# Reporting neonatal intensive care chest radiographs

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S Andronikou, MB BCh, FCRad, FRCR, PhD

Department of Radiology, Stellenbosch University and Tygerberg Hospital

### S Soin, FRCR

John Radcliffe Hospital, Oxford, UK

### Introduction

The chest X-ray (CXR) in a neonate who is being cared for in an intensive care unit (ICU) or special care baby unit (SCBU) is performed for very specific reasons relating to the conditions affecting this age group and the management thereof. These include:

- checking the position of lines and tubes required for monitoring and treatment
- assessing complications of line and tube insertion, ventilation and hydration
- making a diagnosis when the patient presents with respiratory distress
- monitoring progression or resolution and responses to treatment action
- evaluating suspected pathology sustained during the birth
- evaluating pathology detected on prenatal imaging.

### Information is part of your job

To make any sense of the CXR the radiologist must be armed with some useful information:

• Was the neonate born premature or term?

The range of disease varies, e.g. hyaline membrane disease in a premature neonate v. meconium aspiration in a term neonate.

• Was the neonate born today or some days ago?

The range of disease varies, e.g. transient tachypnoea of the newborn should resolve.

• Is this the first film or are there previous films?

This assists with development or resolution of disease and complications.

• Why is the patient being referred?

Is the CXR performed because of an acute deterioration as seen in a pneumothorax or is it performed to assess the position of a new line insertion?

• Is the patient being ventilated using positive pressure?

The patient may be on CPAP (continuous positive pressure ventilation), IPPV (intermittent positive pressure ventilation), oscillation and even ECMO (extracorporeal membrane oxygenation).<sup>1</sup>

### **Stepwise assessment**

Step 1: Check line and tube positions.

Step 2: Look for a complication relating to tube or line placement. Step 3: Look for any complication relating to ventilation or hydration. Step 4: Look for signs of surgery, especially cardiac. Step 5: Determine the cause of respiratory distress.

Step 6: Is there something new – progression or improvement over sequential CXRs?

Step 7: Look for any associated disease, e.g. cardiac or other anomaly. Step 8: Is there any incidental abnormality of significance?

### Step 1: Assessing line and tube positions (Figs 1 - 5)

If you do not do this first you will make a fool of yourself and miss the simplest diagnosis. First get used to the appearances and use of each of the commonly used tubes and lines and then evaluate their position according to an ideal. In neonates, rules of thumb are better than measurements as there are many size variations. Table I lists some of the more commonly used equipment as well as ideal positions and poor positions where appropriate.



Fig. 1. Identification of the variety of lines and tubes and adequate positions for these. • Central line tip in the SVC

• ET-tube tip at the medial ends of the clavicles

- Mediastinal drain enters via a central anterior wall defect
- Pacing wires
- Cardiac monitor leads.

### Steps 2 and 3: Complications relating to tube and line placement and function (Figs 6 - 14)

### Complications of intubation

- Intubation of the right middle lobe bronchus/bronchus intermedius results in right upper lobe collapse with or without left lung collapse (Fig. 9)
- · Traumatic intubation with pneumomediastinum.

### **Complications of ventilation**

• Pneumothorax (Figs 10a - c)



Fig. 2. Identification of the variety of lines and tubes and adequate positions for these.

NGT has its tip below the left hypochondrium and lies within the air of

- the gastric fundus.
- Thermometer has numerous wire elements and differs from the single wire element cardiac leads seen laterally.



- Fig. 3. Identification of the variety of lines and tubes. • Cutaneous  $CO_2$  (capnography) and  $O_2$  monitors have a 'cog' type outline
- The NGT is well sited but the ET-tube is low at the carina.
- Pulmonary interstitial emphysema (Figs 11a and b)
- Pneumomediastinum
- Pneumopericardium
- Chronic lung disease (Fig. 12)
- Pitfall: Skin folds may mimick a pneumothorax but are seen to extend beyond the boundaries of the chest (Fig. 13).

### Complications of a central venous line

- · Fluid overload with pulmonary oedema and increased third space (soft tissue) (Figs 14a and b)
- Arrhythmia when the catheter is in the atrium.
- Thrombosis around the catheter and venous congestion of the neck.
- Incorrect passage of the catheter into a peripheral vessel.

### Complications of umbilical vein catheter

• Hepatic position may result in hepatic cystic masses.



- Fig. 4. Identification of the variety of lines and tubes. There is a left-sided intercostal drain with the side holes located within the thoracic cage

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There are 2 central mediastinal drains An ET-tube, a NGT and cardiac leads are also present.



- Fig. 5. Identification of lines and tubes. A UAC takes a course inferiorly first to enter the internal iliac artery and then should have its tip above the level of the coeliac trunk and below the ductus arteriosus to the left of the midline
- A UVC takes a direct route to the right of the midline to the left portal vein and via the ductus venosus within the liver to enter the inferior vena cava and thereby enter the right atrium.
- · Positions other than the inferior vena cava or the right atrium restrict line use as a central catheter for chemotherapeutic administration and total parenteral nutrition.

### Step 4: Signs of surgery

Are there features of thoracotomy, mediastinal drains, pacing wires or any surgical mediastinal clips? Identification of any features relating to cardiac surgery should trigger a radiological 'cardiac' assessment including evaluation of clips and stents, cardiac size, situs, position of aortic arch, features of pulmonary plethora and cardiac failure (Fig. 15)

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Туре	Ideal position	Undesirable position
External		
Cutaneous O2 and CO2 monitors	N/A	N/A
Cardiac leads	N/A	N/A
Thermometer	N/A	N/A
External part of pacing leads	N/A	N/A
Internal		
Endotracheal tube	Medial clavicles	High = above clavicles
	Away from carina (1 - 1.5 cm)	Low = at / below carina
	Head bent forward = low	
	Head bent back = high	
Umbilical artery catheter	Above coeliac or below renal arteries	High = near ductus
	Via internal iliac artery,	Low = aortic bifurcation
	i.e. first a downward course	
Umbilical vein catheter	Inferior vena cava above liver	Liver in hepatic vein
	Direct course up via left portal	Tributary vein
	and ductus venosus	
Long venous line	Away from renal veins	Near or in renal vein
Central venous line	Superior vena cava	Atrial
Nasogastric tube	Left hypochondrium / stomach or	Oesophagus / bronchus
Nasojejunal tube	jejunum respectively	Looped in mouth
Intercostal / mediastinal drain	Intrathoracic / mediastinal	Side holes external
		Subcutaneous

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Fig. 6. Complications of lines and tubes. Right-sided central catheter has its tip too far - beyond the right atrium. The nasogastric tube and umbilical vein catheter tips are well positioned.

## Step 5: Determining the cause of the respiratory distress

The differential diagnosis of respiratory distress in a neonate can be simplified by considering whether the neonate was born term or premature (Table II). It can be further simplified by knowing the causes of increased



Fig. 7. Complications of lines and tubes. The endotracheal tube has its tip in the right main bronchus and this is complicated by right upper-lobe collapse.

lung density and relating them to lung volumes as well as the distribution (bilateral, symmetrical, and diffuse) of density.

**Pitfalls:** when the position of the ET tube is not considered (affects density), when the patient is being ventilated with positive pressure (affects lung volume), when exogenous surfactant has been adminis-

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Fig. 8. Poor line positioning. The umbilical vein catheter has its tip near the renal veins, which is undesirable. The umbilical artery catheter is positioned above the renal arteries.

tered (may change distribution), when there is superadded infection or haemorrhage (affects distribution and density), if the ductus arteriosus has remained patent (affects density) and when incidental or associated pathology has not been recognised. Remember that the most common cause of a lobar consolidation is a low ET tube or a mucus plug,<sup>1</sup> that the most common cause of diffusely dense lungs is underaeration /expiratory film<sup>2-4</sup> and that surfactant therapy via the ET tube may result in both patchy symmetrical and asymmetrical density as well as pulmonary haemorrhage.<sup>4,5</sup> Also a differential density between the hemithoraces is often due to rotation and may be confused with unilateral diseases such as infection or effusion (Fig. 16). (Tip: This should be assessed every time there is differential density by looking at the length of the anterior ribs visible and not by checking the medial ends of the clavicles against the spinous process as in adults.) Lastly, recognise that you may not always come to one single diagnosis and that infection, haemorrhage and oedema may co-exist with respiratory distress syndrome (RDS).



Fig. 9. Complications of lines and tubes. The endotracheal tube is low and has resulted in both right upper lobe and left lung collapse. The other tubes (NGT and UVC) are well positioned.

Take all findings into account and advise clinicians as to the most likely diagnosis that fits the clinical picture.

**Meconium aspiration:** This occurs when the foetus passes meconium, which is hyperosmolar, in utero due to hypoxaemia.

X-ray findings: Patchy but widespread collapse and consolidation with patchy air trapping. Can result in complete diffuse opacity.

Associations: Pulmonary interstitial emphysema (PIE) and pneumothorax. Susceptible to infection and secondary surfactant deficiency (Fig. 17).<sup>1,6</sup>

**TTN (transient tachypnoea of the newborn)** / **wet lung:** This occurs when there is impaired clearing of fluid from the lungs within 4 - 6 hours of life. It is more common after caeserian section and birth asphyxia.

X-ray findings: Bilateral increased density but predominantly from prominent vessel markings in the presence of normal or increased lung volumes.

Associations: Septal lines and effusions. Rapid recovery occurs

Table II. Causes of respiratory distress in neonates presented on CXR <sup>1,3,4,6,7</sup>			
Term neonate	Premature neonate	CXR findings	
Meconium aspiration	Aspiration		
Infection	Infection	Increased lung density	
Transient tachypnoea of the newborn (TTN)/	Transient tachypnoea of the newborn (TTN)/	Symmetrical or asymmetrical	
Wet lung	Wet lung		
	Respiratory distress syndrome (RDS) / hyaline membrane disease (HMD)	Focal or diffuse	
	Pulmonary haemorrhage		
Spontaneous pneumothorax	Spontaneous pneumothorax	Crescentic lucency	
	Patent ductus /persistent foetal circulation	Linear markings Increased vascularity	

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Fig. 10. Complications of ventilation and poor tube position. (a) There is a large pneumomediastinum as a result of barotrauma due to ventilation. This results in the 'continuous diaphragm sign'. In addition

to Vertiliation. This results in the continuous diaprilagin sign, in addition the ETT is low. (b) There is a right-sided pneumothorax in a patient with underlying hya-line membrane disease. The intercostal drain which is present for treating the pneumothorax can be seen with its tip and side holes subcutaneously rather than in the chest cavity. The ring-like density represents a cutane-ous cannodranhy probe

(c) There is an unusually distributed left pneumothorax causing depression of the diaphragm. The umbilical vein catheter has its tip in the right atrium which is considered appropriate for hyperalimentation by some, and at risk for thrombosis by others.





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Fig. 11. Complications of ventilation. (a) Bilateral symmetrical airspace disease consistent with hyaline mem-brane disease requiring ventilation.

(b) The same patient as in Fig. 11 (a) demonstrating subtle diffusely distributed oval lucent cysts compatible with early pulmonary interstitial emphysema as a result of ventilation.



Fig. 12. Complications of ventilation. After chronic ventilation on oxygen, this patient has developed changes compatible with chronic lung disease, namely heterogeneously distributed areas of collapse and air-trapping, resulting in a coarse pattern.

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Table III. Other causes of respiratory distress in a neonate<sup>1,4</sup>

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Diagnosis
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Diaphragmatic hernia

Pulmonary agenesis / hypoplasia

Congenital cystic adenomatoid malformation (CCAM) / sequestrated segment

Congenital lobar emphysema

Cardiac failure / left to right shunt / congenital heart disease

Persistent foetal circulation / patent ductus arteriosus (PDA)

Pulmonary oedema

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Fig. 13. Pitfalls in image interpretation. A pneumothorax is simulated on the left by skin folds. These are distinguished by the fact that they extend beyond the limits of the chest and because lung markings are present lateral to the 'edge'.

within 12 hours but there may be reticulonodular densities at 48 - 72 hours. If there is no recovery or improvement within this time consider patent ductus arteriosus in a premature neonate or partial anomalous pulmonary venous drainage in a term neonate. (Fig. 18a and b).<sup>16,7</sup>

**RDS** (respiratory distress syndrome) / HMD (hyaline membrane disease): Related to surfactant deficiency, this is the most common cause of respiratory distress in a premature neonate. It results in non-closure of the ductus arteriosus due to hypoxia and raised prostaglandin E2.<sup>34,8</sup>

Findings are progressive: Ground glass density resulting from atelectasis and hyperinflation with decreased volume (unless patient is ventilated when volume may be normal or high).

Increasing density results in loss of cardiac and diaphragmatic margins first, then air bronchograms developing and then filling in of these resulting in 'white out' (Fig. 19a-e).<sup>13,4</sup>

**Neonatal pneumonia:** Usually due to Group B Beta haemolytic Streptococcus.

Findings: Asymmetrical lung density. May co-exist with respiratory distress syndrome (Fig. 20).<sup>1,3,4</sup>

Usually diagnosed prenatally

Delayed diagnosis



Fig. 14. Complications of intravascular fluid therapy.

(a) Fluid-overload using an intravascular catheter may result in increased third space fluid manifesting here as massive soft tissue thickening around the chest. There should be very little soft tissue in a neonate. The ETT is noted to have its tip low in the airway.

(b) Bilateral asymmetrical peri-hilar airspace disease is consistent with pulmonary oedema as a result of fluid overload via the central catheter. This was confirmed as it resolved after diuretic therapy. The ETT in this patient is low in the airway.

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Fig. 15. Previous surgery. In addition to the ETT and NGT there is a clip for repair of the patent ductus arteriosus. There is an abnormal rib noted on the left consistent with this previous surgery. The asymmetrical airspace disease is in keeping with superadded infection.



Fig. 16. Pitfall. Asymmetrical density involving one lung may be simulated by rotation as in this patient who actually has bilateral symmetrical 'granular' airspace disease in keeping with hyaline membrane disease. To determine whether the patient is rotated compare the lengths of the anterior ribs (and not the clavicular ends as done in adults).



Fig. 17. Meconium aspiration. Bilateral airspace disease in a term neonate



Fig. 18 (a) and (b). Transient tachypnoea of the newborn. Clearing of the bilateral airspace disease at 24 hours.

**Other causes of respiratory distress in a neonate:** Other pathology may be diagnosed pre-natally or present acutely with respiratory distress post-natally. Table III summarises some alternatives to the differential diagnosis of respiratory distress in a neonate. Institution of appropriate therapy such as diuretic therapy for cardiac failure may lead to an improvement that helps in the diagnosis (Fig. 21).

A word on CLD (chronic lung disease) /BPD (bronchopulmonary dysplasia): This is the result of oxygen treatment and positive pressure ventilation during the first week of life for a minimum of 3 days.<sup>1,2</sup> The definition of this diagnosis is  $O_2$  requirement at 36 weeks post conception age with an abnormal chest X-ray and respiratory distress.<sup>1,2</sup> It is the most common cause of chronic respiratory failure in children.

Findings: hyperaerated with reticular areas of fibrosis.

Associations: cardiomegaly from cor pulmonale and myocardial toxicity (Fig. 12).

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## Step 6: Is there improvement or deterioration?

Rapid resolution in the X-ray findings in a patient with bilateral diffuse lungs is in keeping with TTN whereas progressive changes that follow the stages of RDS probably support that diagnosis. The position of the ET tube or a vascular catheter may result in a collapsed or consolidated lung lobe and then follow-up X-ray should indicate repositioning and resolution. Sometimes follow-up X-rays after physiotherapy showing





Fig. 19 (a) - (e). Hyaline membrane disease / ARDS. There is progression from a bilateral symmetrical diffuse granularity of the lungs to a more confluent airspace process with loss of cardiac and diaphragmatic margins and eventual 'white out'. There may be some improvement in a patchy distribution when surfactant is 'sprayed' down the ET tube. Lung volumes should be small BUT this is not often a radiological feature as the patient requires positive pressure ventilation which gives the impression of large lung volumes.



Fig. 20. Neonatal pneumonia. Right upper lobe airspace disease.

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Fig. 21. Cardiac failure. The predominant distribution of airspace disease in this patient with cardiac failure is characteristically bilateral peri-hilar. There are features in keeping with cardiac surgery on the radiograph including a mediastinal drain and pacing leads. Looking at the lines and tubes therefore is important for distinguishing causes of airspace disease.



Figs. 22 (a) and (b). Cardiac failure. Complete white out of the lung parenchyma bilaterally in a patient who has features of previous cardiac surgery (PDA clips and thoracotomy changes on the left). After fluid restriction the patient demonstrates re-appearance of the cardiac and diaphragmatic margins consistent with some resolution of the pulmonary oedema.







(a) A patient with pulmonary oedema due to cardiac failure associated with Down's syndrome also demonstrates the common association of duodenal stenosis with a relatively gasless distal bowel.
(b) This patient has cardiomegaly with a visible curvilinear 'scimitar' vessel descending to the diaphragm and a smaller right lung. The features are been of pulmonary by papera.

descending to the diaphragm and a smaller right lung. The features are those of partial anomalous venous drainage and pulmonary hypoplasia constituting the scimitar syndrome.

resolution of a lobar density support the diagnosis of a mucus plug rather than super-infection. Response of cardiac failure and fluid overload as well as third space fluid to diuretic therapy also helps to establish a strong diagnosis of parenchymal density on X-ray (Figs 22a and b). Deterioration of parenchymal lung changes in RDS (Figs 19a-e) with increasing ventilation requirements may result in PIE or pneumothorax which can only be detected on follow-up X-rays.

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Fig. 24. The features below the diaphragm are also important and are often associated with prematurity. In this patient there is mural bowel gas in keeping with necrotising enterocolitis.

### Steps 7 and 8: Are there any associated abnormalities or incidental findings?

There may also be associated anomalies or abnormalities contributing to respiratory distress or the X-ray findings. Remember that where there is one anomaly, such as a cardiac anomaly, this may be associated with oesophageal anomalies (e.g. atresia, tracheo-oesophageal fistula) which may contribute to parenchymal lung density through aspiration or infection. Chest X-ray findings may be confused in situations such as pulmonary hypoplasia where the decreased lung volume and density may be misinterpreted as collapse, or congenital lobar emphysema which may be mistaken for a pneumothorax. Situs inversus associated with congenital heart disease may be mistaken for a deviated mediastinum due to collapse. Always assess for associated anomalies and obtain a history of anything that may have been detected prenatally (Figs 23a and b). Also remember to look under the diaphragm. Necrotising enterocolitis occurs in the same premature neonates as RDS and may be noted on CXR (Fig. 24).

### Conclusion

Have an organised method of reporting in neonatal ICU. Remember to report the lines and tubes first and to interpret any findings firstly relating to the equipment being used and then according to the suspected pathology. Keep the pitfalls in mind at all times and remember that RDS, infection, aspiration and pulmonary oedema may be indistinguishable. Lastly, listen to the ICU clinicians, because they have the 'secret' knowledge, and try to interpret the findings in relation to the clinical situation.

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