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Accidental Haemorrhage

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Abstract

Edward Rigby of Norwich, in a treatise on uterine haemorrhage published in 1776, first shed light on the problem of bleeding from the vagina during later pregnancy. In this essay he differentiated between an accidental haemorrhage and the haemorrhage of a placenta praevia. From his observations accidental haemorrhage was defined as "bleeding from a normally situated placenta, after the 28th week of gestation, and up to the end of the second stage of labour."

Accidental haemorrhage is divided into three varieties :

1. Revealed Accidental Haemorrhage.-where the bleeding is entirely external;
2. Concealed Accidental Haemorrhage.-where there is no sign of blood externally;
3. Mixed or Combined Accidental Haemorrhage.-which shows features of both concealed and revealed.

The most important of these is the concealed variety and there are four ways in which a haemorrhage may remain concealed:

1. A retroplacental haemorrhage occurs in the central area of the placenta, but the margins of the placenta remain adherent to the uterine wall.
2. If the placenta becomes completely separated the membranes remain attached to the uterine wall.
3. The blood may burst through the membranes into the amniotic sac mingling with the liquor, and immersing the foetus in a blood bath (Lesser 1951).
4. When the foetal head is accurately applied to the lower uterine segment so that the blood cannot make its way past it into the vagina.

In spite of these mechanisms it is much more common for blood to escape externally ; a concealed accidental haemorrhage thus becomes a mixed accidental haemorrhage.

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Accidental Haemorrhage

By JAMES MOWAT

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Edward Rigby of Norwich, in a treatise on uterine haemorrhage published in 1776, first shed light on the problem of bleeding from the vagina during later pregnancy. In this essay he differentiated between an accidental haemorrhage and the haemorrhage of a placenta praevia. From his observations accidental haemorrhage was defined as "bleeding from a normally situated placenta, after the 28th week of gestation, and up to the end of the second stage of labour."

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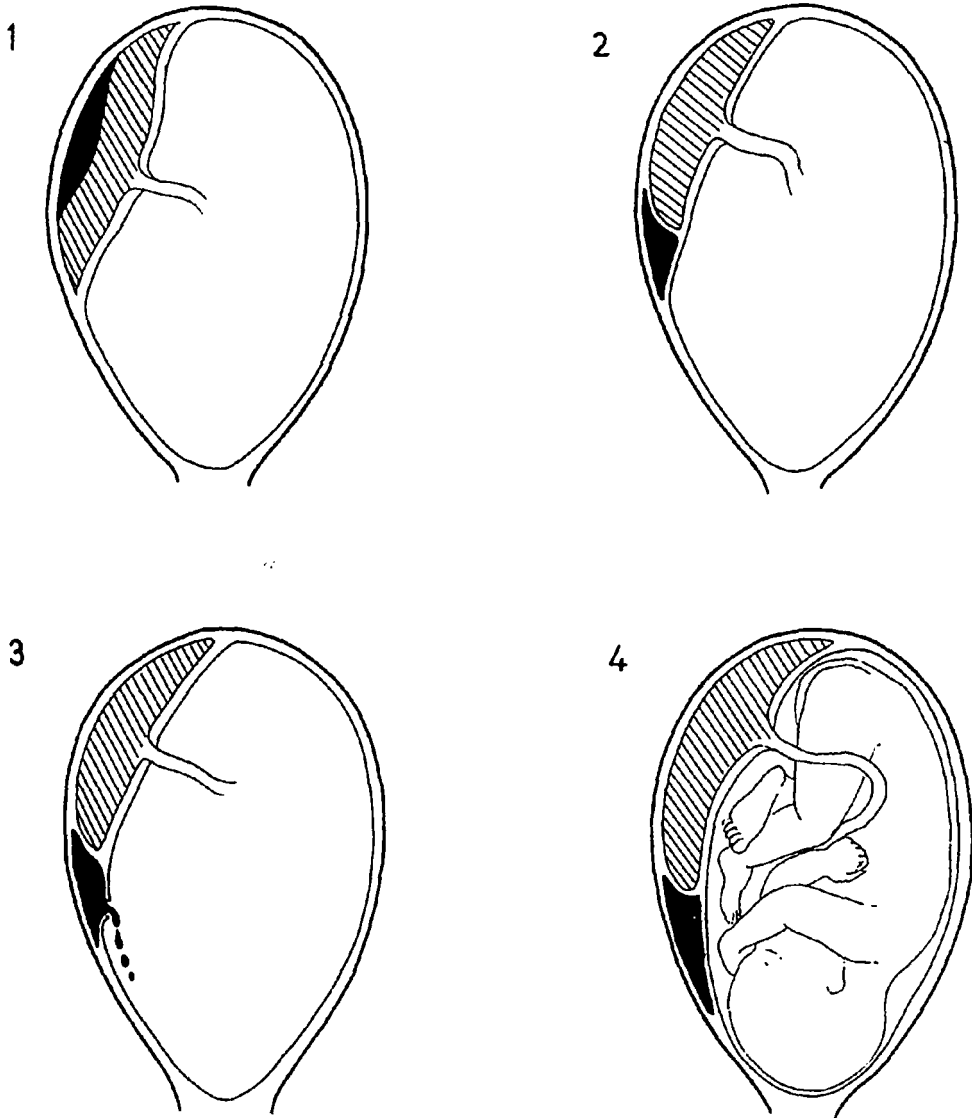
Incidence

This varies in different published series, but in Britain the average incidence is about 1.5% i.e. 3 in every 200 pregnancies. In America however this figure is much lower, being about 1 in every 600 pregnancies. This variation may in part be explained by differing criteria in diagnosis, and in the type of cases included under accidental haemorrhage (S.M.M.P. Annual Report 1960. p. 5). Concealed accidental haemorrhage, makes up about 12 - 16% of the total accidental haemorrhages.

Aetiology

There is no known one cause of accidental haemorrhage. It is probably for this reason that so many causes have at one time or another been evoked.

Rigby conceived of accidental haemorrhage as a feature secondary to certain external factors—"separation of the placenta must be owing to some accidental circumstance, to violence done to the uterus by blows or falls, to



The four types of concealed accidental haemorrhage (for key see text opposited).

some peculiar laxity of the uterine vessels from badness of habit or fever, or to some influence of passion of mind, suddenly excited such as fear or anger."

Trauma definitely seems to be a predisposing factor, but in a minority of cases. The trauma may be direct e.g. a severe blow on the abdomen, or indirect e.g. severe exertion, coitus, or heavy lifting. An important obstetrical factor is ante-natal versions, and this is why these should not be performed under general anaesthesia.

At the present time the most important aetiological factor is thought to be pre-eclampsic toxæmia. The incidence of this toxic variety varies with different authors, some with an incidence as high as 90%, while others are equally emphatic with the incidence of 5%. The criteria for the diagnosis of P.E.T. is obviously of importance in comparing the differing figures. The correct definition of P.E.T. must be used i.e. the raised B.P., with oedema (latent or obvious), and later albuminuria. In many instances P.E.T. has been diagnosed by the presence of albumin in the urine *after* the haemorrhage. This is misleading since the albuminuria may very well be due to the shock accompanying the haemorrhage. That pre-eclampsia is not the entire answer is seen by looking at the small number of accidental haemorrhages in those with P.E.T. Hypertension must also be mentioned as being an important aetiological factor (5 - 10%).

Any abnormality in the attachment of the placenta, or deficient attachment, may be a cause of premature separation.

(1) It is often noticed that women admitted with acc. haems. have had A.P.H.s. (Including abortions) in previous pregnancies. This must surely point to some inherent maternal defect, preventing good placental attachment or causing early separation, since no other pathology can be demonstrated.

(2) Interference with the normal structure of the uterine wall e.g. as a result of previous operations such as D. & C., myomectomy, or Caesarean Section, or if a tumour is present, may result in poor placental attachment.

C. S. Russell (Douglas 1955) has found that there is a fall in the level of urinary pregnanediol prior to accidental haemorrhage in those under observation because of repeated accidental haemorrhages. Whether this endocrine disturbance is primary or is a manifestation of general metabolic or pathological upset, is unknown.

It is thought that hydramnios and twin pregnancies may precipitate premature placental separation. Authors holding these views are divided into two schools of thought, one believing that the mechanism is the sudden release of an abnormally high intra-uterine pressure when the membranes rupture, and the other group considering the cause to be the pressure of a bulky uterus on the inferior vena cava, interfering with the venous return from the placenta.

Among other factors suggested are :

Chronic endometritis,

Severe torsion of the uterus,

Shortness of the umbilical cord (causing traction during labour).

Accidental haemorrhage is seen more often in the multiparous than the primigravida patient, and in a series from the records of the S.M.M.P., the unbooked multips showed a high proportion of the numbers admitted with accidental haemorrhage. Age of the patient seems to be of little significance.

Pathology

The initial lesion is thought to be a decidual haematoma which interferes with the function of the immediately overlying and now separated placental area. If the initial bleeding is from a blood vessel at or near the edge of the placenta, no further separation may occur, the blood tracking down between the chorion and the uterine wall to the cervix. In such cases the haemorrhage is limited and the foetus is not likely to be embarrassed—revealed type of haemorrhage. Fish (1951) claims that a marginal sinus rupture is of this type.

If separation occurs further in towards the centre of the placenta there may be no vaginal bleeding, and only a tender spot felt on abdominal palpation—in such a case the shed placenta shows a small depression containing fresh or

organised blood clot, depending on its age. More often the extent of the haemorrhage increases to produce further placental separation until the margin of the organ is reached and blood escapes. (Holland 1950).

There have been many suggestions as to the reason for the initial bleeding which causes the placental separation. In 1915 Williams described degenerative lesions in the intima of the small uterine arteries, and he suggested these vascular changes were due to the toxæmic process, (and thus anoxia). Bartholomew (1953) describes the placental pathology in acc. haem. as being identical with that found in pre-eclampsia and eclampsia. At any rate there are units of placental tissue which appear grossly or sharply circumscribed, round or oval, slightly firm areas. This may be a local or generalised lesion. Microscopically the villous capillaries are dilated from a diameter of 3 rbc. to 20 rbc., or they may be ruptured. General ischaemia of the placental bed from maternal vascular disease or atherosclerosis of decidual arterioles must have nothing to do with it, Bartholomew argues, since the pathological picture is that of sharply demarcated areas. Infarction may produce haemorrhage and placental separation.

In the more severe case, either at post-mortem or operation, the uterus may be seen to be enlarged, purplish red in appearance, and with extravasated blood infiltrating its muscle fibres, and penetrating into the uterine ligaments. This classical picture, known as the Couvelaire Uterus, is associated with a decreased coagulability of the blood, a hypofibrinogenaemia, which allows the blood to penetrate the pelvic structures owing to the deficiency of the clotting mechanism. The Couvelaire uterus interferes with uterine retraction, and thus leads to further grave bleeding.

Douglas (1955) in a series quotes 75% incidence of Couvelaire uterus in patients whose delivery was beyond 3 hours of placental separation. In a series of 100 cases with 7 maternal deaths, Browne (1952) found Couvelaire lesions in 5, not in one, and unknown in one.

Marginal sinus rupture can only be diagnosed by examination of the placenta (Firth 1951). At the margin of the placenta a grossly dilated blood vessel may be noted, showing evidence of rupture, and containing blood clot continuous with blood clot outside. The placenta must be a wide membrane placenta.

Clinical Features

The signs and symptoms vary in each case from the mild barely noticeable ones, to the typical full blown picture of a severe concealed accidental haemorrhage.

In the revealed type vaginal bleeding is usually the only symptom. Although the vaginal bleeding may be a presenting feature in the mixed variety, pain is more often the first symptom. The pain may vary from backache and slight crampy abdominal pain, to the severe, sudden, and agonising pain characteristic of a severe concealed haemorrhage. After a variable period in the concealed variety there is usually some vaginal bleeding—and the haem. becomes a mixed accidental haemorrhage.

Mild cases of concealed haemorrhage may only be diagnosed after the delivery of the placenta, when retroplacental clot is noticed.

This, however, is not necessary to diagnose the severe case, where the woman presents in a shocked condition, pale, sweating, restless, and complaining of severe abdominal pain. There may be little or no vaginal bleeding, but where it has occurred, the shocked condition of the patient is out of all proportion to the revealed blood loss. The bleeding may not only come from the vagina, but from mucous membranes e.g. gums; haematemesis and haematuria may also occur.

The pulse is taken and shows a tachycardia. The B.P. is low—though a B.P.

of e.g. 140/90 is not uncommon—repeated readings however will show a downward trend. A high reading should make us think of a superimposed toxæmic state.

When the urine is tested, albumen is found, often in massive quantities. The albuminuria may be due to the shock-albuminuria syndrome, or it may have been present before the hæmorrhage, because of pre-eclampsia. Oliguria may occur due to the renal embarrassment and in the most severe cases this may proceed to anuria.

On examination of the abdomen, the uterus is tense, rigid, and exhibits the typical woody hardness of concealed hæmorrhage. It is extremely tender. Because of the rigidity of the uterus it is well-nigh impossible to determine whether there are any uterine contractions, and palpation of the foetal parts is impossible.

Progressive increase in the size of the uterus is an ominous sign, indicative of continuing bleeding. The blood insinuates itself into the uterine musculature and even into the uterine ligaments—producing the Couvclaire uterus as mentioned previously.

It may be impossible to hear the foetal heart beat on auscultation. Indeed in cases of this severity it is probable that the foetus has died in utero, though this must not necessarily be assumed.

Vaginal examination is required to diagnose the position of the foetus and stage of labour.

In retrospect most of the cases presenting with such severe symptoms have had warning episodes, chiefly a deep-seated backache, or a slight lower abdominal pain. Since backache is such a common accompaniment of pregnancy, it is quite to be expected that it will not arouse any suspicion on the part of the woman, or indeed of her practitioner. Lower abdominal pain may produce some anxiety, and prevention, or at least an awareness of the underlying pathology of this pain, may be important in more rapid treatment.

Complications

1. Hypofibrinogenaemia

The normal level of fibrinogen in the maternal blood is 400 - 700 mg.% ; below 100 mg.% the blood is deprived of its coagulative activity.

DeLee in 1901 thought that accidental hæmorrhage was aggravated by the blood being unable to clot, but it was not until 1936 that Dieckmann suggested that decrease in blood fibrinogen was the factor responsible.

Hypofibrinogenaemia follows the placental separation. Then the incoagulable state of the blood gives rise to

1. enlargement of the original retroplacental hæmatoma
2. Couvclaire uterus
2. post-partum hæmorrhage

The mechanism producing hypofibrinogenaemia, is thought to be (Larkin 1957)—thromboplastin is injected into the maternal circulation from the placenta which is a rich source of thromboplastin. As a result intravascular fibrin is formed. At the same time, as a result of shock, an active fibrinolysin is released which becomes absorbed on to the intravascular fibrin as latter is in process of being formed, and destroys it. In this way large quantities of fibrin are continually being formed and destroyed in the circulating blood.

Owing to shock and generalised vascular spasm the blood supply to the liver is reduced. As long as this situation is maintained the liver does not replenish fibrinogen to the circulation, though in its normal state it is capable of doing so quite rapidly.

A vicious circle is thus set up in which the blood becomes incoagulable and will remain so, so long as the patient is shocked. As long as the blood is

incoagulable the patient will go on bleeding. The more bleeding, the deeper the shock becomes. In order to break this circle shock must be combated and the uterus emptied. The condition of shock should not be allowed to continue long enough to endanger the kidneys.

2. Anuria

Fortunately anuria is not a common complication of accidental haemorrhage, although some degree of oliguria is common.

Several theories have been postulated as causing anuria :

1. Shock and its accompanying low blood pressure may produce it. In this case anuria occurs early and is transitory in nature.
2. If the shock is of greater severity and causes primary vasospasm of the arterioles to the nephrons, it may lead to cortical necrosis and its irreversible anuria. [Sophian (1955) associates the utero-renal reflex with this.]
3. Some authors (Browne & Browne 1960) think that a toxin absorbed from the uterine muscle is responsible, while Johnstone & Kellar (1957) mention 5-hydroxytryptamine.
4. Schneider (1954) blames fibrin deposition in efferent and afferent arterioles and in the glomeruli as the cause of renal necrosis. Sheenan & Moore (1952) oppose this theory and claim that these thrombi are the result of vasoconstriction, and not the cause.

Management

In revealed haemorrhage, the management is more or less straightforward. Immediate bed rest in hospital is necessary. Sedation may be needed to allay anxiety. If the patient is already in hospital e.g. a haemorrhage in a pre-eclamptic confined to hospital, oxygen may be administered during the bleeding if it happens to be a bit profuse.

Although the bleeding is usually not enough to warrant blood replacement, there may be some place for blood transfusion in a woman who is slightly anaemic.

An X-ray to exclude the presence of a placenta praevia must be mentioned as essential while the patient is in hospital.

Whatever else is done, increased vigilance on the part of the obstetrician is a duty which should not be neglected.

In the more severe case of mixed or concealed haemorrhage, there is no time for delay in treatment. Usually in such cases, the safety of the mother is the primary consideration, the infant's welfare taking second place. Active treatment is required immediately; in domiciliary practice the flying squad has its part to play in resuscitating the patient and bringing her into a well-equipped maternity hospital.

On arrival at the hospital the mother's condition must first be assessed. Shock must be combated by the use of blood transfusion and stimulants. The longer the delay in instituting treatment for shock, the greater the shock becomes, and the greater is the risk of complications such as renal cortical necrosis arising.

Morphine should be used to combat the severe abdominal pain, and to allay anxiety. Reassurance by the obstetrician is of course of great help to the patient.

Hourly clot observation tests are begun to test for the ability of the blood to clot and for its stability. If there is hypofibrinogenaemia, 2-4G of fibrinogen is given intravenously; it may be necessary to repeat this dosage.

Catherisation will confirm the presence of albumen in the urine. Frequent samples of urine should be taken e.g. every hour, and the volume measured. Should the urinary excretion fall, some obstetricians advocate that bilateral splanchnic block should be performed. Barry (1955) and O'Sullivan and

Spitzer (1946). Recourse to such a measure is not common. Anuria will not of course give rise to trouble until some time after delivery. Bull's regime is important in such cases, and it may be necessary to alert the artificial kidney team. A renal biopsy to differentiate between renal cortical and tubular necrosis is a possibility since renal cortical necrosis has a fatal outcome, whereas a patient with tubular necrosis may be tided over by dialysis, until recovery of kidney function.

Since prompt evacuation of the uterus best serves the interests of both mother and child, this should be the aim, as much as is compatible with safety.

Artificial rupture of the membranes should be done whether delivery is to be by the abdominal or vaginal route, and this is done at the vaginal examination. An A.R.M. is of importance for the following reasons :

- (i) it relieves shock
- (ii) it breaks the utero-renal reflex
- (iii) it reduces thromboplastin liberation
- (iv) it stimulates uterine contractions

Although almost all authorities are agreed on the necessity of doing an A.R.M., the use of pitocin is still a much debated procedure. The main objections to its use are :

- (i) an aggravation of the renal vasospasm,
- (ii) rupture of the uterus in grandmultips,
- (iii) increased expression of noxious substances from the placental site into the maternal circulation—i.e. thromboplastin, which will eventually further decrease fibrinogen concentration. There is also the chance of causing an amniotic fluid embolism.

Only recently has it been advocated that pitocin should be used to speed up delivery, and the indications are that the objections to its use are more theoretical than real.

If there is no progression in labour, and the shock has been corrected—the mother being now in the best condition possible in the circumstances—a Caesarean section should be carried out as soon as possible.

Whether to do a Caesar or whether to wait to see if labour will progress is the most difficult decision to make.

The shorter the period of time from the acute onset of the placental separation until the ultimate delivery, the less is the likelihood of the development of some of the dreaded maternal complications, and of losing a baby whose heart is heard on admission. Little time should be wasted in waiting to see whether a birth via naturalis is going to be possible. Eastman (1956) prefers to wait 10-12 hours to see if the A.R.M. is going to bring about delivery, while Bysshe (1951) agrees to a 6 hour limit—by which time the foetus if not already dead, is in a very precarious position.

Tied up with the question of time to delivery is the question of whether the woman is in labour or not, and whether there is any cervical effacement or dilatation. The presence of labour is not a contra-indication to a Caesar. The progress in the descent of the head is the only real indication of the progress of labour.

The condition of the foetus is, of course, of great importance. Should the foetus be alive and in good condition, then there may be some justification for delaying to see whether vaginal delivery is possible, though this is probably a better reason for doing a Caesar, especially in the more severe case. If there is any slowing or irregularity of the foetal heart, a Caesar becomes imperative. Each case will be decided on its own merits.

Maternal Prognosis

This depends on a number of factors among which are :

1. Type of haemorrhage, i.e. revealed or concealed ;
2. Extent of placental separation ;
3. Total blood loss (including P.P.H.) ;
4. Degree of hypofibrinogenaemia ;
5. Presence of Couvelaire uterus ;
6. No. of hours between placental separation and treatment ;
7. Presence or renal damage ;
8. Associated maternal disease e.g. vascular disease.

In revealed and mixed types of haem. the mortality is about 1.5% whereas in concealed haem. the maternal mortality is as high as 10%. The prognosis has greatly improved recently due to such factors as the flying squad, blood transfusions, fibrinogen, and the artificial kidney.

Foetal Prognosis

Although the prognosis for the mother has shown a vast improvement in the last number of years, there has been little improvement in the foetal prognosis.

Johnstone and Kellar (1955) quote a foetal loss of 45% for mixed and revealed haemorrhages, and 90% for concealed haemorrhages. There is no further need to stress the importance of accidental haemorrhage, the above figures do all that is necessary.

One reason for the high foetal mortality is the high incidence of prematurity. Although those weighing over 2000G (4.4 lbs.) have approx. 100% chance of survival, it falls to 80-90% from 1500 to 2000G, and under 1500G (3.3 lbs.) the survival rate falls off rapidly. Major anomalies e.g. anencephaly, make up a proportion of the unavoidable foetal loss. The main cause of death is, as might be expected, anoxia.

By way of prevention not much can be done ; the aetiology is largely unknown, and the haemorrhage occurs with such dramatic suddenness.

At the moment the greatest benefit will be derived from the quicker and more efficient active treatment of the woman once the calamity has befallen her. The instruments for this are at hand, but a continual reappraisal of the problems involved, and a self-criticism of past failures is an absolute necessity, if a better foetal prognosis is to be anticipated.

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