably an important aetiological factor. In No. 82 the mother's blood was positive, and the child's blood a weak positive. In No. 87 the blood was triple positive.

Cases Numbered 18, 23, 29, 41, 45, 50, 85, and 81 were all Negative.

Thus syphilis was present in three cases, possibly in four - No. 8, and absent in 8 cases.
ASSOCIATED CONDITIONS AND POSSIBLE AETIOLOGICAL FACTORS.

There was a history of Nephritis in 3 cases: Nos. 5, 8, and 93.

One case, No. 34, developed a pyelitis after labour.

There were 2 cases in which Tuberculosis probably played a part. Nos. 10 and 21.

Case No. 14. This patient had an ovary removed 10 months before because it was "enlarged". Probably the patient had a chronic endometritis as well. This patient had antepartum haemorrhage with her second labour.

Case No. 12. This patient had antepartum haemorrhage with the 6th pregnancy.

Case No. 18. Developed double pyosalpinx 10 days after operation. Organism probably gonococcus.

Case No. 29. Had previously had a placenta praevia.

Case No. 30. Patient had been taking an abortifacient.

Case No. 50. Patient an epileptic.
Cases Nos. 62 & 71.

Had had antepartum bleeding in previous pregnancies.

Case No. 64. This patient died of septicaemia. The focus of infection was probably the mouth. The teeth were septic. The accidental haemorrhage was probably due to toxaemia.

Case No. 79. Condition associated with a subperitoneal fibroid.

Case Numbers are given so that the Case Notes can be consulted.

PLACENTAL ABNORMALITIES.

No. 2. Placenta infarcted. Large retro-placental clots.

No. 21. Placenta very much infarcted. Large retro-placental clots.

No. 29. Placenta with many infarcts. Adherent and removed manually.

No. 73. Marked infarction. Large retro-placental clots.

No. 76. Placenta showed several infarcted areas. Also showed marked fibrosis with two large areas where there had been retro-peritoneal bleeding.
No. 80. Placenta infarcted.
No. 82. Placenta infarcted.
No. 83. Placenta showed a few small infarcts.

Greater part of the placenta was fibrosed.

Thus in eight cases definite infarction of the placenta has been noted. In two of these cases there was fibrosis.

In 13 cases retro-placental clots were noted.

There were other placental conditions noted in:
No. 6. Placenta showed small areas of older blood clot.
No. 8. Placenta adherent.
No. 11. Placenta "diseased". Large retro-placental clots.
No. 12. Placenta "very unhealthy".
No. 38. Placenta poorly developed. Large retro-placental clots.
No. 70. Appearance of placenta "suggested Accidental Haemorrhage."

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OBSERVATIONS ON CERTAIN CASES.

Case 42.

This case is noted as Accidental Haemorrhage and Placenta Praevia. There is present an albuminuria and a definite history of toxaemia. Why is it that albuminuria is so common in Accidental Haemorrhage and yet so exceptional in placenta praevia?

If, as we believe, the separation of a normally placed placenta is due to a circulating toxin, why does not separation before labour occur with its accompanying albuminuria more often in cases of placenta praevia? Possibly the position of the placenta may have some influence on the causation of the toxaemia.

In this case the placenta is marginal. There is no evidence to denote from what part of the placenta the bleeding came. It is possible that bleeding came from both the normally placed portion and the praevia. The separation of the upper portion could be by the process that occurs in Accidental haemorrhage. The separation of the lower portion could be either mechanical, due to the intermittent uterine contractions that occur towards the end of pregnancy, or else due to the same process of separation as the upper part.

Case/
Case 46.

This patient was treated as Accidental Haemorrhage. The bleeding ceased and the patient was discharged. She was later readmitted. The condition was then found to be placenta praevia.

This patient had albuminuria on the day of delivery. Towards the end of pregnancy she had a slight oedema of one leg and a slight visual disturbance. One could explain the albuminuria by saying that the toxaemia, of which evidence has been given, became more marked towards the end of pregnancy. It could be explained on Dr Young's theory of placental toxaemia. A portion of the placenta praevia may have become separated before the patient's first visit to hospital. This resulted in bleeding. The separated portion then became necrotic and resulted in toxins being absorbed into the circulation.
SECTION II. C.

SUMMARY AND CONCLUSION.

1. During the five years 1923-1928 inclusive 168 cases of Accidental Antepartum Haemorrhage occurred at the Edinburgh Royal Maternity Hospital. During this time there were 10,119 deliveries in the hospital. This gives an incidence of 1 case in 59 deliveries or 1.6%.

During the same period 24,234 patients were "treated by the hospital. (See p.7). Thus the average incidence was 1 case in 143 patients or 0.7% of all cases treated by the hospital.

This latter figure agrees with that of Whitridge Williams, namely 0.85% of 2000 consecutive labours.

2. In 168 consecutive cases 21.4% were primigravidae.

and 78.5% were multiparae.

In the 100 cases especially investigated 25% were primigravidae.

and 72% were multiparae.

3. The average age incidence was 30.55 years.
4. There was a proportion of 1 abortion to 10 pregnancies.

5. Of 134 multiparae the average parity was 4.2, and the maximum parity 17.

   In 72 cases of the 100 cases especially investigated the total number of pregnancies was obtained.
   The average parity of these multiparae was 4.13 and the maximum parity 15.

6. Of 82 patients with Accidental Antepartum Haemorrhage 53 had an albuminuria.
   This is a percentage of 64.63%.

7. Approximately 3% of all the cases were associated with eclampsia.

8. In 30 cases out of 98 of Accidental Haemorrhage the patient had albuminuria but no other toxic symptoms.

9. Of the 53 cases with Albumin
    39 were Multiparae
    14 " Primiparae.

10. In 77 cases trauma played a possible part in 7 cases.
    In 2 cases only
    Nos. 58 and 84
    was trauma a definite cause of the condition.
11. In 69 cases the umbilical cord was measured. The average length was 20.2". The minimum length was 11".

12. Average period of gestation in 98 cases was 34.6 weeks.

13. In 98 cases considered there were:
   
   72 cases of External haemorrhage.
   7 " " Combined "
   6 " " Concealed "

14. There were 14 cases in which evidence of an aetiological factor was lacking. (See p. 68)

15. The treatment followed in the series of cases is summarised on pages 69 and 70.


In 98 cases of Accidental Haemorrhage there were 7 maternal deaths. On examination of the records of these fatal cases one finds that two cases - No. 4 and No. 64 - were admitted moribund. One notes that two cases - No. 54 and No. 80, in which vaginal packing was carried out, were terminated one by septicaemia, the other by peritonitis.

17./
17. Of 86 children born in hospital 49 were still born and 7 died soon after birth. This is an infantile mortality rate of 65.1%. This is a high rate but no higher than one expects. In the 8 cases of concealed haemorrhage the infantile mortality rate was 100%.

Death of the child in the majority of cases was due to asphyxia, or to prematurity or to cerebral haemorrhage.

18. A Wassermann test was made in 12 cases. Syphilis was present in three cases, possibly in four, and absent in eight cases.

19. In eight cases definite pathological infarction of the placenta was noted. In six other cases placental abnormalities were noted.
CONCLUSION.

The aetiology of Accidental Antepartum Haemorrhage is a difficult problem indeed to solve.

The writer of this paper believes that Accidental Haemorrhage is due to a toxaemia. In most of the cases the toxaemia is shown by an albuminuria, or by signs and symptoms already described in this paper. The toxaemia can be assumed to be due to a Nephritis, a Nephritis in some cases only revealed by the occurrence of pregnancy. This theory so strongly advocated by F.J. Browne is supported by more recent work carried out by the same investigator working in conjunction with Gladys Dodds.

The views in regard to the causation of Accidental Haemorrhage, put forward in 1914 by James Young, and discussed in this paper, have now been modified. Recently he has pointed out how frequently toxaemic conditions tend to recur in subsequent gestations and so too the condition of Accidental haemorrhage. He does not agree that the recurrence of toxaemia is due to a deficiency in renal function. He points out that the toxaemia may be purely incidental. He says that/
that there is one factor alone that is an invariable accompaniment of these abnormal pregnancies. This is placental disease.

He accounts now for all the facts of accidental (3) haemorrhage by assuming the presence of some unknown factor or idiosyncrasy which precipitates a toxaemia by involving the life of the placenta. This idiosyncrasy may persist in the intervals between pregnancies giving no evidence of its presence, but reasserting itself in the same way at the next pregnancy. After the damage to the placenta the presence of toxaemia will depend upon the extent of placental damage and the length of time during which the damaged placenta is retained in the uterus.

Against this theory one can say that the placenta is not invariably diseased. In the present series of cases placental disease was noted in only 14 cases. Either insufficient or inaccurate examinations were made of the placentae or else the majority were healthy.

R.H. Paramore maintains that where Accidental (4) Haemorrhage occurs in toxaemic states, it is not the toxaemia which causes the rupture of the placental sinuses but the raised blood pressure.

In/
In the investigation of the series of cases one would have liked to have had a record of the blood pressure in each case, but this fact was recorded in very few of the cases. The theory of raised blood pressure being the cause of the bleeding is not generally held.


By F.J. Browne and Gladys H. Dodds.


2. Recurrent Pregnancy Toxaemia.

By James Young.

Reprint from same journal as 1. Vol. XXXIV No.2.

3. The Prognosis and Treatment of Eclampsia and Albuminuria in Pregnancy.

By James Young.


By R.H. Paramore.

SECTION II. D.

BIBLIOGRAPHY.

1. G. Blacker.
   "The Limitations of Caesarean Section".

2. Aleck, W. Bourne.
   Synopsis of Midwifery & Gynaecology (4th Ed.)
   p.163.

3. F.J. Browne.
   "Experimental investigation into the aetiology of Accidental Haemorrhage & Placental Infarction".


5. Samuel J. Cameron.
   "The Treatment of Concealed Accidental Haemorrhage".

   Manual of Midwifery 6th Ed.

7. Gibbon Fitzgibbon.
   "A Revised Conception of Antepartum Accidental Haemorrhage".

   "The Clinical Condition of the Uterine Wall in Concealed Accidental Haemorrhage".

   A Practical Handbook of Midwifery & Gynaecology.

    Ante Natal Care.


20. Whitridge Williams, Obstetrics 1924.

21./