PLACENTA PRAEVIA IN U.S.M.

A Review

by

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To my wife and my children, a million thanks for being understanding and patience. God bless you.

Contents

Hospital east wing. It has 8 low risk bed, 5 high risk bed and 2 for eclampsia patients. The patients monitoring during labour in the labour suite is done by the cardiotocography and fetal scalp blood sampling for pH. The monitoring facility in the department is recently further enhanced with the delivery of the new ultrasound machine equipped with Doppler facility which increases our ability to assess high risk pregnancy. The labour suite has its own operation theatre where emergency caesarean sections are done during office hours.

Since starting its postgraduate programme (Masters in Obstetrics and Gynaecology) in 1991, the department's academic programme has been intensified . The activities of the department include:

1. Weekly presentation during lunch hour on Sunday to discuss controversial topics, interesting clinical cases , and review recent papers.
2. Weekly clinical presentation, Viva sessions, or practising multiple choice questions and essays .
3. Monthly histopathological meeting with the Pathology department.
4. Perinatal Audit every Wednesday with the Paediatrics colleague.

The Department conduct the clinics as follows:

Antenatal Clinic - Saturday and Tuesday.

Booking Clinic - Sunday

Combined Clinic - Monday

Postnatal Clinic - Wednesday evening.

General Gynaecology - Saturday and Tuesday evening.

Molar and Oncology Clinic - Monday evening.

Infertility Clinic - Wednesday morning.

Family Planning Clinic - Wednesday evening at Kelantan Family Planning Association's Clinic.

By the middle of 1996, when the construction of the new block is completed, the Obstetric wards and Labour suite will be shifted to its new and more spacious location. This move not only will boost the services provided by the department, but it is also hope that the undergraduates and postgraduates programmes be further improved.

Abstracts:

The Obstetrics and Gynaecological case records of 190 patients with proven placenta praevia have been reviewed between January 1990 till December 1992 . With equal number of controls both groups were compared for predisposing factors namely previous caesarean sections and previous abortions. The outcome between symptomatic (n=148) and asymptomatic (n=42) group were looked into with regards to maternal and perinatal morbidity, maternal age group, parity, gestational age at diagnosis, gestational age at delivery, birthweight, duration of admission, the need of blood of transfusions and mode of delivery. There was significant relation between placenta praevia and previous caesarean section ($p< 0.05$), and previous abortions ($p< 0.05$). Those patients without bleeding delivered a significantly larger baby and spent less time in the hospital. They also were found to have a significantly lower rate of emergency caesarean section. The occurrence of bleeding was significantly more in those with central placenta praevia (type 4) than other types.

Abstrak

Antara bulan januari 1990 dan bulan disember 1992, sebanyak 190 orang pesakit yang telah di buktikan sebagai ' Placenta Praevia' telah diambil untuk kajian retrospektif ini. Antara yang diambil kira ialah 2 faktor penyebab, iaitu pembedahan ' caesarean' dimasa lalu dan keguguran. Ianya kemudian dibandingkan dengan sekumpulan 190 pesakit lain sebagai 'control'. Antara faktor-faktor lain yang dikaji ialah perbandingan antara sekumpulan pesakit tanpa pendarahan (n=42) dan pesakit yang mengalami pendarahan (n=148). Hasilnya , perhubungan antara terjadinya 'placenta praevia' dan pembedahan 'caesarean' dimasa lalu dan keguguran adalah 'signifikan' . Juga dapat dilihat perbandingan antara pesakit-pesakit yang tidak mengalami pendarahan dan pesakit-pesakit yang mengalami pendarahan. Adalah didapati secara keseluruhan dan secara amnya, hasil daripada pesakit-pesakit yang tidak mengalami pendarahan adalah lebih baik daripada pesakit-pesakit yang mengalami pendarahan. Sebagai kesimpulan, pesakit-pesakit yang tidak pernah mengalami pendarahan berkemungkinan tidak memerlukan pemerhatian berterusan didalam wad, malah boleh dirawat sebagai peasakit luar.

The aims.

1. To assess the relationship between predisposing factors such as abortions and previous caesarean sections in relation to placenta praevia.

2. To determine the common causes of maternal morbidity and perinatal morbidity related to placenta praevia.

3. To compare the outcome between symptomatic and asymptomatic patients and to review the policy of expectant management in these cases.

INTRODUCTION

Placenta is a Latin word derived from the Greek " Plakous " meaning a flat plate or cake (plaque in French). The term placenta was first used by *Realdus Columbus* who in the first edition of his book " De Ra Anatomica ", published in 1559 introduced the Latin word for circular cake as a simile in his description as the thick part of human fetal membranes.

The placenta is an organ consisting of both fetal and maternal tissues, and in one form or another it is found in most viviparous vertebrates, including all the Eutheria (True mammals). The placentas of different species of mammals show extreme variability in morphology , a fact that is difficult to explain in term of natural selection. The variability has posed considerable problems both in its definition and classification.

The Chorio-allantoic mammalian placenta has been defined as apposition or fusion of the fetal membranes and uterine mucosa for physiological exchange between the fetus and the mother. The fetus is surrounded by the chorionic sac, to which it is connected by the umbilical cord. The chorionic sac lies in the uterine cavity and has contact with the endometrium over almost its entire surface. The maternofetal contact zone, provided by chorion and endometrium, represent the placenta. In human placenta the fetal and maternal components are interlocked in such a manner as to provide an extensive surface of contact between the two tissues without any mixing of the circulation. The basic architectural design is for extensive contact between maternal blood in the intervillous space and the surface of the chorionic villi.

The human placenta is classified as Chorio-allantoic since it is vascularised by vessels homologous with the allantoic vessels of the lower mammals ; haemochorial because of the nature of its placental membranes ; villous because its villi ; deciduate because maternal decidua is shed at birth along with the fetal placenta ; and discoidal because of its circular shape.

The nonspecific definition points to the great structural and functional variability of the organ. There is probably no other organ that exhibits such an extent of species differences as the placenta. When comparing species, the outer shape and inner structure can vary to such a degree that non-placentologist , who are familiar with the structure of only the human placenta, probably cannot identify any other type as a placenta at all. There is only one structural component that all placental types have in common, that is , the existence of two separate circulatory systems ; the maternal system and the fetal system. Under normal conditions the vessel of two systems remain separated by several tissue layers throughout pregnancy. Even the origin of these separating tissue layers, making up so-called placental barrier or intrahemal membranes varies. They may derived from the fetal tissues such as the blastocyst wall (Trophoblast), the allantois, or the yolk sac, but in some cases these fetal constituents are completed by maternal tissues, that is the remainders of the endometrium.

The placenta is unique among all other organs in that it comprises the functional activities of most fetal organs (except the locomotor apparatus and the central nervous system) from its early beginning onward throughout its development. The following fetal functions are partially or completely devoted to the placenta during pregnancy, as a substitute for still immature embryonic and fetal organs:

1. Gas transfer instead of the lung.
2. Excretory functions, water balance, pH regulation, instead of kidney.

3. Catabolic and resorptive functions instead of the gut.
4. Synthetic and secretory functions of most endocrine glands.
5. Numerous metabolic and secretory functions of most of the liver.
6. Haematopoeisis of the bone marrow (during early stages of pregnancy).
7. Heat transfer of the skin.
8. Immunological functions to a largely unknown degree.

It is highly unlikely that these numerous functional requirement can be fulfilled by phylogenetic development of an identical structural solution for all the species, as the latter exhibit tremendous variations in size, pregnancy length, litter size, and living conditions. The logical result of this situation has been numerous placental types, differing from one another with respect to the outer shape, kind of maternofetal interdigitation, structure of the maternofetal barrier, and maternofetal blood flow interrelations.

The placenta contains, within the intervillous space, about 700ml of maternal blood which reaches it via the uterine and ovarian arteries. The open arterioles discharge into the choriodecidual space, which depends for its integrity only on the adherence of the placenta to the maternal decidua. This normally remains intact until after delivery of the fetus, so long as the placenta is inserted into the upper segment of the uterus. Effacement and dilatation of the cervix causes separation from the chorion, from its attachment to the myometrium, sometimes producing a small amount of bleeding (a show).

DEVELOPMENT OF PLACENTA

The fertilized ovum which has reached the uterine cavity forms a hollow sphere or blastocyst with a mound of cells on one aspect of the inner surface - the inner cell

mass. The outer shell of the blastocyst becomes the trophoblast responsible for the nutrition of the embryo, which eventually develops and becomes the placenta. The primitive trophoblast erodes the surface of the decidua by enzyme action, destroys glands and stroma, and eventually penetrates the large maternal sinusoids which have formed. The blastocyst now lies in the pool of maternal blood fed by the maternal arterioles and drained by maternal veins. The trophoblast cells proliferate and form pseudopodial-like masses which branch repeatedly. This greatly increases the surface area and facilitates feto-maternal exchange. The trophoblast anchors the blastocyst by adhering to the intervening decidual stroma.

The trophoblast differentiates into two layers. The outer or syncytiotrophoblast, in contact with the maternal blood, becomes a multi-nuclear syncytium with no distinct cell boundaries, the inner or cytotrophoblast, also called Langhan's layer, forms a single layer of cuboidal cells.

Villi are present over the whole surface of the blastocyst. As the blastocyst enlarges it compresses the superficial decidua (decidua capsularis) and the pregnancy bulges into the uterine cavity. The compression of the decidua capsularis gradually cuts off the circulation through it. This results in atrophy and dissappearance of the villi in asssociation with it . The surface of the blastocyst becomes smooth and this portion of the chorion is known as the chorion laeve. At the opposite pole of the blastocyst the vili proliferate and enlarge and this is known as chorion frondosum. The connecting stalk of the embryo is attached to the wall of the blastocyst at this point. Ultimately with the expansion of the blastocyst the decidua capsularis comes in contact with the decidua vera and the uterine cavity is obliterated.

PLACENTAL ATTACHMENT

It has been sugggested that the site of placental implantation influences the frequency of fetal malpresentation. *Wingate and Paul* (1968) studied this with 51 CHROMIUM placental localisation in 85 patients. They found no correlation between fundal or lower uterine implantation and fetal malpresentation. The factors that determine the site of nidation of blastocyst are not fully understood. The human blastocyst implants normally in the upper portion of the uterus, nevertheless, abnormal implantation is frequent and may lead to pregnancy complications, such as placenta praevia.

Previous authors had suggested that abnormal location of the blastocyst in the endometrium may also lead to abnormal forms of the mature placenta. An early proponent of such thought was *Schatz (1886),* who envisaged normal, broad, and superficial implantation. These idea was particularly championed by *Torpin (1969),* who related the depth of implantation and its location in the uterus to development of circumvallate placentation. Others have suggested that the location is instrumental in triggering the normal impulse for the initiation of labour and that it was correlated with the length of gestation. In recent years, it has become practical to record and follow the location of the placenta during the course of gestation by sonography. This method has shown that a ' dynamic placentation' can be envisaged. That is, the original location of the implanting blastocyst may be modified during the course of its development to a term placenta.

There are several means by which investigators have approached localization of the implantation and the ultimate placental site. *Hertig and Rock* (1973) summarized their finding from successful search of 34 early human ova and obtained the following results; (see next page)

Sites	Number of cases
Tubal location	1
Free in uterus	7
Implanted normal	17
Implanted abnormal	9

Booth et al *(1962)* came out with the following distribution when post partum palpation was done on the placental sites.

Sites	%
Anterior	53%
posterior	39%
lateral	8%

When this data are expressed with respect to the height of placental location, the following distributions were found;

Fundal position	2%
Fundal & upper segment	42%
Upper segment	47%
Upper & lower segment	9%

A fundal attachment was found in 44% of primigravida and 20% of multigravida, and pre eclampsia was twice as common in the fundal placenta which may merely reflect the fact that this condition is commoner in a first pregnancy. There was no relation to length of pregnancy and labour. *Scipades and Burg (1930)*, in an extensive study on placentation, cite *Orsini (1928)*, who found an anterior placenta in 26.6%, posterior placentation in 41.6%, a fundal site in 2.2%, a lower uterus site in 23%, and other sites

in 3.5%. Later methods have used the distance of the membrane rupture site to the edge of placenta as an indicator of the placental localization *(Little 1962, Little and Friedman 1964)*. This method, of course, needs to take into consideration that the placenta must be carefully extracted and not disturbed after delivery. Among the 10,101 placentas correlated during the evaluation by *Little and Friedman (1964),* the mean rupture site was between 5 and 9 cm (39.3%). In 11% it was 0 cm (possible marginal placenta praevia), and in only 0.3% was it more than 19 cm. These authors were unable to confirm the relation of high uterine position and toxaemia but did not make a distinction with the parity of their patients. They found a twofold higher frequency of vaginal bleeding with low-lying placentas, but otherwise no influence on the length of labour, type of delivery, or neonatal outcome was identified. *Torpin (1958)* used his method of distending the membranous sac after birth in a bucket of water and determined, in 147 cases, that when the placenta had implanted in the " crease made by the reflection of the anterior and posterior walls, the resulting placenta is almost invariably bilobate".

Indium isotope scanning and sonography were employed by *Harris (1975)* in 401 patients for placental localization in an effort to ascertain whether localization over the putative uterine "pacemaker" *(Larks et al 1959)* influences the length of pregnancy. It was found in the upper right quadrant in about the same frequency as in the left (85/79) , although the former had statistically significant shorter pregnancies. This finding disagrees with the theory that the location in the right upper quadrant lengthen the pregnancy.

A more incisive results came from studies using sonographic localization of placenta (*Gottesfeld* et al 1966) , which is now routinely ascertained during the course of pregnancy.

King in 1973 showed that a placenta praevia of the early pregnancy changes to one that does not require surgery for placenta praevia at term, and that this change takes place in most of the cases. He termed this phenomenon '**dynamic placentation**'. In none of his 14 cases of marginal placenta praevia observed near mid gestation , was there true placenta praevia at term, although several had near cervical implantation. He reasoned that this change in position comes about largely by uterine growth and its changing shape as gestation advances. Nearly the same conclusions were arrived at independently by *Meyenburg in 1976* and have since been confirmed by *Winters in 1978* . *Young in 1978*, who studied the same features with arteriography, located the placenta twice as often in the upper uterine segment than as a placenta praevia. He also noted the disappearance of most praevias with term approaching. He suggested that those placentas that do not move may represent accretas, but there was no histological support for this concept.

In a large study of ultrasonographic findings prior to amniocentesis between 16 and 18 weeks, *Rizos et al (1979)* found the following location;

site	praevia(%)	all placentas(%)
anterior	69	37
posterior	10	24
ant & post	21	5
fundal	0	34

Their incidence of placenta praevia at mid term was 5.3% and it converted to a non praevia in 90.4% by term. Their overall term praevia incidence was 0.58 % , similar to that of other studies.

A similar study of placental localization by sonography was reported by *Fried (1978).* When he followed the location over three trimesters, the results of which are summarized in the table below,

sites	10-20 weeks	21-31 weeks	32-40weeks
anterior	25%	41%	44%
posterior	37%	28%	26%
ant-fundal	9%	6%	8%
post-fundal	17%	18%	15%
fundal	12%	7%	7%

In other words, little change occurred in the overall location of placentas. He found many more cephalic presentations with anterior than posterior placentas.

Gallagher et al (1987) performed sonography during the second trimester of pregnancy in 1239 women and found a central placenta praevia in 3 patients and marginal or partial praevia in 48 (5%) . At term only 4 (0.3%) of these patients had a placenta praevia: the 3 patients with central praevia previously and 1 with marginal praevia found during the second trimester. They chose therefore to name this condition "potential praevia" when it is so identified during mid gestation in order to distinguish this sonographic condition from true placenta praevia at term. Studies have shown that transvaginal ultrasonography is more informative with respect to the presence of placenta praevia when third trimester haemorrhage necessitates diagnosis *(Lim et al 1989).*

An incisive study of localization by indium scintigraphy cobalt cervical marker comes from *Nordlander et al 1977.* They showed an anterior placenta in 98 instances and a posterior placenta in 80. An anterior placenta had a significantly greater chance of

being low-lying, and the distance of the placental edge from the uterine internal cervical Os increased with the age of the gestation. Attempts at making the exact diagnosis of placenta praevia during early gestation have ramifications for therapy. Thus *Arias in 1988* reported that the use of cervical cerclage in the treatment of vaginal bleeding from placenta praevia has merits as a temporary measure.

PATHOLOGY OF PLACENTA PRAEVIA

It is easy to understand the pathology of placenta praevia, the membranes has no free margin in the delivered placenta and the edge of placenta is frequently disrupted and haemorrhagic. There are often old clots at this site, varying from being laminated and brown , friable loose blood to partly decomposed material that is sometimes green from haemosiderin. The placenta is often irregular in shape and variable in thickness, and frequently the cord has marginal or vellamentous insertion. The fetal vessels of the chorionic surface , when at the edge may be disrupted.

Low lying portion of placenta are occasionally either atrophied or infarcted. It is most often the case the initially marginal praevia has failed to developed, has undergone atrophy, and has thus becomes a marginal praevia or better, a low lying praevia.

According to *Strassman (1902)* , one often encounters some degree of circumvallation or margination, however it is rarely reported in other series. *Strassman* also correlated the insertion of the umbilical cord with praevias and found that the cord may insert anywhere but it is more often near the site of the cervical Os , however this is not often so. And examples of eccentric placental growth that made Strassmann to such an

ardent champion of the trophoblastic expansion theory of placental tissue (expanding toward the better endometrial grounds above) are not often clearly seen.

Bartholomew et al (1953) suggested a novel mechanism of bleeding in central praevia. They observed that in the central villous portion of the tissue that overlies the internal os there were numerous old clots, apparently from the former bleeding episodes. They held these clots to be the evidence of placental villous disruption and assumed that it is not the detachment of placenta from the uterine wall that causes the bleeding. Moreover , at the edge of the placenta, disruption of the 'marginal sinus' may take place in the marginal placenta praevia, and thus lead to the bleeding when the endocervix dilates during late gestation.

Maternal haemorrhage may originate from the placental margin or the disrupted intervillous space. There is however, also significant neonatal anaemia associatad with the birth from the placenta praevia , more so when the maternal bleeding has been excessive (*Wickster, 1952 , McShane et al 1985*). This anaemia is clearly due to the well recognised , but rarely recorded , fetal bleeding that occurs from disrupted placental villous vessels during labour (*Bramberget et al 1957, Haetman et al 1962, Bar-David et al 1984*). The neonatal anaemia can also result , of course from the disruption that occurs when the placenta is cut during caesarean section, aspect that are well discussed by *Schellong in 1969.*

Naeye in 1978, using material from the Collaborative Perinatal Project, found that the placenta in fatal cases of placenta praevia were growth retarded, with an increased frequency of necrosis of the decidua basalis and thrombi at the placental margin. He also showed an increased frequency of villous hyperplasia in the placentae and excessive erythropoiesis in the liver and other fetal organs, in fatal cases. He attributed the decidual necrosis and marginal to premature separation of the placentae from the

uterine wall, and the villous hyperplasia and excessive fetal erythropoiesis, as a response to fetal haemorrhage.The women with placental villous hyperplasia had a history of recurrent maternal vaginal bleeding often starting in the second trimester of pregnancy. Microscopic abnormalities were no more frequent in the placentae of placenta survivors than in the placentae of survivors who did not have placenta praevia.

Just about more than 10 years ago, antepartum haemorrhage or APH was defined simply as bleeding from the genital tract after 28th week of pregnancy and before labour , however , two major advances have changed our understanding and management of bleeding in late pregnancy. The first is the contribution of diagnostic ultrasound which enable the placental site to be identified in early pregnancy before the appearance of symptoms. The second advance has been in the paediatric care of very low birth weight infants so that even infant born as early as 24 weeks' gestation now have a good chance of survival. This means that intervention in pregnancy for the infant can be undertaken before the arbitrary 26 weeks boundary.

The 28 weeks rule was convenient because in Malaysia and most countries including the United Kingdom, it is the accepted legal limit of viability and the registration of stillbirth. The International Federation of Gynaecology and Obstetrics (FIGO) has recommended that perinatal death statistics should include any fetus born after 22 weeks or weighing 500 grams or more. It is therefore seems reasonable that 22 weeks should also form the new boundary between the definition of APH or bleeding in the late pregnancy and bleeding of early pregnancy.

Bleeding in late pregnancy or antepartum haemorrhage complicates between 2 to 5% of all pregnancies and the various causes of bleeding late pregnancy is shown in table next page.

Causes of APH*

Cause	Incidence (%)
placenta praevia	31
abruptio placenta	22
'Other bleeding'-	47
-marginal	(60.0)
-'show'	(20.0)
- cervicitis	(8.0)
- trauma	(5.0)
- vulvovaginal varicosities	(2.0)
- genital tumours	(0.5)
- genital infection	(0.5)
- haematuria	(0.5)
- vasa praevia	(0.5)
- others	(0.5)

* from *High Risks Pregnancy-Management Options*.1994. edited by *James, D.K.*

The risk of bleeding in late pregnancy can be divided into maternal risk and fetal risk:

Maternal risks;

1. maternal mortality- This has dropped from 5% to less than 0.1% with the introduction of expectant management in mid 40s.

2. Postpartum haemorrhage- This is due to inefficient occlusion of the venous sinuses in the lower segment following delivery.

3. Anaesthetic and surgical complications- These occur especially in those with major praevia delivered by emergency caesarean sections where preparation for surgery is suboptimal.

4. Air embolism- It occurs when the sinuses in the placental bed are torn.

5. Postpartum sepsis- This is secondary to the ascending infection of the raw placental bed.

6. Placenta accreta- This occur in up to 15% of women with placenta praevia.
7. Recurrence- The risk of recurrence is about 4-8% after one previous praevia.

Fetal risks.

1. Prematurity- *Cotton et al (1980)* reported a perinatal mortality of 100% at less than 27 weeks, 19.7% between 27 and 32 weeks, 6.4% between 33 and 36 weeks and 2.6% after 36 weeks. The overall mortality has dropped from 126 per 1000 to 42-81 per 1000 with conservative management.
2. Intrauterine growth retardation- This may occur in up to 16% of cases. The incidence is higher in those with multiple antepartum haemorrhage.
3. Congenital malformation- The incidence is doubled in women with placenta praevia. The most common are those of the central nervous, cardiovascular, respiratory, and gastrointestinal systems.
4. Other risks- Include umbilical cord complications such as compression and prolapse, malpresention, fetal anaemia, etc

Management of any patient with bleeding in late pregnancy should be in the hospital with adequate facilities for transfusion, delivery by caesarean section and neonatal resuscitation and intensive care. Initial management must include a brief history, evaluation of patient's general condition and initiation of various laboratory tests and treatment. In taking quick history, initiating factors such as trauma , coitus, the amount and character of bleeding, association of abdominal pain or regular uterine contractions, a history of ruptured membranes or previous vaginal bleeds, known gestational age either by previous ultrasound scan or by last menstrual period, information about placenta sites from previous scans and fetal movement must be enquired.

The physical examination is aimed at assessing both maternal and fetal conditions and should include the following:

measuring maternal pulse blood pressure and respiratory rate, looking for clinical evidence of shock such as restless, pallor, cold and clammy extremities. Abdominal examination is done to ascertain whether the uterus is compatible with estimated gestational age, the presence of tenderness, the number of fetuses and their viability, and the presence of uterine contractions. Unless placenta praevia has been excluded, this is usually confined to inspection only in order to assess quickly the amount of blood loss and to determine whether bleeding has stopped or is continuing. Where placenta praevia has been excluded, a speculum examination may be performed and , if bleeding is suspected to be fetal, the Apt test can be performed.

As a general rule all patients with antepartum haemorrhage should be initially investigated and managed as outlined below, but subsequent management should determined by the severity and type of bleeding and the gestational age of the pregnancy. The initial management should be the following :

1. An intravenous line with a wide bore cannula preferably size 14-16 must be established.

2. Blood must be obtained for immediate haemoglobin and haematocrit estimation, full blood count and grouping and reserving of serum. If the bleeding is continuing or is heavy , then at least 4 units of blood must be cross matched. Where abruptio placenta is suspected, a coagulation profile, urea and electrolytes and liver function test is performed.

3. Intravenous fluid should be given if there is continuing bleeding or is severe while crossmatched is being awaited. Colloid are most suitable fluids in such situation.

4. Ultrasound scan to exclude placenta praevia if this has not been done or to exclude a major abruption with placental separation if it is suspected. However this should only be performed if and when maternal and fetal conditions are stable.

After this initial management, the patient may fall into one of the following categories where;

a) The bleeding has stopped.
b) The bleeding is continuing but remains mild or moderate and non life threatening.
c) The bleeding is continuing and is severe and life threatening.
d) The fetus is in distress irrespective of bleeding pattern.
e) The fetus is dead.

If the fetus is alive and vaginal bleeding is present on admission to hospital, a sample of vaginal blood should be collected as soon as possible and tested for the presence of fetal haemoglobin . A digital examination is absolutely contraindicated when it highly suspicious of placenta praevia except in theatre when the termination of pregnancy is forced by bleeding or labour, or when the pregnancy has reached an adequate gestation for safe termination. The really dangerous haemorrhage is often the one that has been provoked by ill-advised obstetric interference, such as digital examination of cervical canal at or very shortly after the time of the warning haemorrhage. Although it is widely appreciated that vaginal examination may provoke separation of placenta with massive haemorrhage, it is sometimes not realized that rectal examination is even more dangerous, as it virtually certain that the placenta will not be detected by the examining finger before it separates and serious bleeding is provoked (*Scott et al 1981*).