Intrauterine fetal resuscitation

Hannah Kither
Suna Monaghan

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 whilst 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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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

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.[1]

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. Conversely, in the presence of an abnormal FHR pattern the predictive value drops to 50% for fetal acidosis.[2] 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 either whilst expediting delivery or to allow 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:

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

$$\text{Maternal oxygen content} = [1.34 \times \text{Hb} \times \%\ O_2\ \text{saturation}] + 0.03 \times PO_2$$

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 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

Hannah Kither *MB ChB is a Specialist Trainee in Obstetrics and Gynaecology in The North West Region, UK. Conflicts of interest: none declared.*

Suna Monaghan *MA MB BChir DCH DRCOG FRCA is a Consultant Obstetric Anaesthetist at Central Manchester University Hospitals NHS Foundation Trust, UK. Conflicts of interest: none declared.*

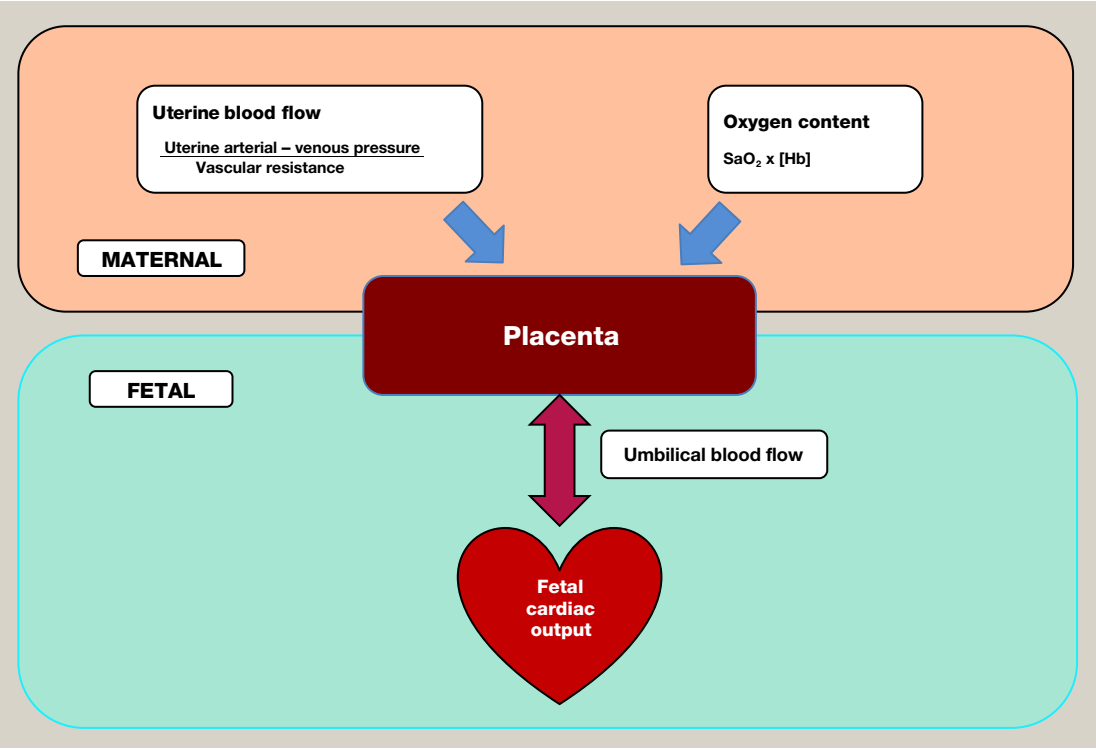

Figure 1 Factors determining oxygen transport to the fetus.

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

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.[3]

Maternal oxygen saturation only increases by 2–3% when oxygen is administered. This effect is achieved with relatively modest increases in FiO_2 (0.3% or greater). Even though healthy women in labour have high SpO_2 and oxygen content is close to maximum, it has been shown that maternal oxygen administration increases fetal oxygen levels.[4] 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 $FSpO_2$ during maternal O_2 therapy. Similarly, only a small decrease in $FSpO_2$ results when maternal oxygen is discontinued.[5] This effect is particularly demonstrable in fetuses that have a low $FSpO_2$ compared to those with a normal $FSpO_2$ 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.[6] 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