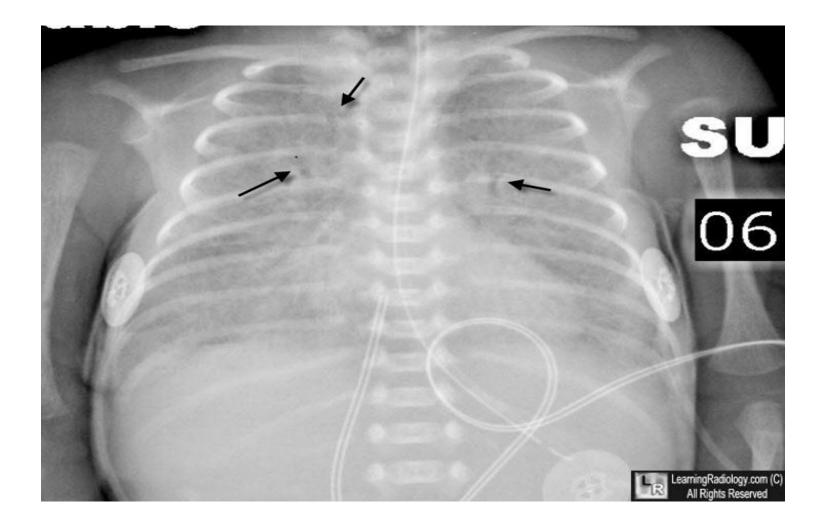
Disease specific ventilation in newborn

Disease specific ventilation

• Case 1

Preterm (30 wk)/1200gms/ Vaginal delivery CIAB/Respiratory distress at birth/ANS uncovered

Delivery room CPAP given Shifted to NICU



RDS

RDS- Key Pathophysiologic Features

1. Lung Surfactant- Quantitative, qualitative, and metabolic disturbances in lung surfactant.

Decreased compliance of distal airspaces- lead to atelectasis, ventilation:perfusion mismatch, and intrapulmonary shunt.

RDS- Key Pathophysiologic Features

 2. Lung Liquid- Reduced clearance Sustained production
3. Development- Canalicular-saccular stage Thickened mesenchyme Immature capillary development

1) Minimize the initial use and/or duration of exposure to any form of invasive mechanical ventilation.

2) Promote weaning and extubation from mechanical ventilation

- RDS lung have low compliance and low resistance short time constant
- Low resistance and short time constant results in quick emptying and collapse of lung in expiration- Managed by adequate PEEP.
- Because of short time constant, a higher ventilator rate is used

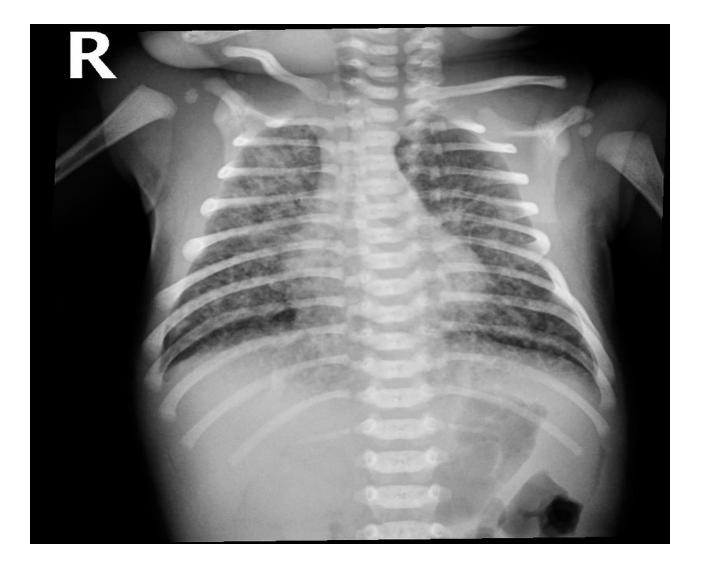
Ventilator settings in RDS

- Target tidal volume- 4-5 ml/kg
- PEEP 5-6 cm H2O
- Respiratory rate- 50-60 /min
- Inspiratory time- 0.25-0.35 seconds
- PIP- guided by visual appreciation of a just adequate chest rise, audible breath sounds and target tidal volume of 4-5 ml/kg.

Case-2

 Term(38 wk)/3000 gms delivered to primigravida mother via LSCS (↓ FHR)

• Baby did not cry after birth- delivery room resuscitation



MAS

MAS-Key Pathophysiologic Features

- MAS has a complex, multifactorial pathophysiology.
- **1. Surfactant Dysfunction-**Disturbances in surfactant metabolism and function lead to decreased compliance of distal airspaces leading to atelectasis and intrapulmonary shunt.

MAS-Key Pathophysiologic Features

2. Airway Resistance- elevated airway resistance because of obstruction from inhaled/aspirated meconium.

3. Pulmonary Hypertension- Fetal hypoxemia and inflammation are thought to be primary contributors to underlying pulmonary hypertension.

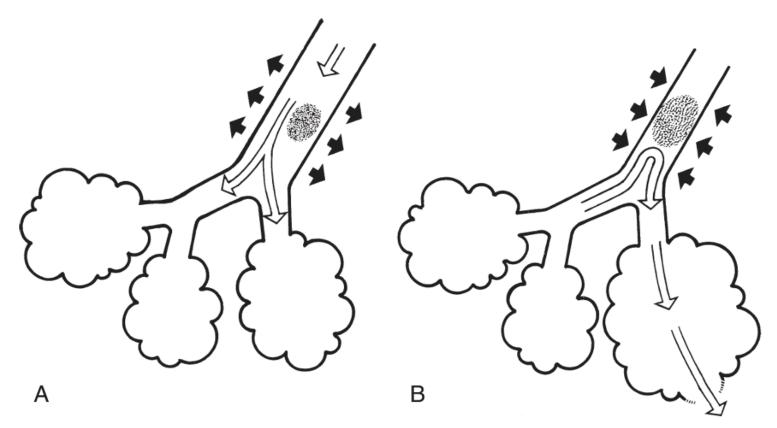


Fig. 25.2 Partial "ball-valve" air trapping behind particulate matter (i.e., meconium) in an airway, which leads to alveolar overexpansion and rupture. **(A)** Tidal gas passes beyond the meconium on inspiration when the airway dilates but **(B)** cannot exit on expiration when airways constrict. (From Goldsmith JP: Continuous positive airway pressure and conventional mechanical ventilation in the treatment of meconium aspiration syndrome. J Perinatol 28(suppl 3):S49–S55, 2008; used with permission.)

- Alveolar Disease w/ Low Lung Volume-↓Surfactant function,↓Lung compliance
 ↑ V/Q mismatch

• Air trapping (caused by dynamic PEEP) because of long expiratory time constant -Thus, set PEEP should be limited

• Ventilator rate should be kept relatively low to ensure adequate expiratory time to minimize gas trapping.

• As inspiratory time constants can also be prolonged- ensure I-time is long enough to complete VT delivery.

• Because the pathophysiology of MAS includes increased alveolar dead space, these infants require slightly larger VT/kg than similar infants with more homogenous lung disease.

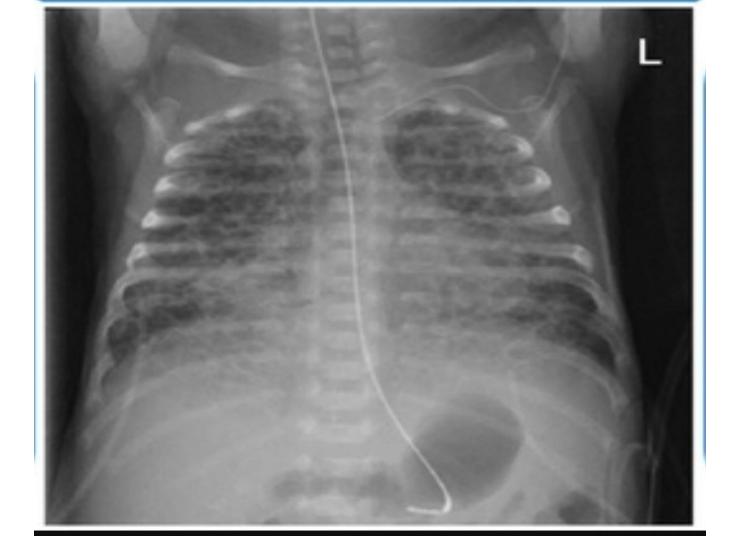
Ventilator settings in MAS

- Target tidal volume- 5-6 ml/kg
- PEEP 4-5 cm H2O
- Respiratory rate- 30-40 /min
- Inspiratory time- 0.35-0.5 seconds
- PIP- guided by visual appreciation of a just adequate chest rise, audible breath sounds and target tidal volume of 5-6 ml/kg.

Case 3

- Day of life-60 (PMA- 34+5 wk)
- Birth History- PT 26 wk/800 gm/ VD
- Required surfactant
- Baby admitted in NICU since birth
- Failure to extubate

BPD



BPD-Key Pathophysiologic Features

Lung Pathology

- Abnormalities in both the airways and the gas exchange areas of the lungs.
- interrupted alveolarization with reduced number and increased size of the remaining saccularalveolar structures
- thickened mesenchymal/septal tissues
- disrupted growth and development of the pulmonary microvasculature
- fibrosis

TABLE 25.14Abnormalities ofLung Function among Infants WithBronchopulmonary Dysplasia

ParameterAbnormalityLung VolumeDecreasedOverall lung volumeDecreasedFunctional residual volumeDecreasedComplianceReducedGas exchangeImpaired diffusion

Airway Function

Expiratory flow velocity Resistance

Decreased Increased

- Increased anatomical and functional dead space increases in delivered VT may be necessary.
- In the developing stages of BPD the optimal approach to ventilator support is similar to that recommended for the management of RDS.

• With more chronic, severe forms of BPD- higher VT may be needed sometimes as high as 10 to 12 mL/kg

Reasons-

- 1. Interrupted/impaired alveolarization with reduced gas exchange surface area
- 2. increasing nonfunctional lung volume because of increased areas of atelectasis coupled with areas of overinflation(increased alveolar dead space)
- 3. dilatation of large airways owing to exposure to cyclic stretch from positive-pressure ventilation (acquired tracheomegaly)

• Tracheal and bronchomalacia in chronicsevere BPD - increased PEEP levels are required

• Increased airway resistance - managed via longer inspiratory times to allow for more complete distribution of VT.

• A longer expiratory time is also needed for the lung to effectively empty during the exhalation phase.

• Thus, the combination of higher VT, longer inspiratory times, and low respiratory rates (allowing for increased exhalation time) is indicated for infants who remain ventilator dependent with more chronic-severe forms of BPD.

Ventilator settings in BPD

- Target tidal volume- 6-10 ml/kg
- Respiratory rate- 20-30 /min
- Inspiratory time- 0.5-0.7 seconds
- PEEP variable, may need 6-8 cm H2O

Ventilation in lung hypolasia disorders

Lung hypoplasia disorders- Key Pathophysiologic Features

- Most obvious pathophysiologic problem is impaired lung growth.
- Severity of impact on airways, terminal respiratory units, and the pulmonary vascular bed depends on how early in gestation lung growth is affected

- Impaired vascular development accompanies the altered lung growth
- 1. Impaired growth and development of pulmonary arteries and arterioles
- 2. increased arteriolar medial muscle thickness
- 3. altered expression of angiogenic factors including vascular endothelial growth factor.
- 4. decreased endothelial NO synthase expression

- Lung is small, with a functional residual capacity that may be considerably less than normal.
- Gentle approach to ventilation- to minimize lung injury from excessive aggressive ventilator strategies to achieve oxygenation.
- Ventilate with adequate PEEP and lowest possible PIP (i.e smaller than usual tidal volume) to achieve oxygenation.

Ventiltor settings in Lung hypoplasia disorders

- Lowest possible PIP (less than 25 cm H2O)
- TV- 4 ml/kg
- Respiratory rate- 50-60/min
- Inspiratory time- 0.25-0.35 seconds
- PEEP 4-5 cm H2O

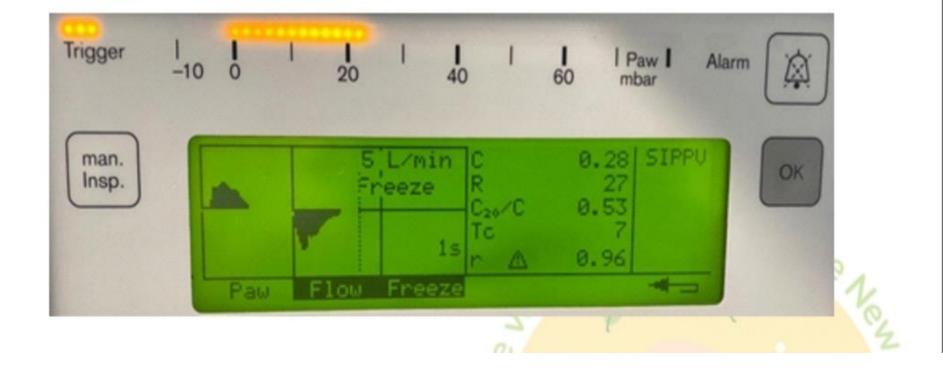


OSCE 1

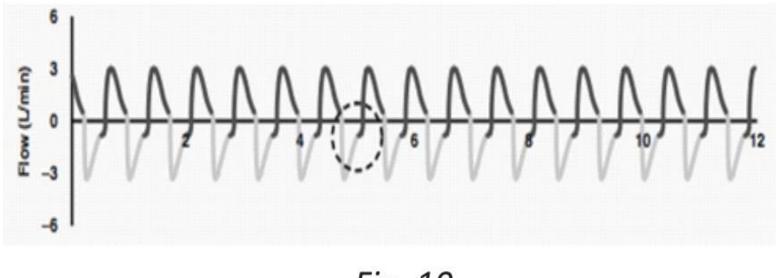
A term baby boy is born limp and gets intubated in LR. Baby put on SIMV mode PIP 17, PEEP 6 FiO2 40% rate of 40/min. His Initial CXR..



- Diagnosis?
- Formula for calculating LHR?
- . What value of LHR indicates a good prognosis?



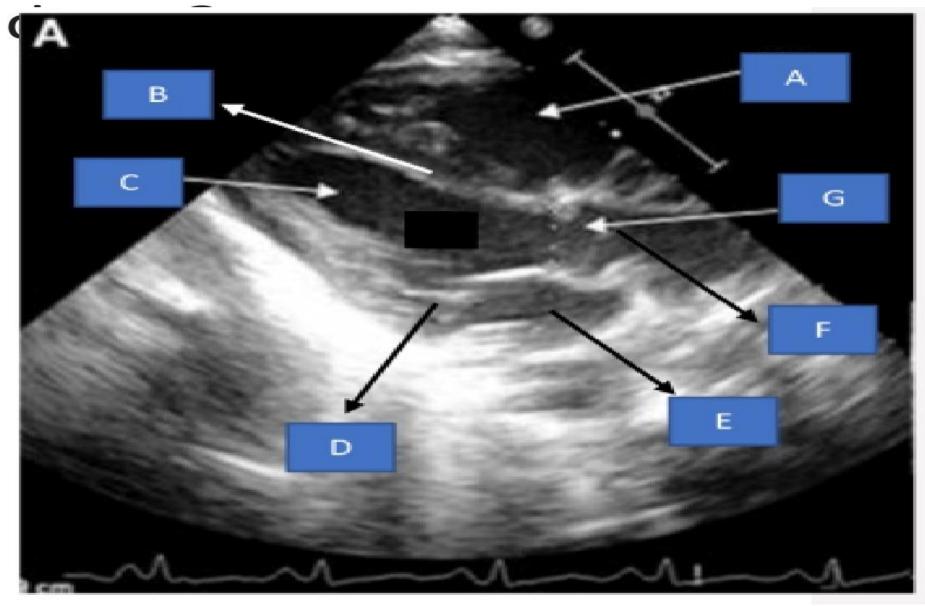
- What kind of waveform is this? And parametes on Y and X aaxis?
- Identify the problem in given waveform?



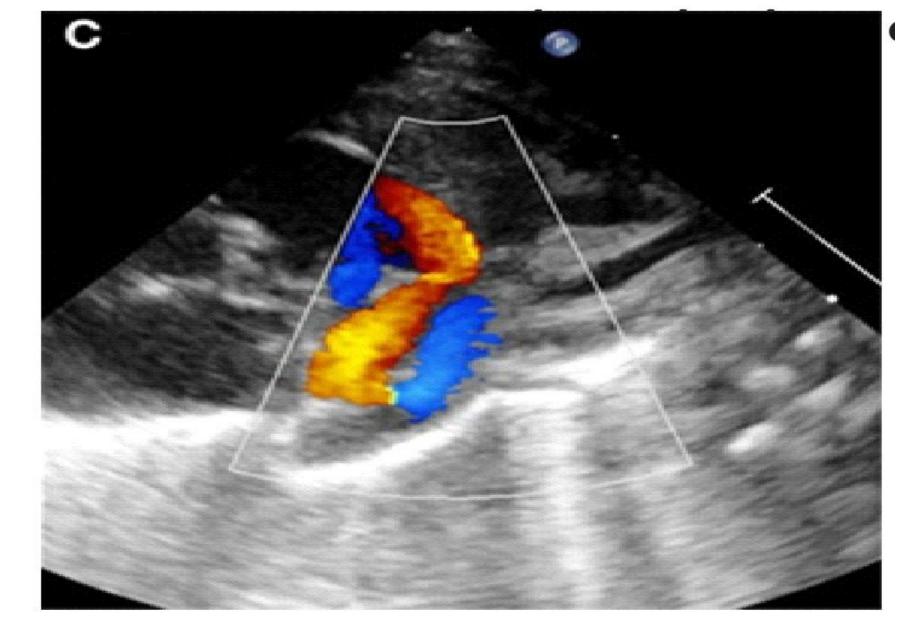
Fia. 10

- Identify the abnormality in given waveform?
- What complication can occur due to this abnormality?

 A 27 wk 980 gm baby on SIMV mode of ventiltion has worsening clinical conditionon day 5 of life. Baby has increasing FiO2 and pressure requirement. Sepsis screen is negative. ECHO was done.



- Identify the view?
- Identify the strucutres?



• Identify the abnormality?

