

# Intrauterine fetal resuscitation

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## Abstract

The delivery of oxygen to the fetus is dependent on adequate maternal blood oxygen concentration, uterine blood supply, placental transfer and fetal gas transport. Any disturbance in these factors, singly or in combination, can result in progressive fetal hypoxia and acidosis. The term fetal distress is non-specific but is usually applied to certain characteristic features on electronic fetal monitoring, confirmed if possible by fetal blood sampling. The aim of intrauterine fetal resuscitation (IUFR) measures is to increase oxygen delivery to the placenta and umbilical blood flow in an attempt to reverse fetal hypoxia and acidosis, so that labour may continue safely or to improve the fetal condition while arranging urgent delivery. IUFR measures include maternal re-positioning into left lateral or alternatives (i.e. right lateral or knee–elbow if necessary), rapid infusion of 1000 ml crystalloid (except in fluid-restricted or pre-eclamptic patients), decreasing uterine contractions by stopping oxytocics and administering acute tocolytics (terbutaline 250 µg SC or IV, glyceryl trinitrate 60–180 µg IV or sublingual spray, two puffs). A vasopressor (i.e. ephedrine) may be required in cases of maternal hypotension.

**Keywords** Aortocaval compression; fetal distress; resuscitation; tocolysis

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In the course of labour, the fetus is monitored by interpretation of its heart rate pattern. In the low-risk setting, this may be intermittent but continuous electronic fetal monitoring (EFM) is used in high-risk pregnancies. EFM is an indirect measure of fetal oxygenation and therefore acidaemia.

The term ‘fetal distress’ is often used to include a suspicious fetal heart trace without proven acidosis. The definition of fetal distress describes a state of ‘progressive fetal’ asphyxia that if not corrected or circumvented will result in decompensation of the physiological responses (primary redistribution of blood flow to preserve oxygenation of vital organs) and can cause permanent central nervous system and other damage or death.<sup>1</sup>

Continuous EFM in labour has a high sensitivity but low specificity. In the presence of a normal fetal heart rate (FHR) pattern there is a 99% predictive value for a non-acidotic fetus.

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## Learning objectives

After reading this article, you should be able to:

- discuss the physiology of normal fetal oxygen delivery and the factors that can disrupt it
- explain intrauterine fetal resuscitation (IUFR) measures and the basis on which they are derived
- describe clinical situations in which IUFR measures may be required

Conversely, in the presence of an abnormal FHR pattern the predictive value drops to 50% for fetal acidosis.<sup>2</sup> Utilization of tools such as fetal scalp blood sampling allow direct measurement of fetal acidosis and therefore decrease rates of operative deliveries for false-positive FHR patterns. However, the ability to perform fetal blood sampling is dependent on operator skill levels especially in early labour. It also requires rupture of membranes and sufficient dilation.

The role of intrapartum fetal resuscitation is to optimize fetal wellbeing while either expediting delivery or allowing labour to continue. Using continuous EFM allows recognition of abnormal heart rate patterns and possible asphyxia at an early stage allowing intervention to prevent injury to the fetal brain.

## Physiology of fetal oxygenation

The fetus is dependent on the placental unit and therefore the mother for exchange of oxygen and carbon dioxide. The rate of exchange relies on maternal, placental and fetal factors (Figure 1). Disruption of this transfer or reduction in its efficacy at any point can lead to acidosis in the fetus if compensatory mechanisms fail (Box 1).

## Maternal factors

Oxygen delivery to the placenta is determined by uterine blood flow and maternal oxygen content, where:

$$\text{Uterine blood flow} = \frac{\text{uterine perfusion pressure}}{\text{vascular resistance}}$$

$$\text{Maternal oxygen content} = [1.34 \times \text{Hb} \times \% \text{O}_2 \text{ saturation}] + 0.03 \times \text{PO}_2$$

*Uterine perfusion pressure* (uterine arterial – venous pressure) is affected in the third trimester when aortocaval compression by the gravid uterus becomes significant. This effect is most pronounced in the supine position and is compounded if systemic

## Fetal compensatory mechanisms in response to hypoxia

- Reduction in fetal movements
- Redistribution of cardiac output to vital organs
- Conversion to anaerobic metabolism
- Decrease in heart rate

## Box 1

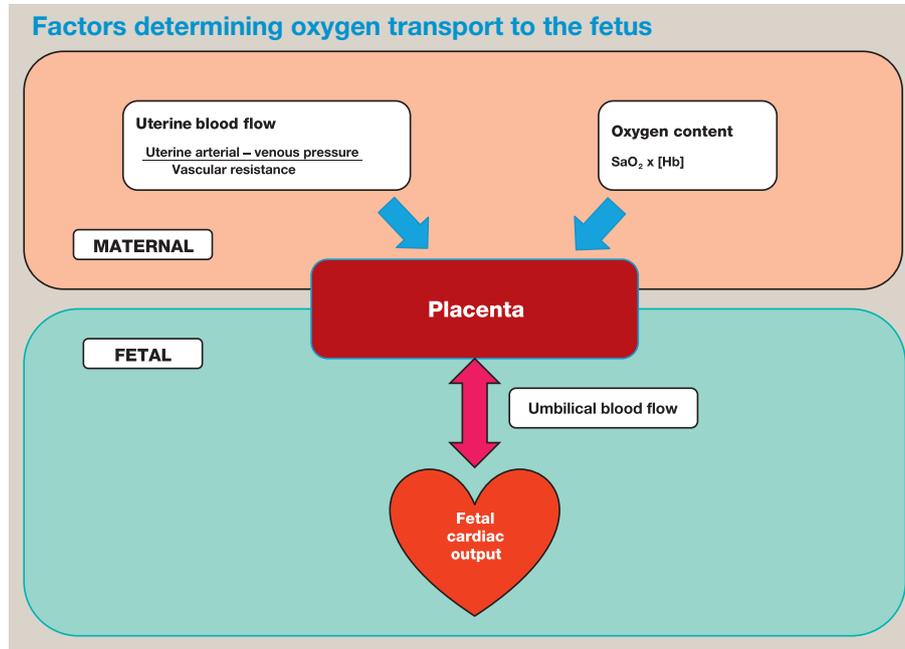


Figure 1

hypotension coexists. Alternating maternal position from supine to lateral relieves aortocaval compression. This compression effect is demonstrable up to 15° tilt within the inferior vena cava and up to 30° within the aorta. It is this change in blood flow distribution that underlines the rationale for the use of a 15° tilt at caesarean section.

*Uterine vascular resistance* is influenced by extrinsic and intrinsic vascular factors. During labour, uterine contractions cause a temporary interruption to maternal intervillous blood flow, causing transient fetal hypoxia. If the contractions are too frequent or last too long then the interruption to blood flow increases with potential for increasing fetal hypoxaemia and subsequent acidosis. The degree to which the fetus is affected is dependent on the pre-labour condition of the placenta and fetus.

Intrinsic vascular factors include vascular tone and blood viscosity. The uterine spiral arteries are generally believed to be maximally vasodilated at term. However, in pre-eclampsia uterine artery vasoconstriction may occur as a significant increase in intervillous blood flow has been shown in the presence of epidural analgesia.<sup>3</sup>

*Maternal oxygen saturation* only increases by 2–3% when oxygen is administered. This effect is achieved with relatively modest increases in  $\text{FiO}_2$  (0.3% or greater). Even though healthy women in labour have high  $\text{SpO}_2$  and oxygen content is close to maximum, it has been shown that maternal oxygen administration increases fetal oxygen levels.<sup>4</sup> This is believed to be secondary to maximizing maternal oxygen partial pressure.

### Placental factors

Oxygen is transferred to the fetus via passive diffusion. The affinity of fetal haemoglobin for oxygen and higher fetal haematocrit produces a steeper increase in fetal oxygen concentration

and  $\text{FSpO}_2$  during maternal  $\text{O}_2$  therapy. Similarly, only a small decrease in  $\text{FSpO}_2$  results when maternal oxygen is discontinued.<sup>5</sup> This effect is particularly demonstrable in fetuses that have a low  $\text{FSpO}_2$  compared to those with a normal  $\text{FSpO}_2$  prior to maternal oxygen administration. Although an increase in fetal oxygen levels may improve an abnormal fetal heart trace, it may not necessarily result in an improved fetal acid–base status.

There are theoretical concerns that hyperoxia may be detrimental to the hypoxic fetus via free radical production and reperfusion injury. The effects of maternal oxygenation for fetal distress during labour and the effects of prophylactic oxygen therapy during the second stage of labour on perinatal outcome have been the subject of a Cochrane review.<sup>6</sup> It was unable to evaluate the effectiveness of oxygen in fetal distress due to the absence of any trials. However studies of prophylactic oxygen in the second stage of normal labour found lower umbilical artery pH in the oxygen group and conflicting conclusions on the effect of duration of oxygen administration on umbilical artery pH. In clinical practice, maternal oxygen should be used for maternal hypoxia. Short-term oxygen therapy (15–30 minutes) for intrauterine resuscitation may be warranted after other interventions have been attempted.

### Fetal factors

#### Umbilical circulation

Compression of the cord can occur intermittently during labour if it is trapped by the presenting part or is wrapped around the fetal head or shoulder. This can result in variable decelerations on the FHR. Prolapse of the cord into the vagina is an obstetric emergency that requires immediate delivery. Relieving the compression on the cord either directly (manual elevation of the presenting part) or indirectly (filling the bladder with 500 ml

saline or adopting a knee–chest or modified Trendelenburg position) restores blood flow through the cord.<sup>7</sup>

### Fetal cardiac output

The fetus is designed by nature to function in a low oxygen environment. In its normal state the brain receives a higher percentage of fetal blood flow in utero than is proportional to its size and needs. This is dependent on fetal cardiac output, which is principally determined by FHR, as stroke volume is relatively fixed.

### Intrauterine fetal resuscitation measures

Most of the methods described have not been subjected to rigorous, randomized controlled trials in the acute situation of fetal distress. It would not be feasible to randomize women to receive no active measures to reverse fetal distress. Enough evidence exists to suggest they are beneficial to the fetus and there is minimal risk of harm when used with clinical common sense (Box 2).

### Tocolysis

In the presence of uterine hyperstimulation, or in cases of presumed fetal acidaemia without facility to immediately expedite delivery then fetal oxygenation can be improved by reduction in uterine contractions. Reduction (or cessation) of syntocinon will decrease uterine contractility. However, this is not an instant effect with only a 48% reduction in contractions at 45 minutes.<sup>8</sup> For rapid cessation of uterine activity the recommended tocolytic is terbutaline 250 µg SC or IV. Alternatively, for immediate action glyceryl trinitrate 60–180 µg IV or sublingual spray, two puffs initially, repeated after 1 minute until contractions stop, (maximum three doses). Potential adverse maternal effects of tocolysis are maternal hypotension and risk of haemorrhage from a relaxed uterus. When uterine hyperstimulation is associated with prostaglandin use for induction of labour then it should be removed immediately and vaginal irrigation performed.

### Maternal position

The left lateral position is advocated on the assumption that aortocaval compression exists. However in the case of umbilical cord compression, the left lateral position will not result in improvement and may cause a further deterioration in the FHR. Anaesthetists should be prepared to site regional anaesthesia in the lateral position if necessary. As most regional anaesthetics are routinely performed in the sitting position, it is advisable that

anaesthetists maintain their skills in the lateral position in case it is required in an emergency situation.

### Oxygen

As has previously been mentioned there is some debate around the benefit and potential harm of oxygen therapy. Oxygen should be administered to improve maternal saturation if required, or if other intrauterine fetal resuscitation (IUF) strategies have not improved the situation. To achieve maximum benefit of oxygen therapy it is important to use the method that delivers the highest FiO<sub>2</sub>. It is difficult to deliver FiO<sub>2</sub> of 1.0 without using an anaesthetic circuit and tight-fitting mask. A tight-fitting non-rebreathing facemask will deliver approximately 80–100% FiO<sub>2</sub> at 10 litres/minute.<sup>6</sup> The maximum practical flow rate above 10 litres/minute should be used.

### Intravenous fluids

In the hypovolaemic or hypotensive mother blood flow is diverted away from the uterus. Fluid boluses increase cardiac output, decrease blood viscosity and also have a temporary tocolytic effect, which in turn increases uterine blood flow and placental perfusion. A rapid infusion of crystalloid is recommended; 1000 ml has been shown to have a greater effect than 500 ml and the positive effect is maintained for over 30 minutes post-infusion.<sup>5</sup> Caution should be exercised in women who are pre-eclamptic, have cardiac disease or have had prolonged syntocinon infusions due to the increased risk of pulmonary oedema.

### Delivery and anaesthesia

The services of the anaesthetist are likely to be called upon urgently at times of fetal distress and so a knowledge of IUF measures is essential.

If caesarean delivery is required precipitously IUF will ‘buy time’ as the mother is transferred to the operating theatre and anaesthesia is induced. It is essential that FHR monitoring is recommenced in the theatre to assess if there has been any improvement or deterioration in the fetal condition that may alter mode of anaesthesia or delivery. The choice of anaesthesia will be determined by surgical urgency, maternal condition, patient preference and the anaesthetist. General anaesthesia is widely regarded as the quickest method. However, skilled practitioners may be able to rapidly induce spinal anaesthesia and a ‘rapid sequence spinal’ has been described although not widely accepted.<sup>9</sup>

The signs of fetal distress may be exacerbated by anaesthesia and elements of IUF may need to be employed. There should be a low threshold for giving vasopressors to maintain maternal cardiac output. Ephedrine crosses the placenta causing an increase in FHR and fetal cardiac output. ◆

### Intrauterine resuscitation measures (‘SPILT’)

Syntocinon: switch off  
 Position: full left lateral or knee elbow if cord compression  
 IV: infusion of 1 litre crystalloid  
 Low blood pressure — IV vasopressor  
 Tocolysis — terbutaline 250 µg SC or IV

### Box 2

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