

Blood gas monitoring during RDS

The frequent monitoring of blood gases is clearly essential during the acute stages of RDS to assess the need for or effect of respiratory support. These aims are most reliably achieved through umbilical artery catheterisation or by indwelling peripheral arterial cannulae. Blood gas monitoring is also essential to minimize the risk of retinopathy of prematurity (ROP). There is little doubt that increased exposure to oxygen is linked to the development of ROP but the aetiology of ROP is multifactorial and other factors are also important. Unfortunately, there is no agreement regarding "safe" arterial oxygen contents or partial pressures in this respect.

Monitoring of oxygen by arterial sampling from an indwelling arterial catheter is the 'gold standard' measurement and continuous monitoring of blood gases including pH and PCO₂ by a catheter tip sensor is optimal [Meyers et al]. These monitors should only be used in conjunction with blood sampling, balancing the known hazards associated with prolonged intravascular monitoring against the improved monitoring provided. Non-invasive methods such as the use of transcutaneous oxygen and / or carbon dioxide tension monitors or pulse oximetry are useful trend detectors.

Pulse oximetry may be a useful guide to oxygenation particularly during neonatal transport, but appropriate levels of arterial oxygen saturation (SpO₂) have not yet been generally agreed, and will vary according to the oximeter used. Acceptable levels for SpO₂ of 85-93 % have been proposed, but because of variation in the relationship between SpO₂ and PaO₂ pulse oximetry cannot be recommended as the **only** form of monitoring arterial oxygen levels in the early phases of RDS. There have been recent suggestions that much lower limits of oxygen saturation may be acceptable [Tin et al] but as yet there is no reliable evidence on which to base this.

Hypocarbia is increasingly a problem in babies who have been treated with antenatal steroids, postnatal surfactant and ventilation from birth. Hypocarbia causes low cerebral blood flow, and a higher incidence of periventricular leukomalacia has been found in infants who had early hypocarbia [Fujimot et al].

It is difficult to set absolute limits for blood gases but the following blood gas values are suggested as a guide:

pH: Avoid arterial pH levels of less than 7.25. Cellular metabolic function is likely to be compromised at levels below this. Lower levels may sometimes be acceptable depending on the cause.

PaO₂: The recommended range is 6 - 10 kPa. The lower acceptable limit of PaO₂ in an infant with RDS may be lower than this (around 5.6 kPa, 40 mmHg) provided oxygen delivery to the tissues is adequate as judged by haemoglobin concentration, peripheral perfusion (and blood pressure), urine output and lactate concentration (but not base excess).

PaCO₂: More important than the PaCO₂ level is the pH and in general terms if this is maintained above 7.25 then the PaCO₂ is probably acceptable. The lower limit of PaCO₂ should be maintained above 5 kPa (37.5 mmHg). Similar values are acceptable from capillary and arterial blood gases.

Base Excess and Lactate

More important than the level of base excess is the pH and in general terms this should be maintained above 7.25. The lactate concentration is important in interpreting the cause of acidosis and should be < 2 mmol/l.

Summary and Level of Evidence

1. Use arterial blood gases to guide ventilation C
2. Oxygen saturation monitoring alone is insufficient to guide oxygen therapy in acute RDS C

References

Meyers PA. Worwa C. Trusty R. Mammel MC. Clinical validation of a continuous intravascular neonatal blood gas sensor introduced through an umbilical artery catheter. *Respiratory Care*. 2002;47(6):682-7

Tin W. Milligan DW. Pennefather P. Hey E. Pulse oximetry, severe retinopathy, and outcome at one year in babies of less than 28 weeks gestation. *Archives of Disease in Childhood* 2001;84(2):F106-10

Fujimot, S.;Togari, H.;Yamaguchi, N.;Mizhumi, F.;Suzuki S.;Sobajima, H. Hypocarbica and cystic periventricular leukomalacia in preterm infants. *Archives of Disease in Childhood* 1994;71:F111-F113