Acute Fetal Hypoxia/Acidosis

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2.1 Definition, Incidence and Main Risk Factors

Fetal hypoxia refers to the condition in which there is decreased oxygen concentration in fetal tissues, and this is insufficient to maintain normal cell energy production by way of aerobic metabolism. Oxygen is supplied to fetal tissues via a long pathway that involves the maternal respiratory system, maternal circulation, gas exchange at the placenta and finally the umbilical and fetal circulations (Fig. 2.1). Problems occurring at any of these levels may result in decreased oxygen concentration in the fetal circulation (hypoxaemia) and ultimately in fetal tissues (hypoxia).

Acute fetal hypoxia refers to the condition in which there is a rapid reduction in oxygen levels, i.e. occurring over the course of a few minutes. Its main causes are considered in Table 2.1.

In the absence of oxygen, fetal cells may continue to produce the energy required for maintenance of basic homeostatic functions during a few more minutes, by resorting to **anaerobic metabolism**. However, the latter yields much less energy than aerobic metabolism and results in the production of lactic acid. The intra- and extracellular accumulation of hydrogen ions, due to increased lactic acid production, results in the development of **metabolic acidosis** (decreased pH caused by acids of intracellular origin) and, because these ions are taken away by the fetal circulation, metabolic acidaemia. The whole process of decreased oxygen concentration in tissues is therefore known as **hypoxia/acidosis**.

Some constituents of fetal blood are capable of neutralising (buffering) hydrogen ions. These are called **bases**, and they include bicarbonate, haemoglobin and plasma proteins. However, their availability is limited, and their depletion (**base deficit**) is directly related to the severity of metabolic acidosis. As there is no direct method of quantifying oxygen concentration within fetal tissues, the only objective way of diagnosing intrapartum fetal hypoxia/acidosis is to measure pH and base deficit in the umbilical cord blood at delivery or in the newborn circulation during the first minutes of life. Metabolic acidosis is defined as a pH below 7.00 and a base deficit in excess of 12 mmol/l (or alternatively a lactate value in excess of 10 mmol/l) in

Fig. 2.1 A representation of
the pathway of oxygen
supply to the fetus

either of these circulations. The umbilical cord does not need to be clamped for
sampling, but it is important to obtain blood from both artery and vein as soon as
possible after birth, to guarantee the quality of results. Sampling of the wrong vessel
may occur when the needle crosses the artery to pierce the vein, and this may also
result in mixed sampling. After blood is drawn into two heparinised syringes, exist-
ing air bubbles are removed and the syringes capped and rolled between the fingers
to mix blood with heparin; blood gas analysis should be performed within the next

Table 2.1 Main causes of acute fetal hypoxia/acidosis

Reversible causes
Uterine hypercontractility
Sudden maternal hypotension
Maternal supine position with aorto-caval compression
Irreversible causes
Major placental abruption
Uterine rupture
Umbilical cord prolapse
Maternal cardiorespiratory disorders
Severe asthma, haemorrhagic shock, cardiorespiratory arrest, pulmonary thromboembolism, amniotic fluid embolism, generalised seizures, etc.
Usually occult causes
Occult cord compression (true cord knot, low-lying cord, nuchal cord with stretching)
Major fetal haemorrhage (fetal-maternal haemorrhage, ruptured vasa praevia)
Specific mechanical complications of labour
Shoulder dystocia
Retention of the after-coming head

30 min. When the difference in pH between the two samples is less than 0.02 and the difference in pCO_2 is less than 5 mmHg (0.7 Kilopascal), samples are likely to be mixed or to have been obtained from the same vessel. When hypoxia/acidosis is of acute onset, there is usually also a large difference in pH between artery and vein.

Increasing concentrations of hydrogen ions that are no longer buffered because of base depletion affect energy production and cell homeostasis, leading to disrupted cell function and ultimately to a cascade of biochemical events that results in cell death. When hypoxia is sufficiently intense and prolonged to disrupt neurological, respiratory and cardiovascular control, this is reflected in reduced **Apgar scores** at birth. Apgar scores however are much less specific indicators of fetal hypoxia than umbilical blood gas values, as they can be affected by other factors such as prematurity, central nervous system depressors administered to the mother, birth trauma without hypoxia (i.e., subdural haematoma), infection, meconium aspiration, congenital anomalies, pre-existing lesions and early neonatal interventions such as vigorous endotracheal aspiration.

The overall incidence of fetal hypoxia/acidosis, as defined by the incidence of newborn metabolic acidosis, varies substantially between different European hospitals, depending on the risk characteristics of the population and on labour management strategies. Reported rates vary between 0.06 and 2.8 %.

The major risk factors for acute fetal hypoxia/acidosis are the ones responsible for its underlying causes: i.e. labour induction and augmentation with prostaglandins or oxytocin are major risk factors for uterine hypercontractility, regional analgesia is a major risk factor for sudden maternal hypotension, and early amniotomy is a risk factor for uterine hypercontractility and umbilical cord prolapse. A detailed description of the risk factors for all causes of acute fetal hypoxia/acidosis is beyond the aim of this book.

2.2   Consequences

Most newborns with metabolic acidosis and low Apgars recover quickly and do not develop short- or long-term functional impairments. However, when fetal hypoxia/acidosis is sufficiently intense and prolonged, changes in neurological function may become apparent in the first 48 h of life, manifested by hypotonia, seizures and/or coma, a situation that is termed **hypoxic-ischaemic encephalopathy**. In its mild forms (grade 1), a short period of hypotonia is documented, but very rarely it evolves into permanent handicap. When the newborn develops seizures (grade 2), the risk of mortality or long-term neurological sequelae is about 20–30 %. When a comatose state occurs (grade 3), perinatal death or long-term handicap is frequent.

Not all cases of neurological dysfunction occurring in the first 48 h of life (neonatal encephalopathy) are caused by fetal hypoxia/acidosis, so to establish the diagnosis of hypoxic-ischaemic encephalopathy, it is necessary to document metabolic acidosis in the umbilical cord or in the newborn circulation in the first minutes of life.

Cerebral palsy of the dyskinetic or spastic quadriplegic types is the long-term neurological sequela most strongly associated with fetal hypoxia/acidosis, although only 10–20 % of cases are caused by this entity. Perinatal infection, congenital diseases, metabolic diseases, coagulation disorders and the complications associated with birth trauma and prematurity constitute the majority of causal factors.

The speed of installation and intensity of acute fetal hypoxia/acidosis varies from case to case, so fetal risk is not uniform. In some cases, there may be a sudden and almost total reduction in oxygen supply, while in others, it may be less intense or of slower onset. The insults can also be transitory and repetitive in nature (uterine hypercontractility, occult cord compression). Finally, there is also some individual variation in the capacity to react to hypoxia/acidosis.

For all these reasons, it is difficult to establish how long a hypoxic insult may last before important injury occurs. However, some information can be extrapolated from cases of sudden maternal cardiorespiratory arrest. No long-term neurological sequelae were reported when the interval between arrest and birth was under 12 min, and perinatal death was common when more than 15 min had elapsed. This evidence is frequently used as an indicator of a 12-min margin of safety for the fetus, in situations where sudden and complete interruption of fetal oxygenation occurs. It is likely that this rule of thumb is only valid for normally grown fetuses at term, receiving adequate oxygenation before the insult occurred, and needs to be adapted in other situations.

2.3   Diagnosis

Acute fetal hypoxia/acidosis almost always manifests as a **prolonged deceleration** – a sudden and sustained decrease in the fetal heart rate (FHR), with an amplitude exceeding 15 bpm and lasting more than 3 min (Fig. 2.2). When the duration exceeds 10 min, it is called **fetal bradycardia**.

Fig. 2.2 Cardiotocographic (CTG) tracing with prolonged FHR deceleration and reduced variability within the deceleration ("paper speed" 1 cm/min)

Decreased oxygen concentration in fetal arterial blood triggers chemoreceptors located near the aortic arch to transmit neurological impulses to brain stem nuclei controlling the vagus nerve and causes a parasympathetically mediated drop in FHR. When fetal hypoxia/acidosis affects the central nervous system, the sympathetic-parasympathetic modulation of FHR is decreased, and this results in diminished signal oscillations, a phenomenon known as **reduced variability** (Fig. 2.2).

Other clinical symptoms and signs may appear in association with a prolonged deceleration, related to the underlying cause of fetal hypoxia/acidosis (see below).

2.3.1   Reversible Causes

The underlying cause of fetal hypoxia/acidosis is frequently reversible, as occurs with uterine hypercontractility, sudden maternal hypotension or aorto-caval compression by the pregnant uterus when the mother is in the supine position.

2.3.1.1   Uterine Hypercontractility

Uterine contractions compress the blood vessels running inside the myometrium, and this may cause a temporary reduction in placental perfusion. The umbilical cord may also be compressed between fetal bony parts or between the fetal head and the uterine wall, transitorily reducing umbilical blood flow. Usually these phenomena occur during the peak of uterine contractions, and the intervals between these events are sufficient to re-establish normal oxygenation. The frequency, duration and intensity of uterine contractions will determine the magnitude of the disturbances, and how much they affect fetal oxygenation.
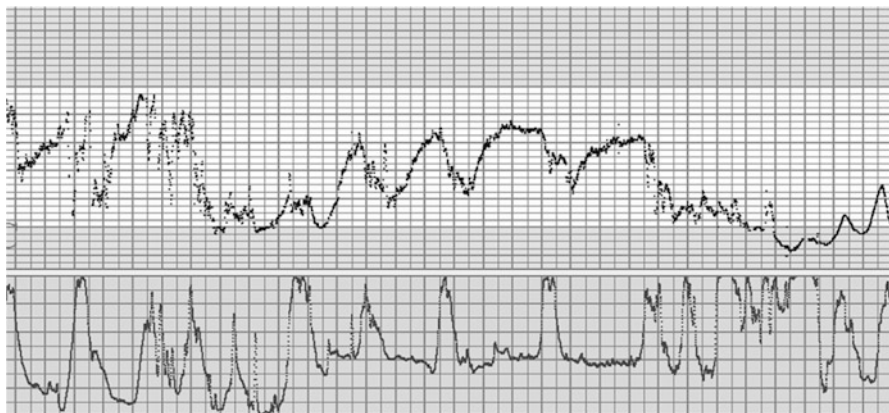
Fig. 2.3 CTG with uterine hypercontractility (tachysystole), prolonged decelerations with attempts to recover between contractions and reduced variability at the end ("paper speed" 1 cm/min)

Hypercontractility may be spontaneous or induced in nature and refers to an increased frequency, intensity and/or duration of contractions leading to reduced fetal oxygenation. Rather than exhibiting a single prolonged deceleration (Fig. 2.2), uterine hypercontractility usually manifests by repetitive decelerations that may merge to become a prolonged deceleration and ultimately exhibit loss of variability but with a tendency for FHR recovery between contractions (Fig. 2.3).

Most cases of uterine hypercontractility are iatrogenic in nature, caused by oxytocin or prostaglandin administration. Local practices for labour induction and acceleration will therefore determine the incidence of this entity, and respecting established doses and intervals for drug administration limits its occurrence. Little is known about the incidence and risk factors of spontaneous uterine hypercontractility, but some cases have been described in association with myometrial infection and partial placental abruption.

Increased abdominal pain is usually referred, but in the context of epidural analgesia, the diagnosis will rely mainly on the detection of increased contraction frequency by cardiotocography (CTG) or on uterine fundus palpation. More than five contractions in 10 min on two successive 10-min periods or averaged in the last 30 min is the definition of **tachysystole** – increased frequency of uterine contractions. With external monitoring of uterine contractions, using a tocodynamometer or fundal palpation, only frequency of uterine contractions can be reliability assessed. Evaluation of their intensity and duration, as well as of basal uterine tone, requires the use of an intrauterine pressure sensor, a technique that is nowadays seldomly used. A sustained rise in uterine contraction baseline or the detection of a permanently contracted uterine fundus is very suggestive of increased basal tone (hypertonus), but intrauterine pressure measurement remains the gold standard for this diagnosis.

2.3.1.2 Sudden Maternal Hypotension

Sudden maternal hypotension is nearly always an iatrogenic complication associated with epidural or spinal analgesia, due to blocking of sympathetic nerves that

regulate vessel tonus. It can manifest by nausea, dizziness, vomiting, blurred vision and loss of consciousness and is usually accompanied by a prolonged deceleration. The drop in blood pressure is usually moderate but sufficient to cause a decrease in placental perfusion and gas exchange.

When epidural analgesia began to be used in labour, maternal hypotension and the resulting CTG changes occurred in almost a third of cases. Prophylactic fluid administration before catheter placement reduced this incidence to about 2%, and recent developments in the technique with lower doses of local anaesthetics have almost eliminated the need for prophylactic fluid administration.

2.3.1.3 Maternal Supine Position with Aorto-Caval Compression

Adoption of the maternal supine position can lead to important aorto-caval compression by the pregnant uterus, with a resulting reduction in placental perfusion and gas exchange. This position has also been associated with uterine hypercontractility due to sacral plexus stimulation. Asking the mother to adopt the upright, half-sitting or lateral recumbent position is usually followed by normalisation of the CTG pattern.

2.3.2 Irreversible Utero-Placental-Umbilical Disorders

These are rare events of an irreversible nature that pose great risk to fetal oxygenation. They include major placental abruption, uterine rupture and umbilical cord prolapse. All of them require rapid delivery to avoid adverse perinatal outcome, and the first two can also be associated with profuse maternal haemorrhage.

2.3.2.1 Major Placental Abruption

Major placental abruption can be defined as a separation between the chorion and decidua of sufficient area to condition fetal oxygenation and/or is associated with maternal haemorrhage of sufficient volume to produce the same effect (Fig. 2.4).

Placental abruption affects about 1% of all labours, but the vast majority of cases are insidious and of small dimension. Placental function needs to be reduced by about 50% before fetal oxygenation is affected. Blood originating from vessels located behind the placenta may detach the fetal membranes and drain to the vagina, or it may accumulate to form a retroplacental haematoma. Occasionally, blood will infiltrate the myometrium and originate a Couvelaire uterus, a structure of petrous consistency that can be palpated through the abdomen when located anteriorly and/or fundally.

The main risk factors for placental abruption are a previous history of similar episodes, hypertensive diseases of pregnancy, abdominal trauma, maternal cocaine consumption, maternal smoking and fetal growth restriction.

Sudden abdominal pain, abdominal tenderness, vaginal bleeding and maternal haemodynamic changes may be present, but frequently the first manifestation is a prolonged deceleration. FHR sounds have been reported to be dulled when there is a large anterior placental haematoma, and in these cases it may be necessary to confirm heart movements on ultrasound. When the presenting part is fully engaged, blood may not exteriorise through the vagina and will accumulate inside the uterine cavity, draining after birth.

Fig. 2.4 Major placental
abruption
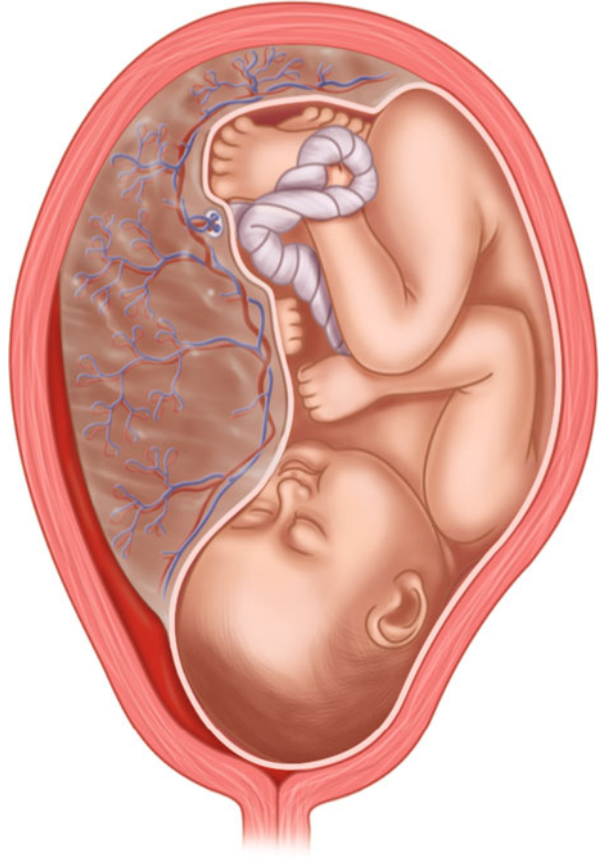
Uterine contractility may be increased in small placental detachments, but in
major abruption, it is generally irregular and inefficient, predisposing to postpar-
tum uterine atony. Myometrial infiltration causes the release of thromboplastins
into the maternal circulation and may result in disseminated intravascular
coagulation.

2.3.2.2 Uterine Rupture

Only about 30 % of cases of uterine rupture are associated with fetal hypoxia/acido-
sis, because lacerations are frequently limited to the caesarean section scar and do
not involve important myometrial vessels irrigating the placental bed nor is there an
accompanying major abruption. Acute hypoxia/acidosis is more frequent when the
fetus is exteriorised into the peritoneal cavity. Maternal mortality associated with
uterine rupture is currently low in high-resource countries. On the other hand, there
is a relatively high rate of peripartum hysterectomy.

Uterine rupture affects about 0.003 % of all births, and the incidence does not
appear to have increased over the last decades. In the majority of cases, there is a

previous history of caesarean section, and the incidence in this population is about 0.1 %. Other risk factors include high multiparity, uterine malformations, oxytocin or prostaglandin use, forceps delivery, placenta percreta, external cephalic version, fetal macrosomia, fetal-pelvic disproportion, abnormal fetal presentation and previous uterine surgery, including curettage and hysteroscopy.

Continuous lower abdominal quadrant pain (3–50 %) and vaginal bleeding (8–12 %) are the most suggestive symptoms of uterine rupture, but a prolonged deceleration (70 %) is frequently the only manifestation. Upward displacement of the presenting part has been reported, but does not seem to be frequent nor is it easy to recognise. Sometimes the diagnosis is only apparent at the time of surgery, where caesarean section scar dehiscence is the most frequent finding. Uterine rupture may extend anteriorly to affect the posterior bladder wall or laterally towards the broad ligaments and the uterine arteries, in the latter case causing severe haemorrhage. Extension to the posterior uterine wall is rare in the absence of previous uterine surgery.

Adherence to established guidelines for labour induction and acceleration and continuous FHR monitoring in women with previous uterine scars are required for avoiding and/or rapidly detecting these situations.

2.3.2.3   Umbilical Cord Prolapse

Umbilical cord prolapse is defined as the presence of a loop of umbilical cord below the presenting part, after the membranes have ruptured. The loop usually passes through the cervix into the vagina (Fig. 2.5), but it can also remain in the uterine cavity or pass through the vaginal introitus to the exterior.
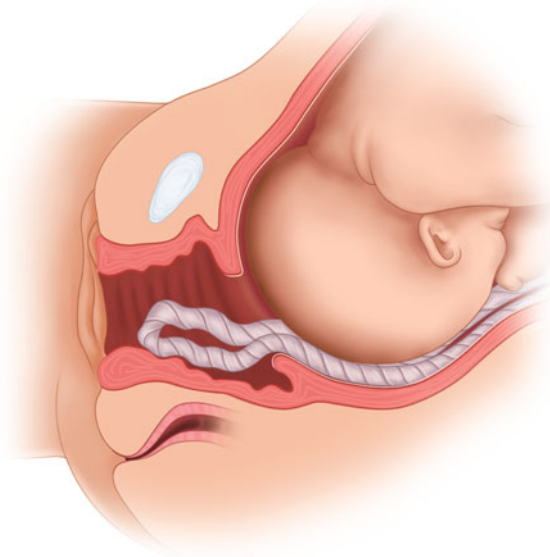

Fig. 2.5 Umbilical cord prolapse

With umbilical cord prolapse, the loop may be continuously compressed between the presenting part and the maternal pelvis, but it can also only be subject to intermittent compressions during contractions. Rarely, no cord compression occurs, a situation that is more frequent with earlier gestational ages, anomalous fetal presentations and in the absence of labour. Cord prolapse can also be complicated by vascular spasm, which has been described when the cord is exposed to cold or is manipulated. During labour, umbilical cord prolapse is almost always associated with repetitive decelerations or with a prolonged deceleration.

Umbilical cord prolapse is reported to affect 0.1–0.6 % of all labours, but the incidence may reach 1 % in breech presentations. Older studies report severe fetal hypoxia/acidosis to occur in 25–50 % of cases, but this number has been decreasing in the last decades, probably due to increased awareness, faster diagnosis and management. Recent studies report perinatal mortality rates between 3.6 and 16.2 %, with causes of death more related to prematurity than to fetal hypoxia/acidosis.

The main risk factors for umbilical cord prolapse are anomalous fetal presentations (transverse lie, knee or footling breech), polyhydramnios, multiparity, long umbilical cord, multiple gestation, preterm labour and low-lying placenta. In a recent case series, 47 % of cases were preceded by an obstetrical intervention such as amniotomy, fetal electrode placement, intrauterine pressure sensor placement and external cephalic version or by expectant management of preterm premature rupture of membranes.

2.3.3   Maternal Cardiorespiratory Disorders

Fetal hypoxia/acidosis may be caused by a number of acute maternal circulatory and respiratory disorders, including severe asthma, haemorrhagic shock, and cardiorespiratory arrest. During labour, however, the most common causes are pulmonary thromboembolism and amniotic fluid embolism. A more detailed description of these complications is provided in Chap. 7.

2.3.4   Usually Occult Causes

It is not always possible to diagnose the causes of a prolonged deceleration before birth, as there is no clear symptom or sign pointing to an aetiology. The occult causes of acute fetal hypoxia/acidosis include occult cord compression and major fetal haemorrhage.

2.3.4.1   Occult Cord Compression
Occult cord compression may occur because of a tight umbilical cord knot, a low-lying loop compressed by the fetal head or a tight nuchal cord that is stretched during descent of the fetal head. Different degrees of compression may occur, resulting in varying levels of circulatory compromise. In the majority of situations, decreased fetal oxygenation and the accompanying FHR changes only occur during

contractions, and the intervals between these events are sufficient to recover fetal oxygenation. The diagnosis is usually retrospective and established at the time of vaginal delivery or caesarean section, but even there low-lying loops may be missed. Depending on the degree of circulatory compromise during and in between contractions, the situation may be relieved by acute tocolysis and by positioning the mother on the left or right lateral, half-sitting or upright positions.

2.3.4.2 Major Fetal Haemorrhage

Fetal haemorrhage of sufficient volume to result in reduced oxygen transport capacity may be chronic, acute or recurring in nature and can be due to fetal-maternal haemorrhage, ruptured vasa praevia or very rarely to lacerations of an umbilical or placental vessel.

In almost all deliveries, there is some degree of **fetal-maternal haemorrhage**, but this is usually of small quantity. A fetal-maternal haemorrhage of more than 20 ml is reported to occur in 0.46 % of births, more than 30 ml in 0.38 % and more than 80 ml in 0.07 %. An increased risk of adverse perinatal outcome is found when blood loss exceeds 20 ml/kg of fetal weight, and about two-thirds of newborns die when it exceeds 80 ml/kg. Other important factors to establish fetal prognosis are the rate of haemorrhage and gestational age. Major fetal-maternal haemorrhage can follow abdominal trauma, external cephalic version, amniocentesis and abruption, and it can also occur with placental chorioangiomas. The vast majority of cases however arise spontaneously and have no identifiable cause. Fetal-maternal haemorrhage is usually asymptomatic, although decreased fetal movements may be reported. The volume of fetal blood present in the maternal circulation should be estimated by the Kleihauer-Betke test or by flow cytometry, and newborn anaemia needs to be documented after birth. Rarely, in the presence of massive haemorrhage, the mother experiences a transfusion reaction with nausea, oedema, chills and fever.

Ruptured vasa praevia refers to the laceration of fetal vessels coursing through the membranes close to the internal cervical *os*, during spontaneous or artificial rupture of membranes. It can occur because of velamentous insertion of the cord or because of vessels running between lobes in a bilobed placenta. The reported incidence is 0.017–0.05 %. Perinatal mortality rate appears to be around 60 % when the situation is diagnosed at membrane rupture, but it can be reduced to 3 % when antenatal diagnosis is followed by elective caesarean section. Ruptured vasa praevia usually presents with vaginal haemorrhage at the time of membrane rupture. Very occasionally the vessels may be palpated on vaginal examination before rupture occurs, and their presence confirmed with transvaginal Doppler ultrasound or with an amnioscope. Risk factors for vasa praevia include bilobed placenta, low-lying placenta diagnosed in the second trimester, multiple pregnancy and in vitro fertilisation.

In major fetal haemorrhage of any cause, a sinusoidal FHR pattern is frequently detected (Fig. 2.6), occasionally with fetal tachycardia during the acute phase, recurrent late decelerations appearing when contractions start or a prolonged deceleration/bradycardia.
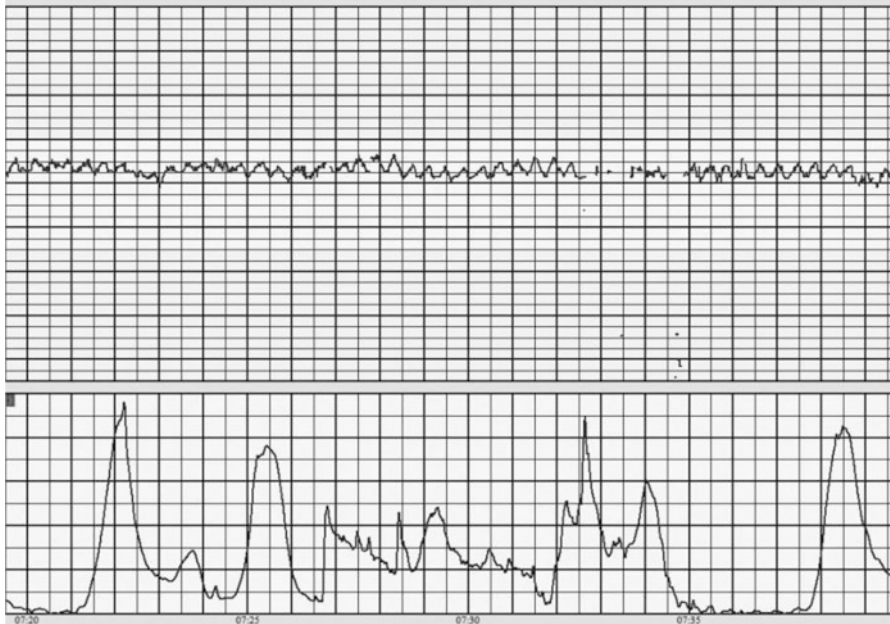
Fig. 2.6 Sinusoidal FHR pattern in fetal-maternal haemorrhage ("paper speed" 1 cm/min)

2.3.5 Specific Mechanical Complications of Labour

Acute fetal hypoxia/acidosis may be caused by specific mechanical complications of labour, associated with umbilical cord compression and/or compression of important fetal blood vessels. The most frequent situations are shoulder dystocia and retention of the after-coming head in vaginal breech delivery. A more detailed description of these situations is provided in Chaps. 3 and 4.

2.4 Clinical Management

The specific management of acute maternal cardiorespiratory arrest, shoulder dystocia and retention of the after-coming head is described in subsequent chapters. The present section is dedicated to management of the remaining causes of acute fetal hypoxia/acidosis.

2.4.1 Immediate Actions in Face of a Prolonged Deceleration

The immediate actions to take when a prolonged deceleration is detected are listed in Table 2.2. The order of these actions may be adapted, if there is a strong suspicion of a specific cause.

Table 2.2   Immediate actions in face of a prolonged deceleration

Assure a continuous and good quality FHR signal
Call for help (two midwives, senior obstetrician)
Stop oxytocin, **remove prostaglandins. Evaluate contractility** (CTG tocograph, palpate uterine fundus to detect tachysystole and/or increased basal tone)
Vaginal examination (to detect umbilical cord prolapse and conditions for instrumental vaginal delivery)
Patient in lateral decubitus (if there are no conditions for immediate delivery)
Evaluate state of consciousness and vital signs (talk to patient, evaluate breathing, radial pulse and blood pressure)

The first consideration should be given to assuring the continuous acquisition of a reliable FHR signal, so that the prolonged deceleration is confirmed, the duration is evaluated, and possible signs of recovery are documented. In addition, the occurrence of reduced variability within the deceleration is very suggestive of fetal hypoxia/acidosis. For all these reasons, continuous CTG should be preferred, if readily available. With external FHR monitoring, it is essential to assure that the fetus rather than the mother is being monitored, so the maternal pulse needs to be simultaneously evaluated to detect coincidences, and frequent repositioning of the Doppler sensor may be required. It is therefore useful to assign one person to assure a continuous good quality FHR signal and to evaluate the maternal pulse intermittently, for an adequate documentation of the situation.

The resolution of most situations of acute fetal hypoxia/acidosis requires a concerted action from a team of healthcare professionals, so therefore help should be promptly summoned, and the presence of at least two midwives and a senior obstetrician assured.

Although many prolonged decelerations revert spontaneously without any intervention, it is sometimes difficult to predict the situations in which this occurs. With the actions described in Table 2.2, all reversible causes of fetal hypoxia/acidosis are quickly identified, and some are immediately corrected. A quick search of the underlying cause is necessary to plan management, as some causes may be quickly reversible (excessive uterine activity, acute maternal hypotension and maternal supine position), while others are irreversible and pose extreme danger to the fetus (major placental abruption, uterine rupture, umbilical cord prolapse). When it is not possible to identify a cause, management should be based mainly on the duration of the deceleration and the evaluation of variability (see below).

Even when there is no apparent tachysystole (see definition above), oxytocin perfusion should be stopped or prostaglandins removed (when possible) to reduce contraction frequency and help recover fetal oxygenation. A vaginal examination is essential to diagnose umbilical cord prolapse and to evaluate the conditions for instrumental vaginal delivery. Even if the patient is in the active second stage of labour, asking the woman to stop pushing and temporary adopting the lateral decubitus position may relieve aorto-caval compression and reduce uterine contraction frequency. In some situations of occult cord compression, it may be the left or the right lateral decubitus that causes the best effect, or alternatively the half-sitting or upright positions.

Table 2.3 Drugs used for acute tocolysis

Salbutamol 125 μg at 25 μ/min IV. One 1 ml vial (0.5 mg/ml) in 100 ml of crystalloid solution, in intravenous perfusion at 300 ml/h for 5 min
Terbutaline 0.25 mg by subcutaneous injection
Atosiban 6.75 mg IV. One 0.9 ml vial (7.5 mg/ml) given by intravenous bolus during 1 min

2.4.2   Uterine Hypercontractility

Uterine hypercontractility occurring when an oxytocin perfusion is in place usually starts to revert 1–3 min after it is stopped, as the half-life of the drug is around 3–6 min. If a faster effect is required or when hypercontractility is spontaneous in nature or prostaglandins cannot be removed, acute tocolysis should be considered (Table 2.3).

Salbutamol and **terbutaline** should not be used in women with coronary artery disease, history of cardiac arrhythmias, high blood pressure, hyperthyroidism or low potassium levels. Their main side effects are tachycardia, tremor and nervousness.

Atosiban has no formal contraindications, but occasional side effects such as headaches, dizziness, vomiting, tachycardia, hypotension and fever have been reported.

Prolonged decelerations should start to revert 1–2 min after acute tocolysis has begun, and waiting for this to occur is the first option when hypercontractility is strongly suspected. During the second stage of labour, instrumental vaginal delivery may be an alternative if there are conditions for a quick and safe procedure. Otherwise, asking the parturient to stop pushing and waiting for reversal of the deceleration is preferable to guarantee the return of adequate fetal oxygenation. It should not be forgotten that uterine hypercontractility may also occur in the initial phases of placental abruption, so the maximum time limits for reversal of a prolonged deceleration should be taken into account (see below).

2.4.3   Sudden Maternal Hypotension

Sudden maternal hypotension secondary to epidural or spinal analgesia is usually quickly reversed by starting or increasing **crystalloid perfusion** and when this is not enough administering **ephedrine** 3–6 mg in intravenous bolus over 5 min. The bolus can be repeated after 5–10 min, until a maximum dose of 10 mg is reached. The drug is contraindicated in patients with cardiac disease, hypertension, hyperthyroidism, phaeocromocytoma and closed angle glaucoma and those who have taken monoamine oxidase inhibitors in the previous 14 days. The following side effects have been reported: paleness, fever, dry mucosae, shortness of breath, chest pain, tachycardia, anxiety, nausea and vomiting, headache, insomnia and mood changes. It can also cause transitory fetal tachycardia.

Administration of colloids is not recommended in these situations, as they can have a negative impact on coagulation, and rare cases of anaphylaxis and acute renal insufficiency have been reported.

Reversal of the FHR deceleration should start very soon after blood pressure begins to normalise, and waiting for this to occur should be the first option unless a very fast and safe instrumental vaginal delivery can be guaranteed. It should not be forgotten that hypotension may also be caused by maternal haemorrhage, as occurs with major placental abruption or uterine rupture, so the maximum time limits for reversal need to be taken into account (see below).

2.4.4   Major Placental Abruption

When major placental abruption is strongly suspected, rapid delivery is required to guarantee the safety of both mother and fetus. Instrumental vaginal delivery may occasionally be possible if there are very favourable conditions, but the majority of cases will present before the active second stage of labour and require emergent caesarean section. The anaesthesiologist and neonatologist should therefore be rapidly summoned and the operating theatre prepared.

Profuse retroplacental haemorrhage and disseminated intravascular coagulation may occur in this situation, so continuous monitoring of maternal vital signs, oxygen saturation and ECG should be started, a vein catheterised with a large bore needle, blood drawn for haemoglobin, coagulation studies and cross-matching and a crystalloid infusion initiated. Blood results need to be monitored regularly in order to identify the first signs of disseminated intravascular coagulation and to anticipate postpartum haemorrhage (see Chap. 7).

If fetal death has occurred and the mother is haemodynamically stable, it is preferable to induce or accelerate labour. In these situations, caesarean section is reserved for absent labour progress, profuse bleeding and/or maternal haemodynamic instability.

As the diagnosis of major placental abruption is not always firmly established before delivery, the maximum time limits for reversal of a prolonged deceleration need to be taken into account (see below).

2.4.5   Uterine Rupture

Uterine rupture requires prompt delivery followed by surgical repair, in order to guarantee the safety of both mother and fetus. Similarly to major placental abruption, when the diagnosis is strongly suspected, continuous monitoring of maternal vital signs, oxygen saturation and ECG should be put in place, a peripheral vein catheterised with a large bore needle, blood drawn for haemoglobin, coagulation studies and cross-matching and a crystalloid infusion initiated. The anaesthesiologist and neonatologist should to be rapidly summoned and the operating theatre prepared.

After confirmation of uterine rupture at laparotomy and delivery of the fetus, surgical correction with a double-layer suture may be technically possible. Several case reports of successful subsequent pregnancies have been described after this type of surgery, usually delivered by elective caesarean section at term. If suturing

of the lesion is judged to be impossible, or when it is anticipated to be a lengthy procedure in the context of an unstable patient, peripartum hysterectomy remains the only alternative. Some centres report subtotal hysterectomy to be safer than total hysterectomy in this context, with lower rates of ureteral complications and maternal mortality, but surgical experience should be the determining factor in this choice.

As the diagnosis of uterine rupture cannot always be safely established during labour, particularly when the woman is under epidural or spinal analgesia, the maximum time limits for reversal of a prolonged deceleration need to be taken into account (see below).

2.4.6   Umbilical Cord Prolapse

When umbilical cord prolapse occurs in the absence of continuous CTG monitoring, cord pulsatility should be evaluated, and if doubt remains as to the occurrence of blood flow, heart movements should be quickly confirmed on ultrasound. A few cases of fetal survival have been reported when rapid action is taken in spite of an apparently non-pulsatile cord. An exteriorised umbilical loop requires as little manipulation as possible to avoid vascular spasm.

If the fetus is alive and the gestational age is viable, immediate measures should be taken to reduce cord compression and quickly deliver the fetus, usually by caesarean section. Cord prolapse is very rare during the second stage of labour with the head fully engaged, so instrumental vaginal delivery is usually not an option. The patient should be rapidly placed in a **head-down position** and the **presenting part raised** by a hand inside the vagina, measures that are maintained until delivery. An anaesthesiologist and a neonatologist need to be rapidly summoned, the operating theatre prepared and the patient transported there as quickly as possible.

When immediate caesarean delivery is not possible (i.e. when prolapse occurs in a health facility that does not have an operating theatre), acute tocolysis should be started (Table 2.3), the bladder catheterised and 500–750 ml of saline solution instilled via the catheter in order to maintain the presenting part elevated. Continuous monitoring by CTG or handheld Doppler is then maintained during patient transport, until a caesarean section is performed.

Currently, there is insufficient evidence on the safety of alternative treatments for umbilical cord prolapse, such as digital reduction of the prolapsed cord followed by expectant management, although there are a few reported cases of success in the scientific literature.

2.4.7   Maximum Time Limits for Reversal of a Prolonged Deceleration

Frequently, the underlying cause of a prolonged deceleration is not clear, and no safe judgement can be made as to its reversible or irreversible nature, nor of the probability of recurrence when contractility is resumed. Abundant vaginal bleeding

is highly suggestive of an irreversible cause (placental abruption, uterine rupture, ruptured vasa praevia) and should prompt immediate delivery. The appearance of a sinusoidal pattern is also very suggestive of fetal haemorrhage (fetal-maternal haemorrhage or ruptured vasa praevia) and should also motivate rapid delivery.

Whatever the cause of fetal hypoxia/acidosis, maximum time limits for a prolonged deceleration need to be established in order to guarantee the absence of permanent injury to the fetus.

Particular care is recommended when prolonged decelerations exceed **5 min** and there is no tendency to recover, especially when there is reduced variability within the deceleration. The healthcare team should start preparing all the requirements needed for a rapid caesarean section or instrumental vaginal delivery. The latter should only be considered if a quick and safe procedure can be guaranteed; otherwise the operating theatre team should be prepared for an emergent caesarean section. The anaesthesiologist and neonatologist should be rapidly summoned. The final decision to intervene depends on local conditions, such as distance to the operating theatre and the team's proficiency with the surgery, as well as on additional clinical information (normal or growth-retarded fetus, previous CTG changes, appearance of vaginal bleeding or other symptoms suggesting an irreversible cause). With an adequately grown and previously well-oxygenated fetus at term, this decision should rarely go beyond **7–8 min**. The whole team must be aware of the urgency of the situation, and the fetus needs to be **delivered within 3–4 min**, so that the total duration of the hypoxic insult does not exceed 12 min.

2.5   Clinical Records and Litigation Issues

The complications of intrapartum fetal hypoxia/acidosis constitute major causes of litigation in high- and medium-resource countries. Extreme care needs therefore to be taken with clinical records, so that it is clear which healthcare professionals were called, at what time they were called, when did they arrive, as well as who took which decisions, and who performed which procedures.

It is also very important to document umbilical artery blood pH and base deficit (or alternatively lactate) in all situations where there has been an obstetric intervention for suspected fetal hypoxia/acidosis and when Apgar scores are low. This information is important to increase the team's knowledge with management of these situations, and it also allows a clarification of many causes of neonatal encephalopathy, perinatal mortality and cerebral palsy. A large number of these complications are not due to intrapartum hypoxia/acidosis, and they can be ruled out with this information.

During caesarean section, it is important to look for causes of acute fetal hypoxia/acidosis, as some may only be revealed at this time. Major placental abruption may result in the formation of a retroplacental haematoma and/or a Couvelaire uterus, and histological examination of the placenta will confirm the diagnosis. Tight nuchal chords, true cord knots, low-lying cord loops and ruptured umbilical/placental vessels can usually be detected during caesarean section or at subsequent inspection of the placenta and membranes. Fetal haemorrhage requires the documentation

of newborn anaemia, and fetal-maternal haemorrhage can be detected by the Kleihauer-Betke test or flow cytometry.

A clear explanation of the situation to the parents, during and shortly after it has occurred, frequently removes main of the misunderstandings and uncertainties that lead to litigation. The obstetric team also needs to remain informed of the health status of the newborn, so that all information conveyed to the mother and family is coherent and adapted to this evolution.

MANAGEMENT OF A PROLONGED FHR DECELERATION

Initial measures	Continuous CTG, evaluate maternal pulse	☐
	Evaluate duration of deceleration and variability	☐
	Call for help (two midwives, senior obstetrician)	☐
	Stop oxytocin, prostaglandins. Evaluate contractility	☐
	Vaginal examination	☐
	Patient in lateral decubitus	☐
	Evaluate state of consciousness and vital signs	☐
1. Hypercontractility	Acute tocolysis if needed	☐
2. Maternal hypotension	Start/increase crystalloid perfusion	☐
	Ephedrin in intravenous bolus	☐

3. Suspected major placental abruption or uterine rupture

	Continuous BP, HR, O_2 sat, ECG	☐
	Venous catheterisation with large bore catheter	☐
	Blood samples (Hb, coagulation, cross-matching)	☐
	Crystalloid perfusion	☐
	Rapid cesarean section /laparotomy	☐

3. Umbilical cord prolapse	Head-down position	☐
	Manual elevation of the presenting part	☐
	Emergent cesarean section	☐

5. Acute maternal cardio-repiratory disorder (see chapter 7)

6. Specific mechanical complications of labour (see following chapters)

7. General rules (adequately-grown, previously well-oxygenated term fetuses)

Deceleration 5-6 minutes	Call anesthesiologist and neonatologist	☐
	Prepare operating theatre or instrumental delivery	☐
Deceleration 7-8 minutes	Rapid c-section or instrumental vaginal delivery	☐
Documentation	Umbilical blood sampling and analysis	☐
	Register all occurrences	☐

Suggested Reading

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