Anaesthesia for non-obstetric procedures during pregnancy

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Key points
Avoid or minimize aortocaval compression.
Perform surgery in the second trimester where possible. Avoid elective surgery until after delivery.
Regional anaesthesia is preferred to general anaesthesia where possible.
Maintain normal maternal physiology. Optimize uteroplacental perfusion and avoid fetal asphyxia.
Avoid unwanted drug effects on the fetus, but be aware that no anaesthetic agents have been shown to be teratogenic in the clinical use.

The pregnant patient can have any disease that any other woman can have—except sterility.

Dr Frederick C. Irving

Up to 2% of pregnant women undergo surgery for non-obstetric conditions each year. The most common indications are acute appendicitis, cholecystitis, trauma, and surgery for maternal malignancies. The main risks of surgery during pregnancy are fetal loss, premature labour, and delivery, which can result from both the disease process itself and the intervention. Intra-abdominal procedures for inflammation immediately adjacent to the uterus are more likely to result in uterine irritability, and the risk of preterm labour or abortion is significantly higher. The anaesthetist must provide safe anaesthesia to the mother while minimizing the risks to the developing fetus.

Maternal considerations
It is important to understand the physiological changes of pregnancy and the effect of drugs on the mother. Maternal physiology changes rapidly from the first trimester, owing to the hormonal effect of increasing progesterone production by the placenta and increased metabolic demands, and, from the second trimester, the mechanical effects of an enlarging uterus. A summary of the major physiological changes and their anaesthetic implications is presented in Table 1.

Fetal considerations
The safety of the fetus is affected by the placental transfer of drugs and by factors predisposing to fetal asphyxia, preterm labour, and delivery. Teratogenicity should not be ignored (see the Anaesthetic drugs and teratogenicity section), but maintaining uteroplacental perfusion is likely to be the main challenge. Maternal hypoxaemia leads to uteroplacental vasoconstriction, reduced uteroplacental perfusion, fetal hypoxaemia, acidosis, and, ultimately, fetal death. Maternal hypercapnia, which may occur during spontaneous ventilation and deep levels of anaesthesia, causes fetal respiratory acidosis, uterine vasoconstriction, and reduced uterine blood flow. Moderate elevations of fetal Pco2 are probably well tolerated, but severe fetal acidosis may cause myocardial depression. Hypocapnia also causes uterine vasoconstriction and a shift in the maternal oxyhaemoglobin dissociation curve to the left, resulting in reduced oxygen release to the fetus. Excessive positive pressure ventilation leads to maternal hypocapnia, increased intrathoracic pressure, reduced venous return, and reduced uterine blood flow. Maternal hypotension of any cause should be treated immediately. Uterine hypertension is associated with an increase in uterine vascular resistance and will decrease uterine blood flow.

Timing of surgery
Timing seems to be critical to fetal outcome, and a decision on proceeding with surgery should be made by a multidisciplinary team, involving surgeons, anaesthetists, and obstetricians. An overall miscarriage rate after surgery of 5.8% has been reported, increasing to 10.5% during the first trimester. During the first 2 weeks of gestation, the fetus is either lost or preserved intact. During the period of organogenesis between the 3rd and 8th weeks, exposure to teratogens can cause major structural organ abnormalities. After this period, drug exposure can cause functional changes or fetal growth retardation, but structural abnormalities are rare. In the advanced stages of pregnancy, the difficulties of manoeuvring around a large gravid uterus and the management of the maternal airway need to be considered. The more advanced the pregnancy, the greater the chance of maternal hypoxia, which increases the risk of fetal asphyxia.

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Table 1 Physiological changes in pregnancy. CO, cardiac output; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance; AP, arterial pressure; ERV, expiratory reserve volume; RV, residual volume; FRC, functional residual capacity; V/VQ, ventilation/perfusion; MAC, minimum alveolar concentration; WCC, white cell count; GFR, glomerular filtration rate

<table>
<thead>
<tr>
<th>System</th>
<th>Physiological change</th>
<th>Anaesthetic implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>† CO up to 50%</td>
<td>Uterine perfusion not autoregulated</td>
</tr>
<tr>
<td></td>
<td>† Uterine perfusion to 10% of CO</td>
<td>Hypotension common under regional and general anaesthesia</td>
</tr>
<tr>
<td></td>
<td>† SVR, ↓ PVR, ↓ AP</td>
<td></td>
</tr>
<tr>
<td>Aortocaval compression from 13 weeks</td>
<td></td>
<td>↑ V/Q mismatch</td>
</tr>
<tr>
<td>Respiratory</td>
<td>† Minute ventilation</td>
<td>Faster inhalation induction</td>
</tr>
<tr>
<td></td>
<td>(Paco2 3.7–4.2 kPa)</td>
<td>Maintain Paco2 at normal pregnancy levels</td>
</tr>
<tr>
<td></td>
<td>† ERV, ↓ RV, ↓ FRC</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↑ V/Q mismatch</td>
<td></td>
</tr>
<tr>
<td></td>
<td>† Oxygen consumption</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Upward displacement of diaphragm</td>
<td>Potential hypoxaemia in the supine and Trendenenburg positions</td>
</tr>
<tr>
<td></td>
<td>↑ Thoracic diameter</td>
<td>Breathing more diaphragm than thoracic</td>
</tr>
<tr>
<td></td>
<td>Mucosal oedema</td>
<td>Difficult laryngoscopy and intubation; breathing during attempts</td>
</tr>
<tr>
<td>CNS</td>
<td>† Epidural vein engorgement</td>
<td>Bloody tap more common</td>
</tr>
<tr>
<td></td>
<td>† Epidural space volume</td>
<td>More extensive local anaesthetic spread</td>
</tr>
<tr>
<td></td>
<td>† Sensitivity to opioids and sedatives</td>
<td></td>
</tr>
<tr>
<td>Haematological</td>
<td>† Red cell volume 30%, † WCC</td>
<td>Dilutional anaemia</td>
</tr>
<tr>
<td></td>
<td>† Plasma volume 50%</td>
<td>Thromboembolic complications (DVT prophylaxis)</td>
</tr>
<tr>
<td></td>
<td>† Coagulation factors</td>
<td></td>
</tr>
<tr>
<td></td>
<td>↓ Albumin and colloid osmotic pressure</td>
<td>Oedema, decreased protein binding of drugs</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>† Intragastric pressure</td>
<td>↑ Aspiration risk</td>
</tr>
<tr>
<td></td>
<td>† Barrier pressure</td>
<td>Antacid prophylaxis, RSI after 18 weeks gestation</td>
</tr>
<tr>
<td>Renal</td>
<td>† Renal plasma flow, † GFR</td>
<td>Normal urea and creatinine may mask impaired renal function</td>
</tr>
<tr>
<td></td>
<td>↓ Reabsorptive capacity</td>
<td>Glycosuria and proteinuria</td>
</tr>
</tbody>
</table>

of uterine irritability and preterm labour. There is no evidence to suggest that any anaesthetic agent, dose, or technique influences this risk; it is more likely to result from the disease process itself and direct manipulation of the uterus during surgery. Lower abdominal and pelvic inflammatory conditions, such as acute appendicitis with peritonitis, are associated with a particularly high risk.

The second trimester is preferred for semi-elective surgery which cannot be deferred. Elective surgery should be postponed until at least 6 weeks postpartum, to allow resolution of physiological changes. However, urgent surgery should not be delayed as secondary complications may increase the risk to both mother and fetus.

Anaesthetic management

The choice of anaesthetic technique should be guided by the indications, nature, and site of the surgical procedure. Laparoscopic rather than open techniques may be preferred when abdominal surgery is undertaken.

Pre-anaesthetic visit and premedication

Verbal reassurance is usually preferred to pharmacological premedication. A standard preoperative assessment is carried out with careful attention to the airway. Gestational age should be noted, and the possibility of miscarriage and preterm labour should be discussed. The mother should be informed about the low risks of teratogenicity associated with anaesthetic agents in the current use (see the Anaesthetic drugs and teratogenicity section). The obstetric team should be involved, and also a paediatrician if preterm labour is anticipated. Antacid prophylaxis is recommended after 14 weeks of gestation, and deep venous thrombosis prophylaxis should always be considered.

Choice of anaesthetic technique

Regional anaesthesia is preferred to general anaesthesia where feasible. This enables the mother to maintain her own airway, minimizes fetal drug exposure, and provides good postoperative analgesia. Nevertheless, evidence demonstrating superior safety is lacking, and general anaesthesia is frequently required.

Monitoring

In addition to the standard monitoring, cardiotocographic (CTG) monitoring may be considered after 24 weeks of gestation. This would only be warranted if continuous observation of the CTG monitor is possible in theatre and an obstetrician is available for immediate delivery if necessary. Before 24 weeks, confirmation of fetal well-being should be performed at an appropriate time in the postoperative period. The loss of fetal heart rate variability may be related to the administration of drugs rather than fetal distress.

Conduct of general anaesthesia

Avoid aortocaval compression from 20 weeks (or earlier with larger gravid uteri, e.g. polyhydramnios or multiple gestation) by adopting a lateral position when possible, or by maintaining uterine displacement manually or through tilt when supine. Head-up tilt may help increase functional residual capacity (FRC), reduce breast interference with intubation, and alleviate gastrointestinal reflux. Airway management, mask ventilation, laryngoscopy, and intubation may all be challenging due to weight gain, breast engorgement, and increased vascularization, which can predispose to bleeding during intubation attempts. A smaller tracheal tube may be required. Equipment for difficult intubation should be available. Preoxygenation with a close-fitting mask for at least 3 min is recommended, as hypoxia develops up to three times more quickly in pregnancy, due to reduced FRC and increased oxygen consumption. Pregnant women are at higher risk of regurgitation and aspiration during both induction and emergence.
Lower oesophageal sphincter tone is reduced from early gestation and intra-abdominal pressure increases from the second trimester, so rapid-sequence induction with cricoid pressure is recommended.

Once the airway is secured, ventilation should aim to keep the \( P_{\text{CO}_2} \) in the normal range for pregnancy. Care must be taken to avoid excessively raised intra-abdominal pressures during laparoscopy.\(^5\) Pregnancy is associated with an increased sensitivity to volatile anaesthetic agents, with MAC values slightly reduced. All volatile agents up to an MAC of 1.5 dilate uterine arteries and increase uterine blood flow, but at higher concentrations, this is offset by decreases in maternal arterial pressure and cardiac output. Volatile agents also reduce uterine tone. Nitrous oxide is avoided during the first trimester (see the Anaesthetic drugs and teratogenicity section). Light anaesthesia and pain are avoided to prevent maternal catecholamine release and consequent reduced uteroplacental perfusion. Extubation should be done with the patient fully awake and, preferably, in the lateral position.

**Regional anaesthesia**

Regional anaesthesia is highly desirable, although there are particular considerations during pregnancy. Obesity and oedema can obscure anatomical landmarks. Interspinal ligaments are hormonally softened, causing difficulty with epidural loss of resistance techniques. The spread of the local anaesthetic within the epidural or spinal space is greater in pregnancy, and lower doses are required. This may be due to both the mechanical effect of the enlarging uterus and hormonal changes, increasing sensitivity to local anaesthetic agents. Albumin concentration is reduced, with lower plasma binding and a higher risk of local anaesthetic toxicity.

Sympathetic activity is increased in pregnancy, and the sympathetic block due to central neuraxial block can cause significant maternal hypotension in the presence of hypovolaemia. Hypotension needs to be treated promptly by a left lateral tilt and a vasoconstrictor such as phenylephrine, and by giving i.v. fluids in the presence of hypovolaemia. Physiological changes mask the early signs of blood loss and subclinical hypovolaemia will compromise placental perfusion. Hypotension may not be evident until 25–30% of blood volume is lost.

**Tocolytic therapy**

If premature labour occurs, tocolysis will be necessary to preserve the pregnancy. Prophylactic therapy can be considered in the third trimester in patients undergoing lower abdominal or pelvic surgery for inflammatory conditions, although its efficacy during non-obstetric surgery is unproven and its use is also controversial because of maternal side-effects. The use of volatile anaesthetic agents has been advocated as they relax the uterus, although high concentrations can cause undesirable hypotension. Other agents used include magnesium sulphate, \( \beta \)-mimetics (terbutaline), calcium-channel blockers (nifedipine), and vasodilators (GTN).\(^6\)

Prostaglandin synthetase inhibitors, such as indomethacin, are no longer recommended.

**Anaesthetic drugs and teratogenicity**

Almost all anaesthetic agents can be potentially teratogenic,\(^7\) although teratogenicity may also be caused by infection, pyrexia, hypoxia, acidosis, radiation, or the pathological process itself. Teratogenicity depends on genetic predisposition, dose, route of administration, duration, and timing of exposure. Most of our knowledge is based on animal studies, which are difficult to extrapolate, and retrospective human surveys.

The use of nitrous oxide during pregnancy has long been controversial because it inhibits methionine synthetase. Exposure to concentrations \( > 50 \% \) for prolonged periods has been proven to be teratogenic during the peak organogenic period in animal studies. However, no adverse reproductive outcome has been detected in women during short periods of exposure. A Swedish registry study involving 5405 women having anaesthesia and surgery during pregnancy failed to implicate nitrous oxide in adverse perinatal outcome.\(^5\) It would seem prudent to use inhaled concentrations of 50%, to limit the duration of use to reasonable intervals, and to avoid it completely during the first trimester. Some retrospective studies have shown a link between sustained maternal diazepam use and cleft palate defects. A single dose of benzodiazepine has never been associated with teratogenicity. Drugs increasing uterine tone should be avoided, including ketamine and i.v. local anaesthetics. Endogenous or exogenous sympathomimetics can increase uterine vascular resistance, an effect seen in anxious patients or during light general anaesthesia.

All commonly used induction agents, opioids, neuromuscular blocking agents, and volatile anaesthetics can be used in pregnancy. They are not teratogenic when used in clinical concentrations and when maternal physiology is maintained. Thiopental is the most commonly used agent for rapid-sequence induction, although a lower dose may be required.\(^9\) Propofol is increasingly used as an alternative, especially in early pregnancy, as it is not teratogenic in animal studies. Early pregnancy does not appear to decrease the concentration of propofol required for the loss of consciousness.\(^10\) Neuromuscular blocking agents are generally ionized and cross the placenta in only very small amounts. Plasma cholinesterase concentration can be reduced by up to 35% in pregnancy, but recovery from succinylcholine is not usually prolonged, as an increased volume of distribution and relative resistance may offset the effect of lower concentration. The intensity of fasciculations and muscle pain after succinylcholine is generally less, reflecting hormonal changes.\(^11\) Since the introduction of sugammadex, the use of rocuronium has been advocated as an alternative. Atropine may be preferred to glycopyrrolate to antagonize the muscarinic effects of neostigmine during reversal. Neostigmine crosses the placenta and fetal bradycardia can occur, while glycopyrrolate does not. Opioids are highly lipid-soluble and readily cross the placenta. Although brief exposure is safe, long-term exposure will
cause symptoms of withdrawal when the fetus is delivered. Non-steroidal anti-inflammatory drugs in early pregnancy may be associated with increasing fetal loss and in the third trimester may cause the premature closure of the ductus arteriosus. Single doses are unlikely to be harmful. Table 2 summarizes the major side-effects and clinical considerations for anaesthetic agents and adjunctive drugs.

**Summary**

Despite the many concerns, safe anaesthesia and surgery has been demonstrated for a wide range of non-obstetric procedures during pregnancy. The indications for surgery and its timing are major determinants of maternal and fetal outcome.

**Declaration of interest**

None declared.

**References**


Please see multiple choice questions 29–32.