Clinical Guideline:

Saturation targeting in the neonate admitted to the Neonatal Unit

Authors: **Neonatal Saturation Targeting Working Group**

For use in: East of England Neonatal Units
Guidance specific to the care of inpatient neonatal patients
Guideline does not cover the provision for home oxygen, nor saturation targeting in this group

Used by: All neonatal/paediatric medical & nursing staff

Key Words: Saturation Targeting, Oxygen Therapy, Neonate, Retinopathy of Prematurity (ROP), Chronic Lung Disease (CLD), Prematurity

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Approved by:

Neonatal Clinical Oversight Group	
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Ratified by ODN Board:

Date of meeting	

Contents

Table of Saturation Targets and Limits

Gestation	Air/Oxygen	Target	Alarm Limits
Preterm <37 weeks Or Birth Weight <1.5kg	Oxygen	91 - 95%	90 - **96%***
	Air	91 - 95%	90 – 100%
Term Infant ≥37 weeks	Oxygen	≥95%	94 - 99%
	Air	≥95%	94 - 100%
Preterm infant with corrected gestation ≥37 weeks	Oxygen	≥93%	92 - 99%
	Air	≥93%	92 – 100%

All infants <34 weeks gestation, and all infants on supplementary oxygen or any invasive or non-invasive ventilatory support must be commenced on continuous pulse oximetry

Special Circumstances			
Circumstance	**Air/Oxygen**	**Target**	**Alarm Limits**
Risk of PPHN/ Established PPNH**	Air/Oxygen	>92%	90 - 98%
Suspected or Confirmed Cyanotic Heart Disease	See PaNDR Clinical Guideline: **"Management of a Duct Dependant Congenital Heart Disease"**		
	*Avoid Hyperoxia – Particularly in duct-dependent lesions**** supplemental O2 to achieve SatO2 in the 75-85% range *Liaise with Specialist Cardiac Centre for Advice*		

Special Instructions:

***Preterm infants with saturations >95% in oxygen is at significant risk of hyperoxia**

- PaO_2 will be significantly elevated
- Act with the same urgency as a significant desaturation

****Risk Factors for PPHN**

Term or Near-Term Infant:

Male, African or Asian maternal race

Maternal Factors- *raised BMI, Cigarette use, Ill-health through asthma / diabetes mellitus,* antenatal exposure to *Aspirin / Non-Steroidal Anti-Inflammatory Drug (NSAID) / Selective Serotonin Receptor Inhibitor (SSRI), cyclooxygenase inhibitors (COXi)*

C-section

Perinatal infection - Chorioamnionitis, GBS sepsis or congenital pneumonia

Meconium-stained amniotic fluid

Severe perinatal Hypoxic Ischaemic Encephalopathy (HIE)

Structural Lung Disease: *Pulmonary hypoplasia, congenital diaphragmatic hernia or congenital pulmonary malformation*

Others - Hypocalcaemia, acidosis, Polycythaemia

Syndrome - Trisomy 21

Preterm Infant:
Lower GA, weight at birth
Small for gestational age
Pulmonary haemorrhage
Sepsis, Oligohydramnios and anhydramnios
Prolonged duration of invasive respiratory support or Severe BPD

*****Limited evidence base for oxygen targeting in Cyanotic Heart Disease**

- Saturations >85% are unlikely to be achievable without significant hyperoxia (↑P_aO_2), due to the physiological effect of shunting/mixing
- Hyperoxia must be avoided due to risk of unintended ductal closure
- Saturations may be less than 75% in certain cyanotic cardiac lesions – liaison with cardiac specialists is imperative to guide targeting further

Assessment & Management of the Desaturating Neonate

The neonate, particularly at preterm, will commonly display variation in oxygen saturations. Fleeting and self-correcting desaturations are unlikely to be of clinical significance and efforts must be made to ***avoid the hyperoxaemic swings*** that are created by the addition of unnecessarily high fractions of inspired oxygen (FiO_2).

If a significant desaturation (i.e. prolonged or severe) does occur, manage the infant according to the ABC routine. Ensure adequate respiratory effort and air entry ***prior*** to increasing the FiO_2. Many desaturations will correct without intervention, and failing that, with simple stimulation or manual/ventilator breaths.

An increase in FiO_2 alone is likely to be ineffective, and potentially detrimental, in an infant demonstrating desaturation due to apnoea, reflux or any cause of hypoventilation.

Never walk away from an infant who is requiring an acute increase in FiO_2: ***they are at risk of both rebound hyperoxaemia and further deterioration***

The algorithm below (next page) may be used as an example of the appropriate management of the desaturating neonate. Several steps may be taken prior to increasing the FiO2. Please note it is imperative that where appropriate the UK NLS algorithm 2025 is followed while using the algorithm below as an adjunct to the appropriate management of a desaturating neonate.

The following should be clearly recorded on an infant's observation chart:

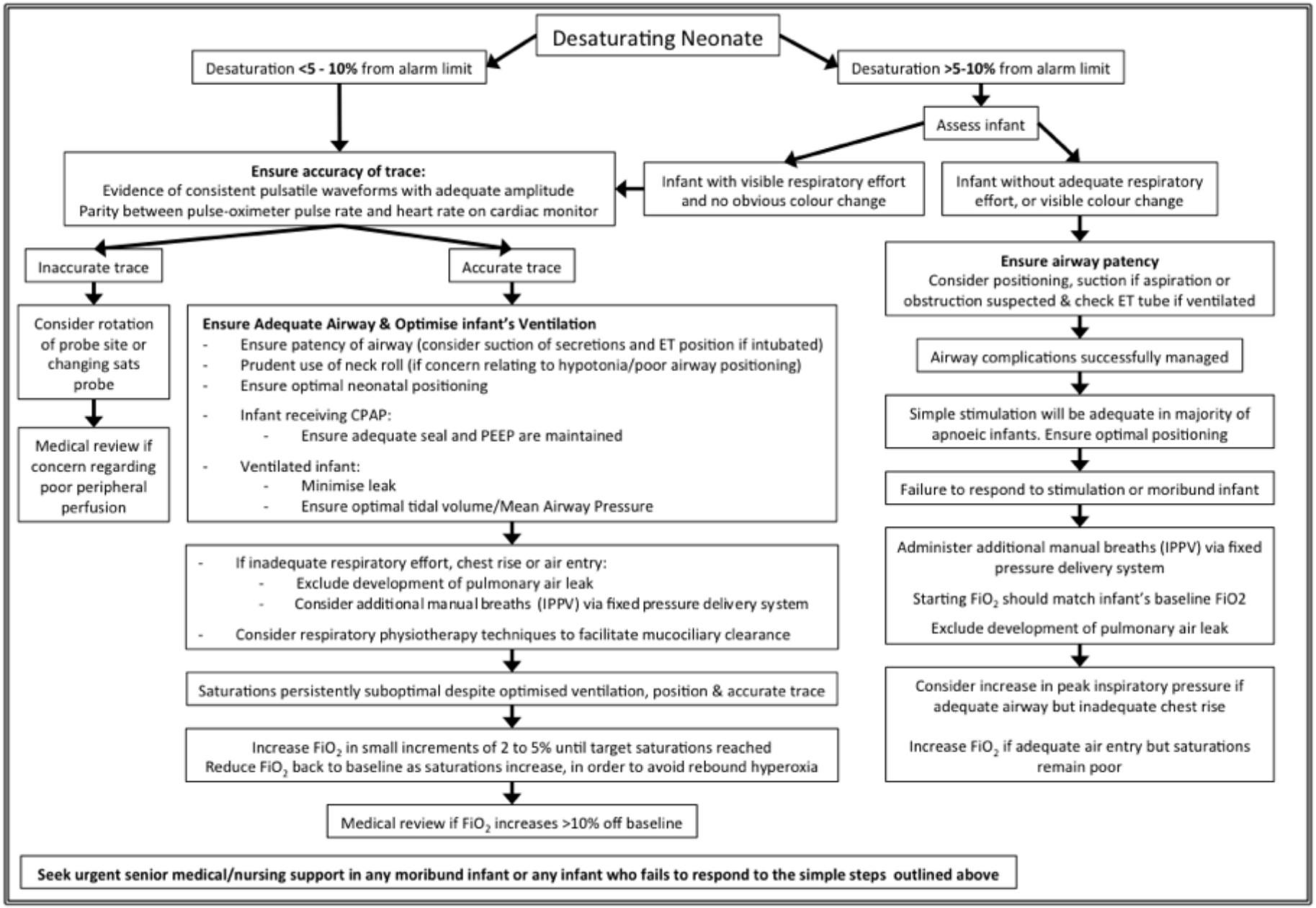
- Significant desaturations (not artefactual *and* prolonged or requiring intervention)
- Bradycardia
- Apnoeic spells (sustained absence of respiratory effort combined with significant desaturation or bradycardia, **or** absence of respiratory effort for >20 seconds without other clinical manifestation)

- Ensure that the lowest saturation, lowest heart rate and intervention required are cited

A preterm infant with an increase in frequency or severity of desaturations or bradycardias should receive prompt medical review.

Symptomatic apnoeic episodes are of particular concern and low threshold should be held for escalation of respiratory support and appropriate septic management.

Desaturation, bradycardia and apnoeic episodes in the term infant are almost always pathological and warrant medical investigation and intervention.



Special Circumstances

No guideline can cover all potential clinical scenarios faced on the neonatal unit.

The table of recommended saturation targets should cover most clinical scenarios. Individual saturation ranges may be prescribed as and when necessary – though the reasons for doing so should be clearly documented in the infant's medical notes.

The following sections aim to further explain particular special circumstances and, where available, reference/signpost to the appropriate specific regional/national guideline covering that scenario.

Saturation Targeting in the Infant with a Spontaneous Pneumothorax

Detailed instruction of the management of neonatal pneumothorax is beyond the scope of this guideline. These infants are at risk of deterioration; they require close monitoring, continuous pulse oximetry and management according to the ABC approach.

Supplemental oxygen should be provided to maintain the infant's oxygen saturations within the recommended target range for their gestation. **Evidence does not support the routine use of high FiO_2 to provide the 'nitrogen washout' technique – additional inspired oxygen, beyond that required to maintain saturations within the targeted range for gestation, does not affect pneumothorax resolution time but does expose the infant to the risk of hyperoxia [14].** In addition, the unnecessary administration of a high FiO_2 can mask deterioration in the neonate's condition, as any true increase in **oxygen requirement** is concealed until it surpasses the FiO_2 that is being administered.

Saturation Targeting in the Preterm Infant

Chronic Lung Disease (CLD) & Retinopathy of Prematurity (ROP)

Very premature and very low birth weight infants are at particular risk of chronic lung disease and retinopathy of prematurity. Although targeting saturations at a range 85-89% does reduce the incidence of ROP and, to some extent chronic lung disease, it leads to an unacceptable increase in mortality. As such, a saturation target range of 91-95% should be followed. Of note, although incidence of ROP increases when targeting saturations of 91 to 95%, when compared with 85 to 89%, no significant difference in visual impairment (including nystagmus, strabismus, the use of corrective lenses and unilateral/bilateral blindness) has been observed in the NeOProM studies reporting follow-up to 18-22 months.

The saturations target range of 91-95% should be maintained in extremely and very preterm infants, even after 32 weeks corrected gestation and ***regardless of the presence or absence of CLD, ROP or confirmed complete vascularisation of the retina.*** Targeting saturations >95% in this group substantially increases the duration of their inpatient oxygen requirement and significantly increases their risk of requiring home oxygen therapy without any significant difference in growth profile, neurodevelopmental status and mortality rate at 12 months.

Saturation Targeting at 37 Weeks Corrected Gestation and Prior to Discharge

In the very premature and extremely premature infant with corrected gestation at or beyond 37 weeks (or prior to this if being considered for discharge from the neonatal unit) saturations ≥93% should be targeted to aid transition to community care and paediatric standards. Infants discharged

home are susceptible to pulmonary hypertension, secondary to chronic hypoxia, if exposed to saturations <93% over a prolonged period.

The specific provisions and workup of the infant likely to require long-term home oxygen therapy is beyond the scope of this guideline. Further reference to the latest national guideline by the British Thorax Society (BTS), "**BTS Guidelines for Home Oxygen in Children**" & **Home Oxygen therapy for neonates, NHSGGC Paediatrics** is recommended.

Persistent Pulmonary Hypertension of the Newborn (PPHN)

Infants suffering from Persistent Pulmonary Hypertension of the Newborn (PPHN) are extremely unstable and critically unwell. They require senior management and are highly likely to require tertiary neonatal care. The management of PPHN is complex, multi-factorial and beyond the scope of this guideline, other than comments relevant to oxygen saturation targeting.

The infant at risk of PPHN (see box on page 3 (Delaney & Cornfield, 2012)) should have oxygen saturations maintained between 90–97% for term and late preterm infants following the initial stabilization where the administered oxygen may have been titrated starting at 100% for a short period to aid the diagnosis and supportive care. The infant with established PPHN will benefit from the vasodilating properties of liberal oxygen therapy, and as a result their oxygen saturations should be targeted as high as is physiologically possible within the desired range.

Comparison of pre and post-ductal saturations will be highly informative. Measurement of the PaO_2 will also allow for calculation of the **Oxygen Index (OI)** (Noting that differences in pre and post-ductal PaO_2 may occur; the OI is classically, though not necessarily universally, calculated using the post-ductal PaO_2). The OI will help inform decisions relating to both nitric oxide and Extra-Corporeal Membrane Oxygenation (ECMO) therapy.

In PPHN, a significant difference in pre and post ductal PaO_2 (>2.5 kPa) or O_2 saturations (5-10%) may occur.

Oxygenation Index (**OI**) = [MAP x FiO_2] / [PaO_2 x 7.5]

Given:
MAP: Mean Airway Pressure (**cmH_2O**)
FiO_2: 'Fraction' of Inspired Oxygen as percentage (**%**)
PaO_2: Partial Pressure of Oxygen in Arterial Blood gas (**kPa**)

Suspected Congenital Heart Disease (CHD)

Infants with congenital and/or cyanotic heart disease require specific individualised saturation targets according to the precise anatomical defect, whether the defect is duct dependant and whether any palliative or corrective procedures have been undertaken. Liaison with cardiac specialist centres is recommended.

In the emergent presentation of an infant with a previously unsuspected congenital heart lesion, please refer to the Paediatric and Neonatal Decision Support and Retrieval service, PaNDR Clinical Guideline "**Duct Dependant CHD PaNDR**".

This is available on the PaNDR website – follow the links to **'FOR PROFESSIONALS'** and **'Clinical Guidelines'** and select guideline **'Cardiovascular' under which** you should find "**Duct Dependant CHD PaNDR**".

Direct Hyperlink: Neonatal Guidelines | PaNDR

Prompt liaison and referral to a cardiac specialist centre and the PaNDR team should then take place.

Saturation targeting in the delivery suite

Term & Preterm Infants

This guideline advocates the use of UK resuscitation council NLS recommendations (Resuscitation Guidelines 2025) for the resuscitation of term and preterm infants at delivery.

Term infants, and infants above 32 weeks gestation, should be first resuscitated in **air** – subsequent FiO_2 delivery should be adjusted according to clinical response and pre-ductal saturations (see table below). The preterm infant of less than 32 weeks gestation should be resuscitated in a starting FiO_2 of 30%, again with adjustment according to clinical response and pre-ductal saturations (bradycardia, despite adequate ventilation, and pre-ductal saturations below the 'acceptable' limits, detailed in the table below, are both indications for upwards titration of delivered FiO_2).

When required at delivery, saturation probes should be placed on the right wrist (**Pre-ductal**). A pulse oximeter signal may be detected more rapidly if the probe is placed on the infant's wrist ***first*** and the machine is turned on ***before*** connecting the probe to the machine.

It is recommended that pulse oximeters used at delivery are set to a rapid saturation averaging time: i.e. around 2 seconds.

2025	Starting Oxygen Saturation
< 32 weeks	= 30 % or >30 %
32 weeks and above	Air

Time from Birth	Acceptable Pre-Ductal Saturation
3 minutes	70 – 75 %
5 minutes	80 - 85%
10 minutes	85 -95 %

*Tabulation of Resus Council UK (*Resuscitation Guidelines 2025*) recommendations for acceptable pulse oximeter saturations at delivery, according to the gestational age*[21].

Background & Theory of Practice

Oxygen Targeting in the Neonatal Period

Pulse oximetry may be used in the neonatal period to help mitigate and negotiate the risks posed by both relative hypoxaemia and hyperoxaemia.

Term infants

Limited evidence exists regarding pulse oximeter targeting in term Infants; with no randomised control trials comparing saturation ranges. Whilst preterm babies > 32 weeks or >1.5kg are not at risk of ROP, hyperoxaemia may still have deleterious effects on cerebral perfusion and may exacerbate oxidative stress in the presence of a hypoxic-ischaemic insult. Guidance relating to pulse oximeter saturation targeting in term babies must therefore be based on observed normal ranges, aimed at avoiding the precipitation of PPHN and aimed to minimise the risk of missing a potential congenital cardiac lesion.

In this light, pulse oximeter saturation targeting, and thus decisions to start oxygen therapy, must also be informed by clinical assessment of individual infants.

Observational studies reveal that healthy term infants' pulse oximeter saturations range from 89-100% (Median 98.3%) in the first 24 hours of age, and 92 to 100% (Median 97.6%) in the first week of age. In most infants, acclimatisation to pulse oximeter saturations >95% (pre & post-ductal) occurs rapidly, taking on average 12 to 14 minutes post-delivery. (O'Brien et al, 2000; Poets et al, 1996; Toth et al, 2002)

Preterm infants

A significant body of evidence now exists regarding the study of pulse oximeter saturation targeting in preterm infants and well-designed prospective meta-analysis regarding saturation targeting in extreme preterm infants is now available. Hyperoxaemia in the preterm infant is associated with the development of both Chronic Lung Disease (CLD) & Retinopathy of Prematurity (ROP). Hypoxaemia is associated with increased mortality and the development of Necrotising Enterocolitis (NEC).

The **BOOST** and **STOP-ROP** trials provide evidence that targeting saturations >95% is deleterious to preterm infant health.

The original **BOOST trial** (Benefits of Oxygen Saturation Targeting) was a multi-centre double-blinded RCT involving a total of 358 participating infants born at <30 weeks gestation (mean gestation was 26.6). The trial compared target saturations of 91 to 94% vs 95 to 98%; starting at corrected gestations >32 weeks and continued until oxygen therapy was no longer required. Between the two groups, no significant difference in growth profile, neurodisability at 12 months, grade of ROP, nor progression to surgical treatment of ROP, was observed. Despite observed absence of benefit, infants in the higher saturations group demonstrated a greater incidence of CLD; they required double the duration of inpatient oxygen therapy (40 vs 18 days), had a significantly increased frequency of home-oxygen therapy requirement (17 vs 30%, RR 1.78, CI 1.20 to 2.64, P=0.004) and a statistically non-significant increase in deaths relating to pulmonary causes.

The **STOP-ROP** trial (Supplemental Therapeutic Oxygen for Prethreshold Retinopathy of Prematurity) recruited 649 infants born at <31 weeks gestation, or <1.5kg birth weight, with pre-threshold ROP. Randomisation to two target saturation groups - 89-94% Vs 96-99%, took place at 6 – 10 weeks of age and continued until either regression of ROP, or progression of ROP to threshold had occurred. The authors reported an increase in exacerbations of CLD and pneumonia, length of stay and diuretic therapy in the higher saturations group. Effect on ROP progression was minimal and no difference in growth and developmental milestones was observed.

The **NeOProM** collaboration provides evidence that targeting saturations <91% in extremely preterm infants contributes to an increase in mortality and the development of NEC.

The **NeOProM** collaboration (formed of 5 prospectively planned RCTs with similar methodology: **COT**, **BOOSTII** [UK, NZ, AU] & **SUPPORT**) compared death, neurodisability and rates of ROP, plus multiple secondary outcomes, in extreme preterm infants in two randomised groups: 91-95% vs 85-89%. Relative risk of death was increased in the lower saturations groups (RR 1.18-1.41) and rates of NEC increased in the lower saturations group by 25% (RR 1.25). A substantial 26% reduction in severe ROP was observed in the lower saturations group (RR 0.74), though this effect is somewhat negated, given that no significant increase in bilateral blindness or visual impairment has been observed in the 3 trials published so far with follow-up to 18 to 24 months (BOOST II NZ, COT & SUPPORT). The occurrence of physiologic BPD tended towards the higher saturation group, but this effect was small (37.6% vs 39.7%, RR 0.86, CI 0.77-0.96). No substantial differences were observed between the two groups in the occurrence of PDA & 'brain injury'.

Benefits of Saturation Monitoring

The implementation of pulse oximetry has considerably reduced the need for repeat arterial blood sampling to Monitor PaO_2.

Pulse oximetry:

- Is non-invasive
- Enables continuous monitoring
  - Thus, facilitating the detection of acute deteriorations in an infant's oxygenation status -
- Is a reflection of a greater proportion of an infant's total oxygen content than PaO_2 interpreted in isolation

Limitations and Pitfalls of Saturation Monitoring

Pulse oximetry is only as useful as the accuracy of the reported saturations. In fact, acting on an inaccurate trace will lead to either unnecessary therapeutic action or inaction and subsequent detriment to the infant.

Pulse Oximetry is liable to inaccuracy in the following instances:

- Movement artefact
- Cool/poorly perfused peripheries
- Ambient light (Causing interference with the probe's spectrophotometer)
- Infants with high total carboxyhaemoglobin levels (CarboxyHb has a similar absorbance to oxyhaemoglobin and may falsely elevate pulse oximeter readings)

Furthermore, it must be considered that pulse oximeter saturations only represent a ***percentage*** of the haemoglobin-bound oxygen content of blood. Pulse oximeter saturations ***do not reflect*** *an* ***absolute value*** for the haemoglobin-bound oxygen content of an infant's blood.

> ***i.e.*** an *anaemic* infant's total oxygen content of blood, or total oxygen-carrying capacity, will be much less than a *polycythaemic* infant, *even if their pulse oximeter saturations are identical*
>
> An infant's *total oxygen content* is also dependent on their PaO_2 and the absolute value of circulating haemoglobin

Risk of Burns

There are multiple reported cases of significant burns occurring from pulse oximeter probe sites.

Burns are more likely to occur:

a) In premature infants with thin skin
b) If pulse oximeters are malfunctioning and not regularly serviced
c) **I**f probes are not placed according to manufacturer instructions *(often probes are supplied with protective plastic coverings which should be stuck over both ends of the electronic probe)*

The risk of burns may be mitigated, but not eliminated, by regular skin integrity checks and probe site rotation.

Aerobic Respiration & Oxygen Delivery

Oxygen is a vital element for **aerobic respiration** to occur. Through aerobic respiration, each cell in the human body is able to produce 'chemical' energy, known as **ATP** (**A**denosine **t**ri**p**hosphate). ATP is required to fuel almost every metabolic process in the body. At the final stage of aerobic respiration, oxygen acts as the final electron acceptor in a process known as the **Electron Transport Chain**, which takes place in the mitochondria. Without oxygen aerobic respiration cannot take place and consequently cells have to switch to inefficient **anaerobic respiration**; this produces far less energy and leads to the accumulation of **lactic acid**.

It is therefore vital that the body maintains **oxygen delivery** to all tissues via the blood supply. Oxygen delivery is the product of the blood flow through the body each minute (**Cardiac Output**) and the **oxygen content** of that blood.

The **oxygen content** of blood is the sum of both the amount of oxygen bound to **haemoglobin** (**Hb**) in the blood, plus the amount of unbound oxygen dissolved within the blood (the **partial pressure** of oxygen).

Oxygen is delivered to the body's tissues via arterial blood. Following consumption of oxygen within the capillary bed and tissues, deoxygenated blood returns to the heart via the veins, where it is subsequently pumped to the lungs to be re-oxygenated. This natural process may be disturbed in diseased states or following therapeutic interventions – such as the interruption of gas diffusion in pneumonia or the administration of oxygen at supra-physiological values (>21%). It therefore follows that the oxygen content of blood may fall out of normal physiological parameters, such as in the following definitions:

Hypoxaemia:	Low total oxygen content within the blood
***Hypo*xia:**	The resultant *oxygen deficiency* within the body's tissues and organ systems
***Hyper*oxaemia:**	High total oxygen content within the blood – specifically expressed as a high **PaO_2** (Partial Pressure of Oxygen in **arterial** blood)
***Hyper*oxia:**	The resultant oxygen excess within the body's tissues and organ systems

Haemoglobin Oxygen Saturations

Each haemoglobin molecule is able to bind to a maximum of four oxygen molecules. It is therefore possible to express the oxygen saturation of the blood as a percentage, based on how many oxygen molecules have associated with each haemoglobin molecule.

Pulse oximetry provides a non-invasive estimate of the **oxygen saturation of haemoglobin** in **arterial** blood. The pulse oximeter probe uses **spectrophotometry** (the relative absorbance of red and infrared light of each haemoglobin molecule) to distinguish between haemoglobin with a high number of associated oxygen molecules (**oxyhaemoglobin**) and haemoglobin with a low number of associated oxygen molecules (**deoxyhaemoglobin**). The probe is able to differentiate arterial blood due to the pulsatile nature of the arteries. The pulse oximeter will then provide a percentage saturation of oxygen, which has been averaged over a predefined period of time (Usually around 15

seconds – though pulse oximeters at deliveries should be set to a much shorter averaging time of around 2 seconds).

Functional Versus Fractional Pulse Oximetry

Traditional pulse oximeters measured 'fractional' oxygen saturations, i.e. the percentage of oxygenated haemoglobin when compared with total haemoglobin content (including oxyhaemoglobin, deoxyhaemoglobin, carboxyhaemoglobin & methaemoglobin). Newer pulse oximeters, and those used in the most recent clinical trials, report 'functional' oxygen saturations, i.e. the percentage oxygenation of haemoglobin which may ***functionally*** transport oxygen within the bloodstream (Oxyhaemoglobin vs deoxyhaemoglobin).

Functional pulse oximeters report oxygen saturations approximately 1.5% higher than fractional pulse oximeters. ***The target oxygen saturation ranges recommended within this guideline are in reference to functional pulse oximeters***.

The Relationship Between PaO_2 and SaO_2

As outlined above, the PaO_2 is the measure of oxygen dissolved within the arterial blood stream (Partial Pressure of oxygen). The percentage of oxygenated haemoglobin within arterial blood (SaO_2) is proportional to PaO_2 through a dynamic relationship that changes according to the *sigmoid shaped* ***oxygen-haemoglobin dissociation curve***. A schematic of this curve is displayed below.

Haemoglobin's *affinity* for oxygen decreases as the PaO_2 decreases. This facilitates the dissociation and supply of oxygen from haemoglobin, as oxygen is consumed within the body's tissues. Conversely, once the red cells (erythrocytes) carrying the haemoglobin molecules within the blood are returned to the alveoli within the lungs, the partial pressure of oxygen is higher and thus oxygen molecules may re-associate and bind with the haemoglobin molecules.

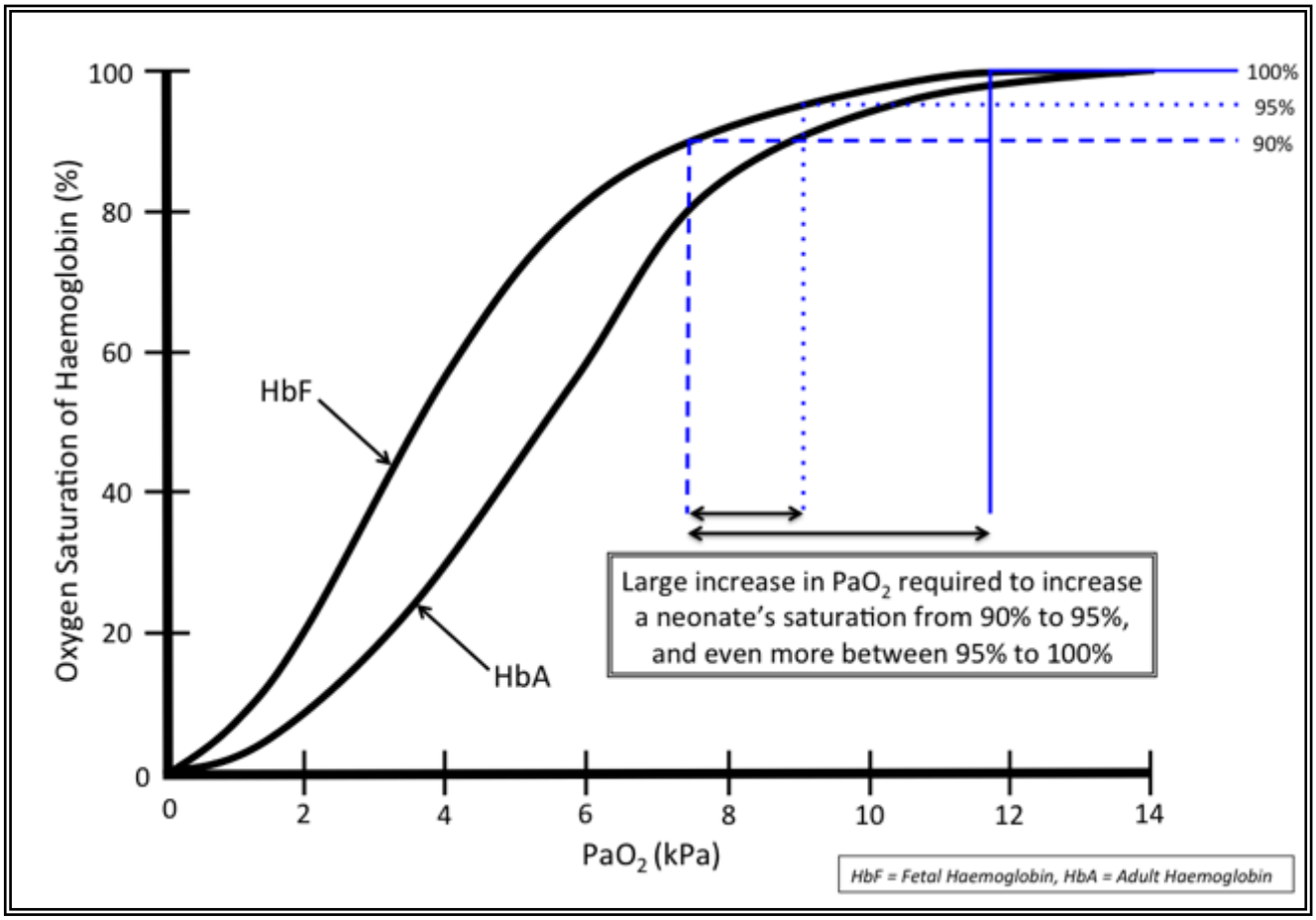
Other factors also influence how readily oxygen will dissociate from haemoglobin; for instance, an acidotic environment will lead to greater oxygen dissociation. In addition, the majority of a neonate's haemoglobin fraction will be ***Fetal haemoglobin*** (HbF). HbF is gradually replaced by adult type haemoglobin (HbA) over the infant's first 6 months. HbF displays a much higher affinity for oxygen than HbA; Oxygen delivery in the newborn infant may be maintained at lower PaO_2's, despite this, due to the higher total haemoglobin content.

The oxygen dissociation profile of HbF is also displayed on the schematic curve below. As a result of the flattening of the top of the HbF curve, it may be appreciated that as saturations increase beyond 90%, large increases in PaO_2 are required to increase the oxygen saturations by only small amounts over the final 10%. Equally, due to the sharp gradient before that, small decreases in oxygen saturation less than 80% to 90% correspond to large decreases in PaO_2 and thus dramatic falls in total oxygen content and hypoxaemia.

Parental Information and Communication

In keeping with the BAPM and NICE principles of family centred neonatal care, parents or carers should be informed, where clinically appropriate, about the use of oxygen saturation monitoring. This should include an explanation of the purpose of monitoring, the target saturation ranges, and how saturation values are interpreted alongside other clinical observations to inform clinical

decision making and escalation of care. Information should be provided in a clear, timely, and sensitive manner, taking into account parental understanding and the clinical circumstances, and should be documented in the clinical record where relevant.

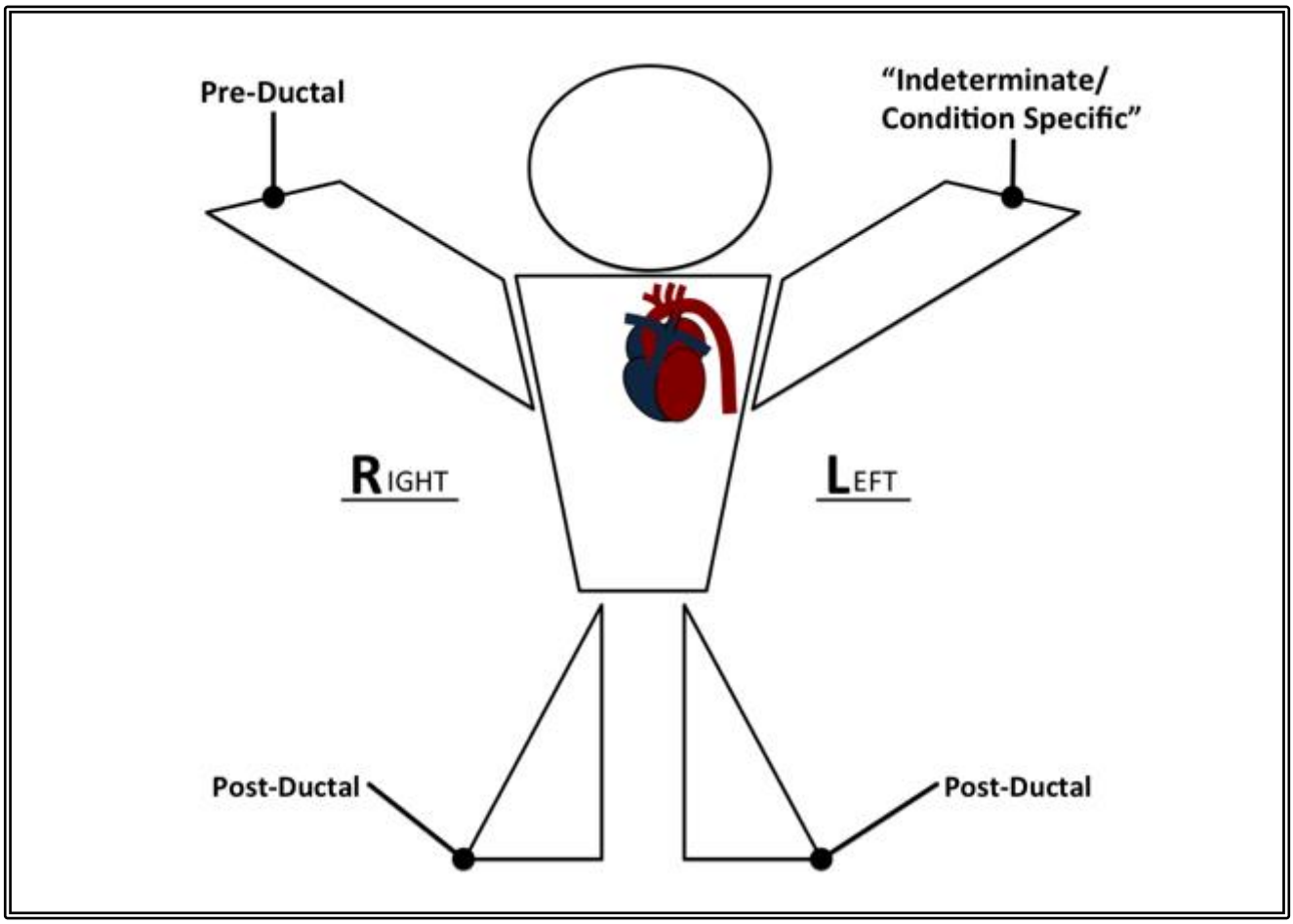


Schematic diagram to demonstrate, and compare, sigmoid dissociation of oxygen from HbF & HbA in differing partial pressures of oxygen.

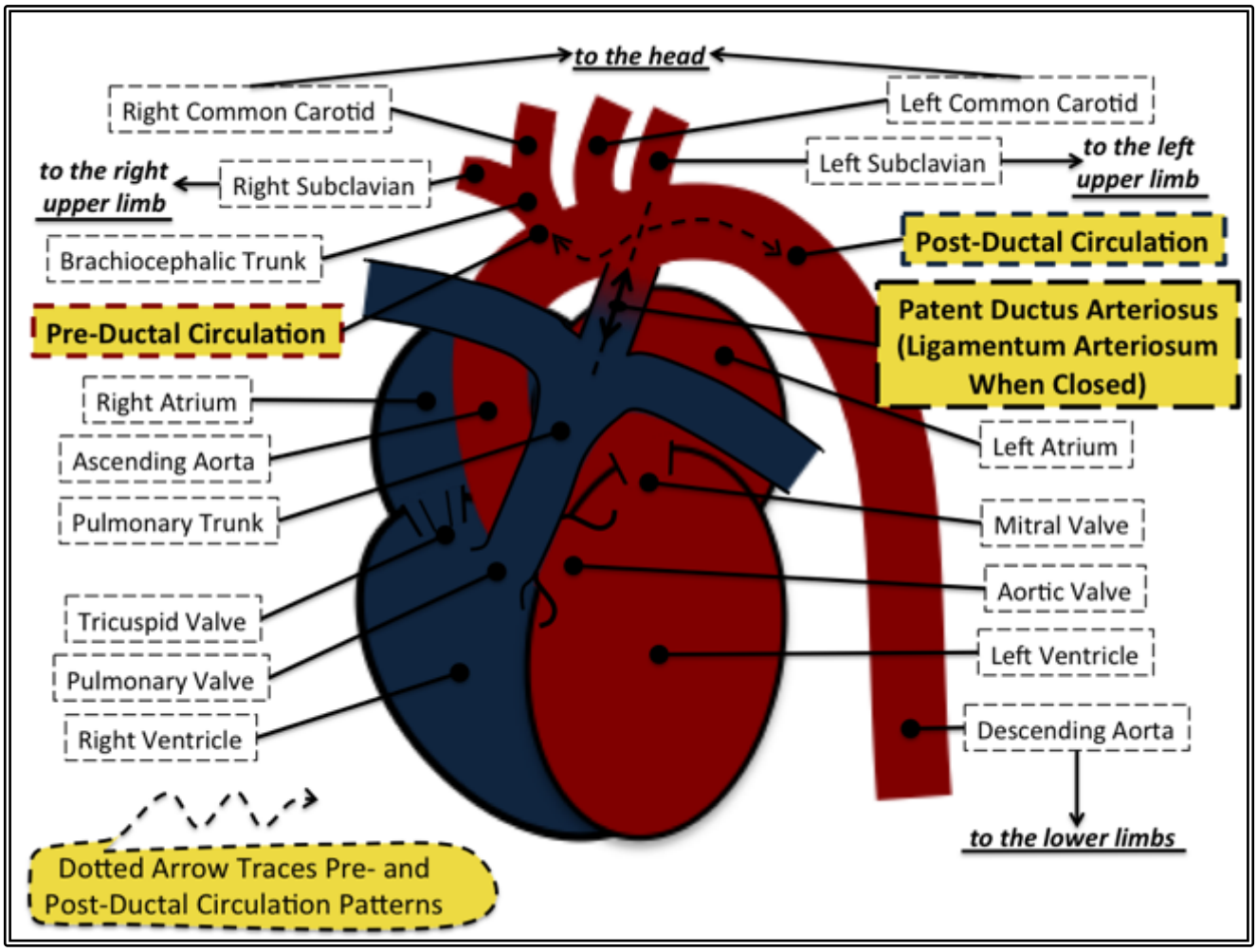
Placement of Pulse Oximeter Probes

The pulse oximeter probe may be placed on any of the infant's 4 limbs. According to anatomical site, a given pulse oximeter reading may be interpreted as pre- or post-ductal. 'Pre-ductal' refers to all arterial branches of the aorta *prior* to the connection of the ductus arteriosus, from the pulmonary arteries to the aorta. Likewise, 'post-ductal' refers to all arterial branches of the aorta *after* the connection of the ductus arteriosus from the pulmonary arteries to the aorta. The following 3 figures provide a series of anatomical schematics to demonstrate this. Physiological studies demonstrate that, in the majority of cases, the left arm is pre-ductal equivalent, though this should not be assumed in infants with complex congenital cardiac lesions or in infants with established PPHN. (Rüegger et al, 2010)

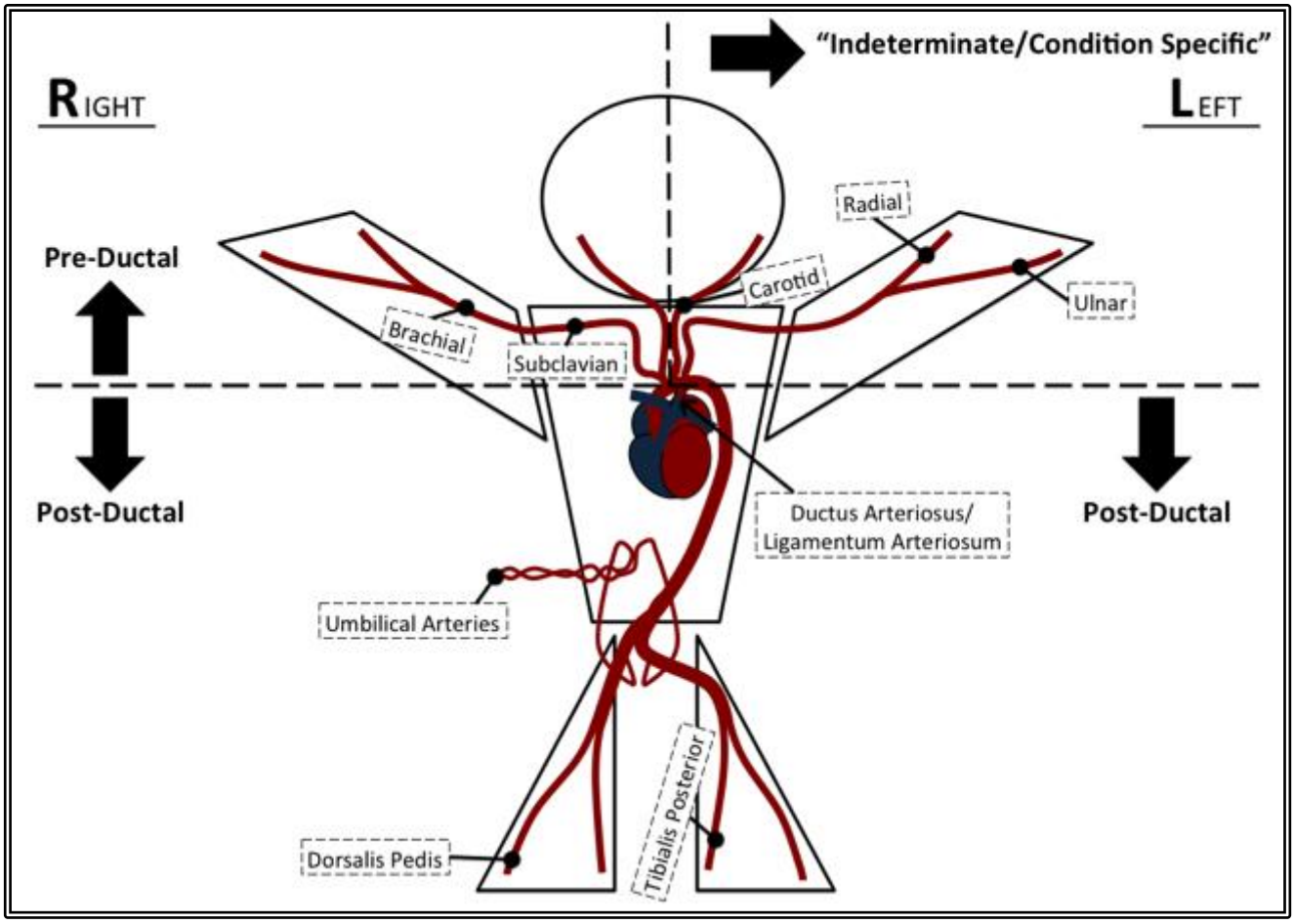
A fabric wrap should be used around the pulse oximeter probe site in order to minimise ambient light interference.



Schematic to demonstrate which limbs are attributable to pre- and post-ductal circulation. In the healthy term infant, the left hand may be considered "pre-ductal equivalent".



Schematic diagram to demonstrate the physiological pre- and post-ductal circulations splitting at the aorta



Schematic diagram to demonstrate the pre- and post-ductal circulations according to the body's major arteries. In the healthy term infant, the left hand may be considered "pre-ductal equivalent", but this may not be the case in established PPHN, transitional circulation & in certain congenital cardiac lesions.

Table of Evidence

Recommendation	Rationale	Grade of Recommendation* & Reference	
Target saturation range 91-95% preferable to 85-89% in the extreme preterm neonate	Reduced mortality and rate of NEC without increase in risk of neurodisability	A	Saugstad & Aune, 2014
Saturations >95% should be avoided in the very low birth weight & extremely premature neonate	Increased rate of ROP & CLD, without benefit in mortality or other co-morbidities	A	Saugstad & Aune, 2011
Saturation range of 91-95% applicable to corrected gestational ages up to 37 completed weeks	Targeting saturations >95% in extreme preterm neonates at corrected gestational ages >32 weeks bears no advantage in growth profile, neurodisability, or grade of ROP. Targeting saturations >95% is associated with a greater incidence of CLD, length of stay, duration of inpatient oxygen therapy and progression onto home-oxygen therapy requirement	B	BOOST STOP-ROP
Alarm limits should be prefixed to encourage tight control within the targeted range (i.e. to alarm at 1% above and 1% below target range)	In comparing major trials, better control was achieved in the RCT with specified alarm limits (COT) versus those that did not specify limits (BOOST II). Even in specifying limits within 2% (COT), or suggesting limits within 3% (SUPPORT), of target range - median oxygen saturations were out of range for a significant portion of time. Compliance with target limits is poor in all trials to date; no studies provide a definitive solution to alarm fatigue, nor definitive answer with respect to alarm limit to target tolerance.	-	COT BOOST II SUPPORT
Target saturations ≥93% in preterm infants with corrected gestation ≥37 weeks, or prior to this if planning for discharge	Infants with CLD are at increased risk of Apparent Life-Threatening Events (ALTE) with saturations below 90% - at saturations >93% they are not. Saturations less than 92% long term, may be	C	British Thoracic Society, 2009

	associated with suboptimal growth in infants with CLD. Saturations above 94–95% appear to reduce pulmonary hypertension, while levels below 88–90% may cause pulmonary hypertension. (Not applicable to congenital cardiac lesions, nor idiopathic pulmonary hypertension)		
Target saturations ≥95% in term infants	Saturations >95% reached on average 12 to 14 minutes post-delivery in healthy term infants. Median average saturations of 98.3% in the 1st 24 hours in healthy term infants. No trials directly compare saturation target ranges in term infants.	D	Toth et al. 2002 O'Brien et al. 2000
In an infant with pneumothorax, supplemental oxygen should be provided to maintain oxygen saturations within the recommended target range for their gestation	Unnecessary administration of oxygen beyond that required to maintain an infant's saturations within target range for gestation does not speed up pneumothorax resolution time but may expose the infant to the risks of hyperoxia as well as conceal or mask a true rise in the infants required FiO_2	C	Shaireen et al. 2014

*Guyatt et al, 2008.

References and Further Reading

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Appendices

Appendix A: *Acknowledgements*

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Appendix B: *The Prescription of Oxygen Therapy & Oxygen Prescription Stickers*

The provision of oxygen to an infant should be regarded as the administration of a medication. As such, trusts should ensure an adequate system is in place to streamline oxygen prescription on medication cards/drug charts or within their electronic prescribing.

As a standard of good practice, the oxygen prescription should then be acknowledged and validated by administering nursing staff by signing against the prescription at twice daily intervals (For example, at each shift change) or within the patient electronic notes.

An Oxygen prescription sticker template may be requested from the EoE neonatal network (Separate file). An example of an appropriate oxygen prescription template is given below:

Oxygen Prescription: Titrate FiO_2 to selected target range		
Preterm (<37 weeks or <1.5kg):……	*91-95%*	☐
Term (≥37 weeks)……..	*≥95%*	☐
Preterm, corrected gestation ≥37 weeks:...	*≥93%*	☐
Risk of /established PPHN:……	*>95%*	☐
Other:…… ____% to ____%		☐
Signed: **Date:**		

Exceptional Circumstances Form

Form to be completed in the **exceptional** circumstances that the Trust is not able to follow ODN approved guidelines.

Details of person completing the form:	
Title:	Organisation:
First name:	Email contact address:
Surname:	Telephone contact number:
Title of document to be excepted from:	
Rationale why Trust is unable to adhere to the document:	
Signature of speciality Clinical Lead: Date:	Signature of Trust Nursing / Medical Director: Date:
Hard Copy Received by ODN (date and sign):	Date acknowledgement receipt sent out:

Please email form to: kelly.hart5@nhs.net requesting receipt.

Send hard signed copy to:

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