Beischer & MacKay’s Obstetrics, Gynaecology and the Newborn is a highly illustrated, evidence-based resource on the theory and practice of caring for women and their newborn babies.

Drawing on the clinical experience and research base of the specialist authors, Beischer & MacKay’s Obstetrics, Gynaecology and the Newborn provides a solid foundation of knowledge across obstetrics, gynaecology and neonatal paediatrics to ensure expert care.

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“The image of delivery of a healthy infant remains the greatest experience patients will enjoy in their journey through life”
Norman Beischer AO, MD, MGO, FRCS(Ed), FRCOG, FRACS, FRACOG, D Med. Science (Honi)
Beischer & MacKay’s
OBSTETRICS, GYNAECOLOGY AND THE NEWBORN
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Since the third edition of *Obstetrics and the Newborn* was published in 1997, there have been enormous changes in the practice of care before, during and after pregnancy, and in gynaecological management of both pre- and postmenopausal women. Routine investigations are more numerous and sophisticated. Caesarean section rates have doubled, but the image of delivery of a healthy infant remains the greatest experience patients will enjoy in their journey through life. Cooperation between carers, midwives, medical officers, and both clinical and laboratory scientists remains an important requirement.

Likewise, in gynaecology the number of subspecialties has increased and marvellous new technology and investigative procedures have altered the techniques of surgical procedures. A new range of drugs have given respite from infertility, and improved chemotherapy has significantly advanced the treatment of gynaecological cancers.

Unusually, this volume combines the disciplines of obstetrics, neonatal medicine and gynaecology into a single source that facilitates learning for medical students, midwives and postgraduates. This creates harmony, from adolescent gynaecology through to reproductive life and the management of pregnancy, and finally the gynaecological problems of the postmenopausal woman.

This volume contains a unique collection of photographs taken many years before restrictions in teaching hospitals, which have now made collecting clinical photographs more difficult.

In my view there are no more appealing medical occupations available than the practice of obstetrics, neonatal care and all aspects of gynaecology.

It should not be overlooked that in spite of modern changes in practice, the vast majority of women—with conventional methods of support—continue to have a normal labour with spontaneous delivery of a normal, healthy infant.

We practitioners should aim to be the custodians of normality.

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Early pregnancy assessment clinics (EPAC) are now common in hospitals managing pregnancy. They help to streamline the diagnosis and management of early pregnancy bleeding and provide a point of contact for ongoing medical care and support. Table 8.1 gives a simple differential diagnosis for bleeding in early pregnancy.

In assessing the patient with early pregnancy bleeding, two important clinical questions need to be answered.

1. Is the pregnancy intrauterine?
2. Is the pregnancy viable?

Is the pregnancy intrauterine?

All patients with bleeding and a non-localised pregnancy should be considered to have an ectopic pregnancy until proven otherwise. Ultrasound (usually transvaginal) and quantitative serum β-hCG form the basis for localising the pregnancy (Fig 8.1). The key component of this algorithm is the use of transvaginal ultrasound irrespective of the serum β-hCG. While intrauterine pregnancy contents are not expected to be visualised if the β-hCG < 1500 IU/L, an ectopic pregnancy with an inappropriate β-hCG for gestation may still be seen.

Is the pregnancy viable?

Once an intrauterine pregnancy is confirmed, its viability should be determined. Serum β-hCG has little to offer in the management of an intrauterine pregnancy and the presence of fetal heart activity confirms viability. Any one of the following criteria is indicative of a failed pregnancy according to the guidelines of the Australian Society of Ultrasound in Medicine (ASUM):

- mean gestational sac diameter > 25 mm with no fetal pole
- fetal pole > 7 mm and no fetal heart activity
- inadequate growth of the gestational sac or fetal pole over the course of a week (i.e. < 1 mm per day).

Poor prognostic features include a bradycardia (< 85 bpm) and significant subchorionic haematoma formation (Fig 8.2).

Miscarriage

A miscarriage is the presence of a non-viable intrauterine pregnancy before 20 weeks. It is not necessary for there to be an embryo or fetus present. Clinically recognised miscarriage occurs in approximately 15% of pregnancies.
If unrecognised biochemical pregnancies are included, the figure is much higher.

### AETIOLOGY

The aetiology of miscarriage is as follows.

- **Chromosomal abnormalities.** These are the most common cause of miscarriage and are responsible for approximately 50% of spontaneous miscarriages. Most of these abnormalities are non-recurring and include trisomies (e.g. trisomy 13), monosomy X (Turner syndrome) and polyploidies (triploidy and tetraploidy). The incidence of autosomal trisomies increases with advancing age.

- **Endocrine.** Poorly controlled endocrine disorders are risk factors for both infertility and miscarriage.

- **Thrombophilia.** Antiphospholipid syndrome is a cause of recurrent miscarriage. The association of other hypercoagulable states and miscarriage is less certain as there are conflicting reports. It may be that thrombophilia has a greater association with late (> 10 weeks') first trimester miscarriage.

- **Preexisting diabetes, thyroid disease and hyperandrogenism** (e.g. polycystic ovary syndrome [PCOS]) are associated with miscarriage. Obesity is also an independent risk factor for miscarriage. Although progesterone is essential for successful implantation and continuation of pregnancy, studies have shown neither a consistent correlation between progesterone levels and risk of subsequent miscarriage nor a benefit in reducing miscarriage with the use of exogenous progesterone.

---

### TABLE 8.1  DIFFERENTIAL DIAGNOSIS OF EARLY PREGNANCY BLEEDING.

<table>
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<td>Not pregnant</td>
<td>In rare cases, a woman will have a false positive pregnancy test or for some other reason believe she is pregnant; bleeding may therefore be a menstrual period</td>
</tr>
<tr>
<td>Intrauterine pregnancy</td>
<td>Viable intrauterine pregnancy</td>
</tr>
<tr>
<td></td>
<td>Non-viable (miscarriage, gestational trophoblastic disease)</td>
</tr>
<tr>
<td>Ectopic pregnancy</td>
<td>Most commonly tubal but may be ovarian, cervical or abdominal</td>
</tr>
<tr>
<td>Incidental cause for bleeding</td>
<td>Cervical polyp/cancer, ectropion, other genitourinary tract cause</td>
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### FIGURE 8.1

Management of bleeding in early pregnancy.

Early pregnancy bleeding +/- pain

- Transvaginal ultrasound
- Intrauterine pregnancy
  - Viable
  - Non-viable
- Extrauterine pregnancy
- Unlocalised pregnancy
- B-hCG < 1500 IU/L
- B-hCG > 1500 IU/L
- Highly suspicious of ectopic pregnancy
- Consider admission
- Repeat B-hCG in 48 hours
- Appropriate rise in B-hCG
- Inadequate rise in B-hCG
- Repeat ultrasound in 1 week
- Manage accordingly
- Senior obstetric review

---
**Chapter 8  Bleeding in Early Pregnancy**

**Bleeding**

The amount of bleeding with a miscarriage is variable. In complete miscarriage, the loss is variable during the process but thereafter usually ceases entirely. In the majority of women with threatened miscarriage, the pregnancy continues uneventfully. The risk of loss is usually proportional to the amount of bleeding; if the loss continues or recurs or if there is any associated pain, the prognosis is less favourable. First-trimester bleeding predisposes to later pregnancy complications including preterm birth and preterm prelabour rupture of the membranes.

**Pain**

Pain is experienced as the uterus contracts or when the cervix is dilating and products of conception are being passed. The pain is felt in the lower abdomen or back, is usually cramp-like and follows the bleeding—in contrast to the sequence in ectopic pregnancy, discussed later in this chapter.

**Passage of products of conception**

The passage of definite tissue defines incomplete or complete miscarriage. A confusing picture is presented when the decidual lining of the uterus is passed in ectopic pregnancy (decidual cast), since this simulates trophoblastic tissue (Fig 8.3). In many women, no fetus is passed at any time, since it is either absent (blighted ovum) or rudimentary and unnoticed.

**CLASSIFICATION**

Miscarriages were traditionally classified according to clinical criteria as listed in Table 8.2; that is, whether the cervix is open/closed, whether any products of conception have been passed and the size of the uterus relative to dates. More recently, however, ultrasound has played a more important role in the diagnosis and classification of miscarriage. The far greater availability of first-trimester ultrasound has led to the diagnosis of significantly more missed miscarriages in asymptomatic women. Note that the term ‘missed miscarriage’ means a ‘non-viable pregnancy that has not yet had any vaginal bleeding’. It does not mean that the diagnosis has not been made.

**INITIAL MANAGEMENT**

**Assessment**

Take the patient’s history to elicit details of the presenting complaint as outlined in the Clinical features section. Clinical examination should include vital signs, abdominal palpation, speculum examination and bimanual palpation. The following investigations are useful: full blood examination (FBE), blood group and save serum, quantitative β-hCG and ultrasound.
should be asked to present earlier if she experiences significant pain or bleeding.

**Inevitable miscarriage**
While the patient will go on to pass the products of conception, this may occur after a variable amount of further bleeding and/or pain. Expectant or medical management is most appropriate. Occasionally patients will require a curette if there is sustained heavy bleeding.

**Incomplete miscarriage**
This common presentation causes the most trouble from bleeding and shock; blood transfusion may be required in a few women.

Cervical shock may occur if products become trapped within the cervix. This presents as bleeding, significant pain and often vagal/parasympathetic symptoms (bradycardia, hypotension, sweatiness and nausea/vomiting). Speculum visualisation of the cervix and removal of the products with sponge forceps may be necessary (Fig 8.4). It is often necessary to perform a curette to ensure complete removal of products of conception. If there is no evidence of cervical tissue and bleeding is heavy and ongoing, ergometrine 0.5 mg intravenously may be trialled while theatre is arranged for an urgent curette.

Usually, the bleeding is much less troublesome, and after the diagnosis has been made, ongoing management options are discussed, as detailed in the next section.

**Missed miscarriage**
Ultrasound criteria for confirming a non-viable pregnancy are described earlier in this chapter. Where there is no suggestion of a septic miscarriage or haemodynamic instability, ongoing management may be expectant, medical or surgical. A Cochrane review has revealed that the rates of infection are similar for each management strategy. While the likelihood of success is a critical factor in determining ongoing management, other issues to consider include the patient’s:
- tolerance of potential ongoing bleeding and/or pain at home
- acceptance of having a complete miscarriage in a setting outside of the hospital

**FIGURE 8.4**
Incomplete miscarriage at 10 weeks’ gestation in a 21-year-old primigravida who presented with heavy vaginal bleeding and abdominal pain. A Placental tissue is seen protruding through the cervical os. Sponge-holding forceps grasp and remove the placental tissue. B Products of conception visible at a partly dilated external os.
Source: Courtesy of Prof. Norman Beischer.
< 5 IU/L. Patients should present if they experience significant abdominal pain and or bleeding. If the β-hCG level does not drop by 15%, consideration is given to either a second dose of methotrexate or surgical management.

The multi-dose regimen has a similar success rate to the single dose for tubal ectopics; however, side effects are more common. It is generally only considered in cases of cornual, scar and cervical ectopic pregnancy.

**Surgical treatment**

Surgical management is indicated if medical treatment is contraindicated (as outlined earlier in Box 8.1) or if medical treatment fails or is declined by the patient.

**Laparoscopy or laparotomy?**

Laparoscopy is appropriate for almost all ectopic pregnancies (even those with haemoperitoneum) except those with significant haemodynamic compromise. Ultimately the decision depends on the experience and skills of the attending gynaecologist and anaesthetic team.

**Salpingectomy or salpingostomy?**

Laparoscopic salpingectomy (Fig 8.11) is the standard surgical treatment for tubal ectopic pregnancy. Research has shown similar rates of future intrauterine pregnancy and recurrent ectopic rates compared with medical management.

Salpingostomy (incision of the affected tube with removal of the products of conception) is occasionally performed with a view to increasing future intrauterine pregnancy rates. Non-randomised studies suggest that intrauterine pregnancy rates are greater after salpingostomy compared with salpingectomy ONLY in those with contralateral tubal disease. Against salpingostomy, there is increase in both persistent trophoblast requiring treatment and future ectopic pregnancy relative to salpingectomy.

**Other treatment options**

Live cervical, scar and cornual ectopics may be managed with intra-gestational potassium ± methotrexate injection.

**GESTATIONAL TROPHOBLASTIC DISEASE**

**CLASSIFICATION**

Gestational trophoblastic disease (GTD) encompasses a range of conditions characterised by a proliferative disorder of trophoblastic cells. These disorders can be broadly classified into benign or invasive/malignant (see Box 8.2) and are largely distinguished on histopathology.

**Localised GTD**

Localised molar pregnancies are a result of aberrant fertilisation and as such are primary entities and cannot follow from a clinical pregnancy. Invasive GTD, on the other hand, may follow either a molar or clinical pregnancy including term, preterm, miscarriage or ectopic pregnancy.
Mole
A hydatidiform mole is characterised by oedematous avascular villi with trophoblastic proliferation (Fig 8.13). Typically, a mole is initially diagnosed following an ultrasound performed because of vaginal bleeding (95%) in early pregnancy. Other clinical features may include hyperemesis gravidarum, theca lutein cysts (Fig 8.14), preeclampsia (may be before 20 weeks’ gestation), hyperthyroidism and vaginal passage of hydropic vesicles. Symptoms from metastatic spread of molar tissue may also occur (e.g. haemoptysis and/or pleuritic pain from spread to the lung).

Choriocarcinoma
The clinical presentation of choriocarcinoma is varied depending on the antecedent pregnancy. Following a complete molar pregnancy, it may be diagnosed in asymptomatic women with routine \( \beta \)-hCG monitoring.

Invasive GTN
In the case of invasive moles, the degree of invasion may be local or may involve metastases, usually to the lungs or vagina. Invasive moles follow approximately 15% of complete moles and 3% of partial moles. The diagnosis is generally made clinically based on persistent \( \beta \)-hCG elevation after molar evacuation.

Choriocarcinoma is a malignant disease characterised by abnormal trophoblastic hyperplasia and anaplasia, absence of chorionic villi, haemorrhage and necrosis. This malignant tumour of the trophoblast follows a hydatidiform mole in 50% of cases, normal pregnancy in 25% and miscarriage or ectopic pregnancy in 25% (see Fig 8.12).

PREDISPOSING FACTORS
Rates vary significantly based on geographic region, ethnicity and maternal age. The condition is more common in Australasia (1 in 750 pregnancies) than in the United States, the United Kingdom and Europe (1 in 1500), but is most frequent in South-East Asia and Mexico (1 in 500). Choriocarcinoma is more common in older and younger women and there is a 1% recurrence risk if there is a past history of GTD.

HISTOPATHOLOGY AND CYTOGENETICS
Complete and incomplete moles differ significantly in certain characteristics (Table 8.4). Complete molar pregnancy occurs as a result of fertilisation of an empty ovum with either two sperm or one that divides. The karyotype is generally 46XX (occasionally 46XY) and all of paternal origin. Partial molar pregnancy results from the fertilisation of an ovum with two sperm or one that divides resulting in triploidy 69XXX, 69XXY or, rarely, 69XYY. There is often a co-existing fetus that is prone to fetal death in utero and growth restriction. Partial moles have a much lower malignant potential.
TABLE 8.4  CHARACTERISTICS OF PARTIAL AND COMPLETE MOLES.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Partial mole</th>
<th>Complete mole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Karyotype</td>
<td>69XXY, 69XXX, 69XYY</td>
<td>46XX or 46XY</td>
</tr>
<tr>
<td>Embryonic fetal tissue</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>Villi</td>
<td>Focal hydropic villi</td>
<td>Diffusely hydropic</td>
</tr>
<tr>
<td>Theca lutein cysts</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>Malignant GTN</td>
<td>3%</td>
<td>15% (invasive mole 90%, choriocarcinoma 10%)</td>
</tr>
<tr>
<td></td>
<td>Almost exclusively invasive mole</td>
<td></td>
</tr>
</tbody>
</table>

FIGURE 8.13
Section of a hydatidiform mole showing considerable trophoblastic proliferation. The chorionic villi show the typical enlargement (hydrops) and avascularity.
Source: Courtesy of Norman Beischer.

FIGURE 8.14
Uterus and bilateral theca-lutein cysts seen after hysterectomy at 18 weeks’ gestation in a woman with a hydatidiform mole. Suction curettage is the usual treatment of a hydatidiform mole.
Source: Courtesy of Monash Health.

Following a normal pregnancy, persistent vaginal bleeding is the most frequent symptom. Vaginal bleeding after 6 to 8 weeks’ should prompt consideration of GTN along with the other more common conditions such as retained products of conception and endometritis. There may be evidence of metastatic tumour—vaginal metastases are present in 30% cases. These lesions are highly vascular and prone to bleeding. Patients may exhibit respiratory, gastrointestinal or neurological symptoms reflecting sites of distant metastases.

DIAGNOSIS

Ultrasound
Ultrasound of a complete mole reveals a central heterogeneous mass with numerous discrete anechoic spaces. There is no fetus or amniotic fluid (Fig 8.15)—unless there is a co-existent twin (4 to 6%, Figs 8.16 and 8.17). A partial mole may also have a co-existent fetus. A choriocarcinoma appears as a hypervascular heterogeneous mass.

β-hCG
β-hCG is universally elevated as compared with other intrauterine or ectopic pregnancies. Occasionally, the diagnosis of a partial mole may only be made on histology of curettings from a suspected incomplete abortion.

MANAGEMENT

Localised GTD
Initial management
The initial treatment is similar to that outlined for the woman with suspected miscarriage. Heavy bleeding may complicate this process, so adequate blood must be cross-matched. Suction curette is first-line management for complete and partial molar pregnancy. Hysterectomy
would rarely be performed as first line management (Fig. 8.18). Tissue is sent in normal saline (not formalin) for histological analysis and karyotyping if necessary.

If the diagnosis of a complete mole is established preoperatively, baseline investigations with a quantitative β-hCG and chest X-ray should be taken.

**Follow-up**

Due to the possibility of persistent GTD (invasive molar pregnancy/choriocarcinoma), especially after complete molar pregnancy, ongoing follow-up with serial β-hCG measurements for 6 months is important. Contraception should be commenced and if there is a trophoblast registry in the region, the case should be notified. Use of the oral contraceptive pill does not increase rates of invasive disease. The theca lutein cysts are managed conservatively although they may take months to resolve. Surgery may be necessary if torsion ensues.

**Invasive GTN**

**Initial management**

The preferred management of invasive GTN is with chemotherapy. Repeat curette is contraindicated due to the significant risk of maternal haemorrhage and uterine perforation. Chemotherapy is the cornerstone of management. ‘Staging’ into low- and high-risk groups takes place to determine the most appropriate chemotherapy. Low-risk women receive single-agent chemotherapy but the high-risk group are prescribed a multi-agent chemotherapeutic regimen (etoposide, methotrexate, actinomycin D, cyclophosphamide and vincristine [EMA/CO]). Cure rates approach 100% for those treated with single-agent chemotherapy. For high-risk disease necessitating a multi-drug regimen ± adjuvant radiotherapy or surgery, cure rates are approximately 90%.

**Follow-up**

Once chemotherapy has been completed and β-hCG levels have returned to normal, the β-hCG levels should be monitored at monthly intervals for a further 12 months. The risk of relapse is 3% in the first year and rare after that. Pregnancy should be avoided during the first 12 months.
after \( \beta \)-hCG returns to normal in order to facilitate \( \beta \)-hCG surveillance. An effective method of contraception should be used. The combined oral contraceptive pill is considered safe. Due to the risk of recurrence in subsequent pregnancies, women should have an ultrasound in the first trimester, placental histopathology and a serum \( \beta \)-hCG at 6 weeks.