Neonatal Ventilation Workshop

Organized by the National Neonatology Forum, Delhi
& IAP East Delhi
Venue: UCMS & GTB Hospital, Date: 21st March 2021

Time	Topic	Scope
Registration at 9:00am -9:30 am		
9:30 am -10:10 am	BASICS OF VENTILATION	
9:30 am-10:10am	ABC's of Ventilation	Terminology, Applied physiology Dr. Kumar Ankur
10:10 am -10.50am	Non-Invasive Ventilation	CPAP/HHHFNC Dr. Avneet Kaur
10.50 am - 11.00 am	Inaugration ----Tea & Breakfast	
11:00am – 01:00pm	WORKSTATION (30 mins each)	
11:00am -11:30am A	Setting up of a ventilator (Also Discussed about- Display Parameters)	Intubation, different ventilator circuits, humidifiers, setting up of a ventilator Dr. Avneet Kaur/Dr Varun Vij
11:30am -12:00 pm B	Disease Specific Neonatal Ventilation (HFOV/iNO- would be discussed in another workstation)	RDS/MAS/PPHN/Asphyxia (Talk about only Non Invasive/Invasive Ventilation & Surfactant) Dr T j Antony/ Dr Sankalp Dudeja
12:00pm -12:30pm C	Non Invasive Ventilation: Neonates	T-piece/HHFNC/CPAP/NIMV Dr Anil Batra/ Dr Gaurav Jawa
12:30pm -1:00pm D	Modes of Ventilation	SIMV/PSV/SIPPV or AC/VG Dr Tapas Bandyopadhyay/Dr Vinay Kumar Rai
1:00pm – 2:00pm	LUNCH BREAK	
2:000pm -2:30pm	Weaning & NIV	Dr Ravi Sachan
2:30pm- 4:30pm	WORKSTATION (30 mins each)	
2:30pm- 3.00pm A	HFO Ventilation + iNO	When/How to set up/How to wean Dr. Anup Thakur/Dr Abhishek Chopra
3.00pm- 3:30pm B	Supportive care of ventilated infants	Weaning strategy/VAP/Care/Feeding Dr. Sanjeev Chetry/ Dr Surender S Bisht
3:30pm- 4:00pm C	How to Read Chest Xray	Dr. Jagjeet Dalal/Dr Jay Kishore Singh
4:00pm- 4:30pm D	Pumonary Graphics	Dr Vivek Choudhury/ Dr Vikram Bedi
4:30pm-5.30 pm	Panel Discussion (Difficult Case Scenario)	**Moderator:** Dr Naveen Gupta **Panelists:** Dr Anup Thakur/ Dr T J antony/Dr Jagjeet Dalal/Dr Abhishek Chopra/Dr Jay Kishore Singh
Valedictory & Feedback		

NEONATAL VENTILATION WORKSHOP MANUAL

21st MARCH 2021

NNF DELHI
UCMS & GTB HOSPITAL, DELHI

NEONATAL VENTILATION

Contents

Introduction

Mechanical ventilation is an age old proven technology commonly used in neonates, infants and older children in intensive care units for various indications.

It is often confusing for the pediatrician who is starting to learn regarding ventilation to keep up with various technical terms and abbreviations being used by ventilator companies and neonatologist and pediatric intensivists.

This manual has been prepared with a view to be simple, practical , concise and easy to read for Pediatriciansand NICU residents and fellows using conventional mechanical ventilation for commonly seen conditions such as respiratory distress syndrome(RDS), meconium aspiration syndrome(MAS), persistent pulmonary hypertension (PPHN), pneumonia and various neurological conditions .

Standard Terminology has been used and defined in the text for convenience of the reader. All abbreviations used in the text are explained in separate page to avoid confusion. Ventilation technique specifically applicable to neonate or an older child has been indicated wherever necessary to avoid any confusion.

We hope that this manual is found to be helpful to the user in management of common conditions requiring CPAP, invasive and noninvasive mechanical ventilation in neonates.

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Basics of mechanical ventilation

Praveen Khilnani

Introduction

Basic principles of physics and gas flow apply to all age groups, anatomical and physiological differences play a significant role in selecting the type of ventilator as well as the ventilatory modes and settings.

Upper airway in children is cephalad, funnel shaped with narrowest area being subglottic(at the level of cricoid ring),as compared to adults where the upper airway is tubular with narrowest part at the vocal cords. Airway resistance increases inversely by 4th power of radius ; i.e. in an already small airway even one mm of edema or secretions will increase the airway resistance and turbulent flow markedly necessitating treatment of airway edema, suctioning of secretion ,measures to control secretions. Low functional residual capacity (FRC: Volume of air in the lungs at end of expiration) reduces the oxygen reserve ,reduces the time that apnea can be allowed in a child.

Respirations are shallow and rapid due to predominant diaphragmatic breathing, and inadequate chest expansion due to inadequate costovertebral bucket handle movement in children. Therefore a child tends to get tachypneic rather than increasing the depth of respiration in response to hypoxemia. Oxygen consumption/kg body weight is higher therefore tolerance to hypoxemia is lower.

Susceptibility to bradycardia in response to hypoxemia is also higher due to high vagal tone. Pores of Kohn and channels of Lambert (broncho alveolar and interalveolar collaterals) are inadequately developed making regional atelectasis more frequent. Closing volumes are lower and airway collapse due to inadequate strength of the cartilage in the airways is common making a child particularly susceptible to laryngomalacia and tracheo- bronchomalacia as well as lower airways closure at a higher lung volume.

Therefore, children tend to require smaller tidal volumes, faster respiratory rates, adequate size endotrachel tubes and adequately suctioned clear airways for proper management of mechanical ventilation. Other important factors for choosing ventilatory settings include the primary pathology ie asthma, ARDS, pneumonia, airleak syndrome, raised intracranial tension, neuromuscular weakness, neonatal hyaline membrane disease, or neonatal persistent pulmonary hypertension(PPHN).

Basic mechanics of ventilation

During spontaneous breathing pleural pressure is negative. During inspiration active work is done to generate the gradient between the mouth and pleural space as the driving pressure for inspired gases to enter the alveolus, and this gradient is needed to overcome resistance and to maintain the alveolus open, by overcoming elastic recoil forces.

Therefore, a balance between elastic recoil of the chest wall and the lung determines lung volume at any given time. Expiration is passive. During positive pressure ventilation pressure gradient generated by the ventilator at the mouth (or endotracheal tube) is higher than the

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pleural pressure which is also positive, however at the end of inspiration,expiration is again passive though it can be manipulated by application of positive pressure to prevent complete deflation at the end of expiration.(PEEP : positive end expiratory pressure).

Two main issues are important physiologically during mechanical ventilation: Ventilation and Oxygenation

Ventilation

Ventilation washes out carbon dioxide from alveoli keeping arterial PaCO2 between 35-45 mm of Hg. Increasing dead space increases the PaCO2.

PaCO2= k x $\frac{\text{Metabolic production}}{\text{Alveolar minute ventilation}}$

Alveolar MV = respiratory rate x effective tidal volume

Effective TV = TV - dead space

Dead Space = Anatomic(nose, pharynx, trachea, bronchi) + Physiologic(alveoli that are ventilated but not perfused)

Adequate minute ventilation is essential to keep PaCO2 within normal limits.

Oxygenation

Partial pressure of oxygen in alveolus (PAO2) is the driving pressure for gas exchange across the alveolar-capillary barrier determining oxygenation.

PAO2 = ({Atmospheric pressure - water vapor}xFiO2) - PaCO2 / RQ

RQ= respiratory quotient

Adequate perfusion to alveoli that are well ventilated improves oxygenation.

Hemoglobin is fully saturated 1/3 of the way through the capillary.

Hypoxemia can occur due to:

    a.Hypoventilation
    b.V/Q mismatch(V- ventilation,Q- perfusion)
    c.Shunt(Perfusion of an unventilated alveolus, atelectasis, fluid in the alveous)
    d.Diffusion impairments

Hypercarbia can occur due to:

    a.Hypoventilation
    b.V/Q mismatch
    c. Dead space ventilation

Gas Exchange

Hypoventilation and V/Q mismatch are the most common causes of abnormal gas exchange in the PICU.

Hypoventilation can be corrected by increasing minute ventilation.

V/Q mismatch can be corrected by increasing the amount of lung that is ventilated or by improving perfusion to those areas that are ventilated.

Concept of Time constant

Time constant is the time required to fill an alveolar space (or empty it). It depends on the resistance and compliance. In the pediatric age group one time constant that fills an alveolar unit to 63% of its capacity is 0.15 seconds. It takes three time constants to achieve greater than 90% capacity of the alveolar unit filled.

Time constant = Resistance (Pressure X Time/Volume) x Compliance (volume/pressure)

This signifies that a certain minimum inspiratory time is required to fill the alveoli adequately which is generally two to three time constants ;i.e. 0.3 to 0.45 seconds. This is important when selecting the inspiratory time on the conventional ventilator.

Indications of mechanical ventilation

Indications remain essentially clinical and may not be always substantiated by objective parameters such as blood gas analysis.

Common indications include:

1. Respiratory Failure
    a. Apnea / Respiratory Arrest
    b. Inadequate ventilation
    c. Inadequate oxygenation
    d. Chronic respiratory insufficiency with failure to thrive
2. Cardiac Insufficiency /Shock
    a. Eliminate work of breathing
    b. Reduce oxygen consumption
3. Neurologic dysfunction
    a. Central hypoventilation/ frequent apnea
    b. Patient comatose, GCS(Glasgow Coma Score) ≤ 8
    c. Inability to protect airway
4. Post operative ventilation

Commonly used Nomenclature for Mechanical ventilation

Airway pressures

Peak inspiratory pressure (PIP)
Positive end expiratory pressure (PEEP)
Pressure above peep (PAP or δp)
Mean airway pressure (MAP)
Continuous positive airway pressure (CPAP)

Continuous Positive Airway Pressure (CPAP)
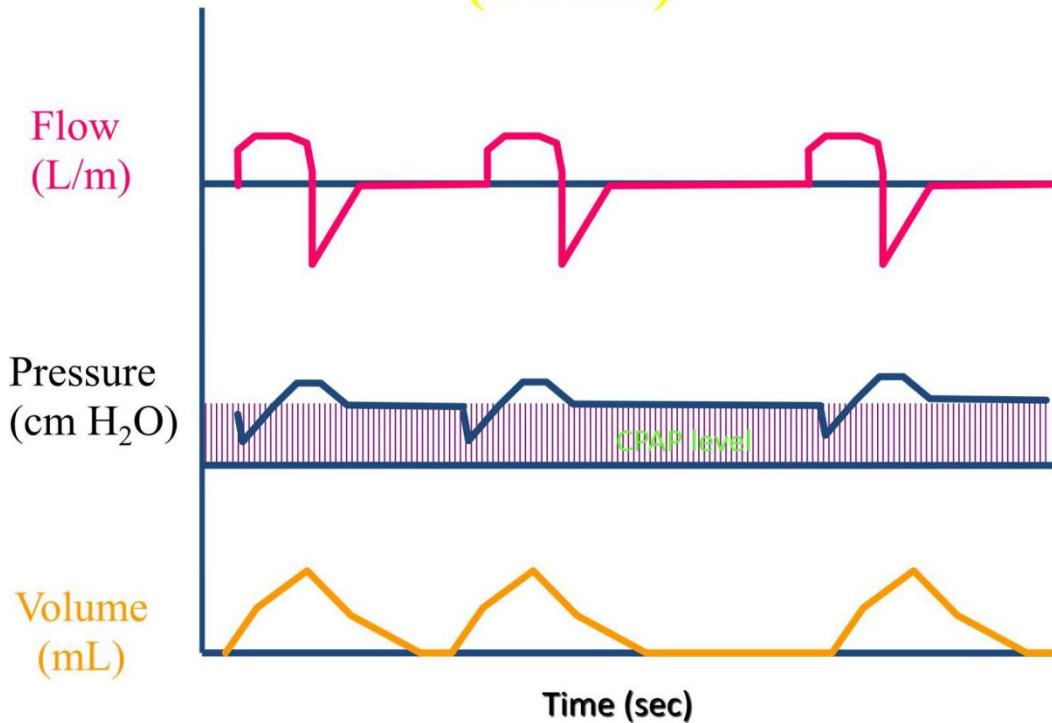
Graph showing Factors affecting Mean airway Pressure (and oxygenation)

1 Inspiratory time
2.Peak inspiratory pressure and
4. Expiratory time (and pause time with PEEP)
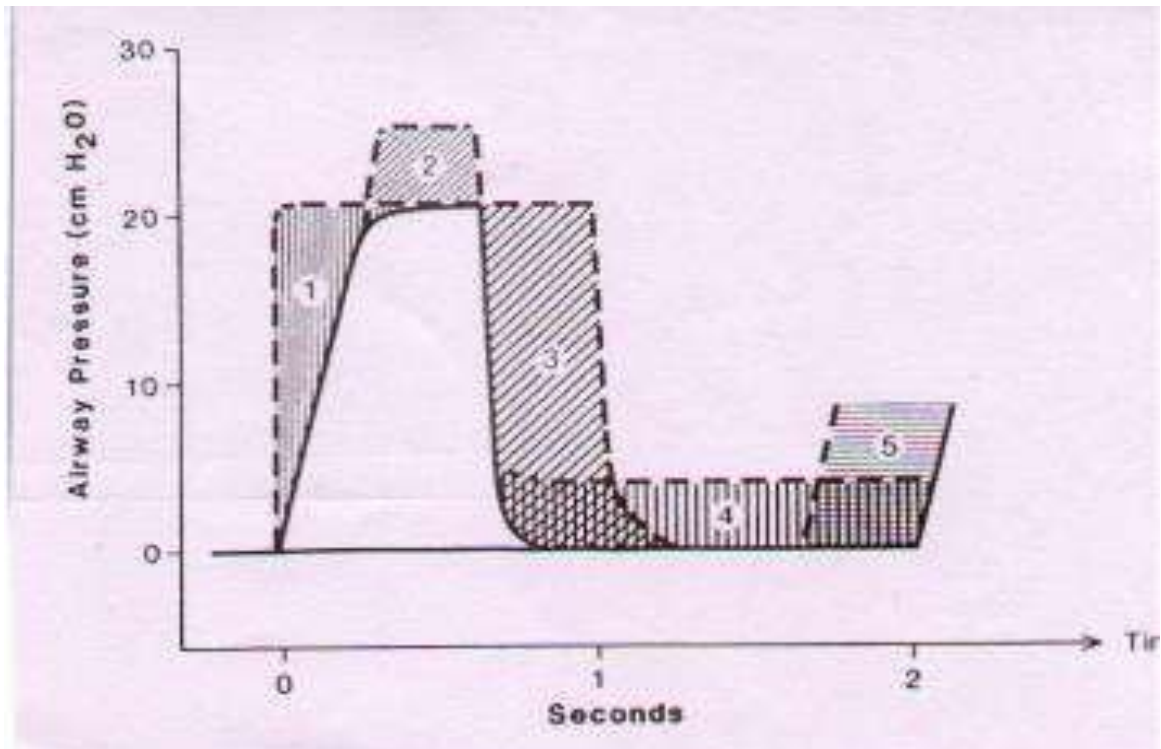
Inspiratory time(Ti)

I:E ratio:Ratio of inspiratory time and expiratory time in seconds

Frequency(f): Ventilatory rate(breaths/min)

Tidal volume(Vt): Amount of gas delivered with each breath
Expired Tidal volume (Ve): Amount of gas measured by the machine at expiration.
Expired Minute volume MV: Volume of gas in L expired per minute

Modes of ventilation

Praveen Khilnani

Control modes:
In this mode every breath is fully supported by the ventilator. In classic control modes, patients were unable to breathe except at the controlled set rate. In a conventional controlled mode, weaning is not possible by decreasing rate, the patient may hyperventilate if agitated leading to patient / ventilator asynchrony. Patients on control modes will need sedation and or paralysis with a muscle relaxant In newer control modes, machines may act in assist-control, with a minimum set rate and all triggered breaths above that rate are also fully supported.

IMV (Intermittent mandatory ventilation) modes:
In this mode breaths "above" the set rate are not supported. Most modern ventilators have synchronized intermittent mandatory ventilation(SIMV)

SIMV: Ventilator synchronizes IMV "breath" with patient's effort.
Patient takes "own" breaths in between (with or without pressure support) the set SIMV rate. There is a potential for increased work of breathing and patient / ventilator asynchrony, if the ventilator interferes with the patient's effort to breath or if there is insufficient flow for the spontaneous breaths. Ventilators would have an inbuilt latent period of about 25% of the inspiratory time in which to recognize the patient's effort in order to synchronise the mandatory breath in order to reduce asynchrony.
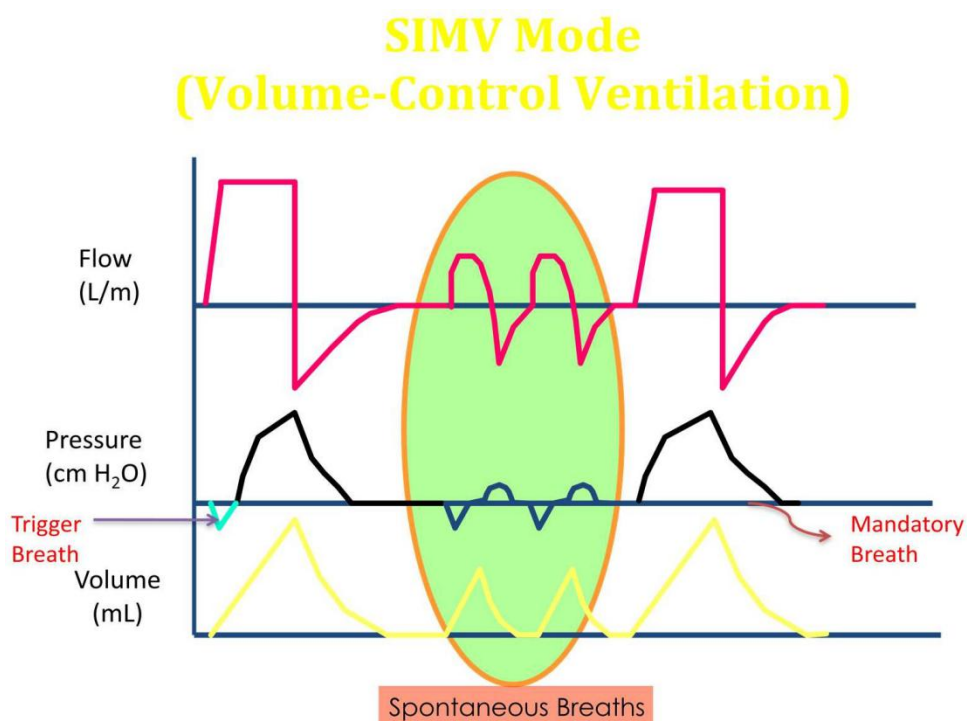
SIMV Mode
(Pressure Control Ventilation)

Flow (L/min)

Set PC level

Pressure (cm H₂O)

Volume (ml)

Time (sec)

Spontaneous Breath
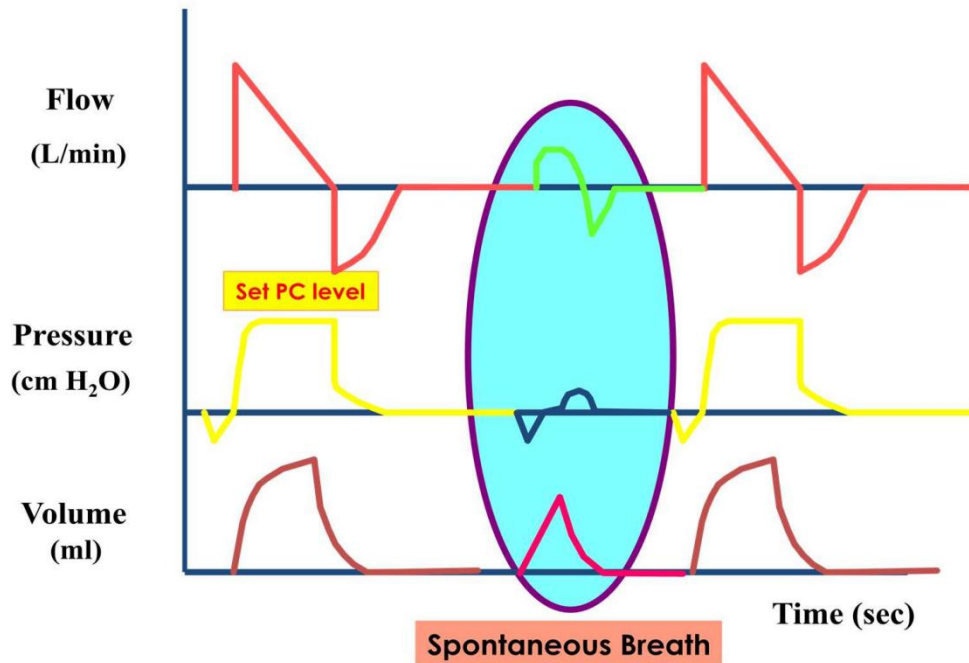
Support mode

Pressure Support Ventilation

Patient Triggered, Pressure Limited, Flow Cycled Mode

Flow (L/m)

Flow Cycling

Set PS level

Pressure (cm H₂O)

Volume (mL)

Time (sec)
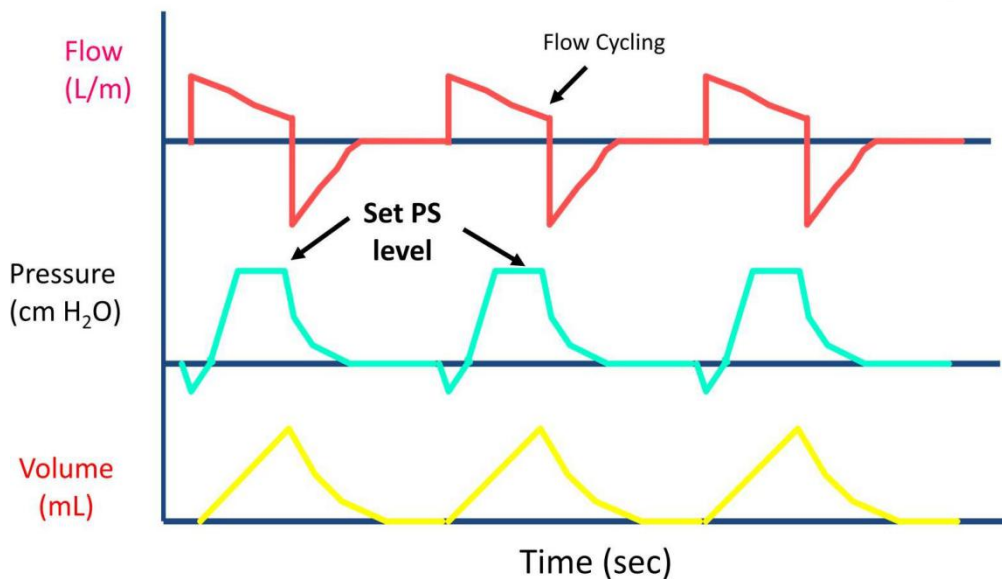
Pressure support

Ventilator supplies pressure support(flow) at a preset level but rate is determined by the patient, expiration begins passively when inspiratory flow decreases below a certain level preset in the ventilator (flow cycled); Volume support is also available in Servo 300 ventilators following the principle of pressure suport(delivery of the set volume over the patients natural inspiratory time duration keeping the pressure to a minimum.

Pressure support can decrease work of breathing by providing flow during inspiration for patient triggered breaths.It can be given with spontaneous breaths in IMV modes or as stand alone mode without set rate as well as for weaning to retrain coordination of respiratory muscles in patients on ventilation for longer than few weeks.

Trigger

Trigger is defined as the variable that initiates the breath from the ventilator.The trigger variable is usually pressure or flow.

Pressure trigger: With pressure triggering, in order to trigger the ventilator and initiate the inspiratory flow, the patient must decrease thepressure in the ventilator circuit to a preset value, which will then open a demand valve.

Flow trigger:With flow triggering, the patient triggers the ventilator when the respiratory muscles generate a certain preset inspiratory flow. It is generally believed that triggering of the ventilator is better with flow than with pressure.

The real clinical significance is unclear in terms of the work of breathing and patient ventilator interaction. Pressure sensors in current ventilators are much improved, reducing any difference between flow- and pressure triggering systems. Recent studies in patients with different diseases show that the difference in the work of breathing between flow and pressure triggering is of minimal clinical significance.

Trigger setting: A pressure trigger setting of greater than 0 (cm of water) makes it too sensitive(meaning the triggered breath from the ventilator will be too frequent).A negative setting(negative1 or negative 2) setting is usually acceptable.Too negative setting will increase the work of the patient(to generate a negative pressure)to trigger a ventilator breath.

Basic fundamentals of ventilation

Ventilators deliver gas to the lungs using positive pressure at a certain <u>rate</u>. The amount of gas delivered can be **<u>limited</u>** by time, pressure or volume. The duration can be **c <u>ycled</u>**by time, pressure or flow.If volume is set, pressure varies; if pressure is set, volume variesaccording to the compliance.

Compliance =Δ volume / Δ pressure

Chest must rise no matter which mode is chosen

Following are three main expectations from the ventilator:

1. Ventilator must recognize patient's respiratory efforts (trigger)
2. Ventilator must be able to meet patient's demands (response)
3. Ventilator must not interfere with patient's efforts (synchrony)

Whenever a breath is supported by the ventilator, regardless of the mode, the limit of the support is determined by a preset pressure or volume.
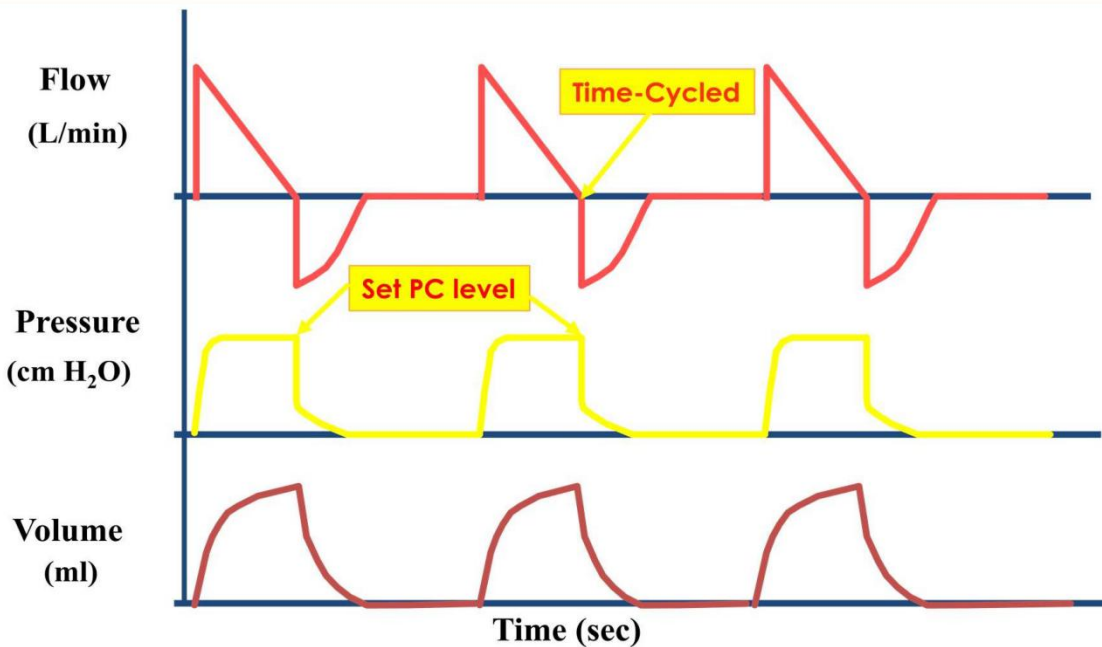
Volume limited: preset tidal volume

Pressure limited: preset PIP

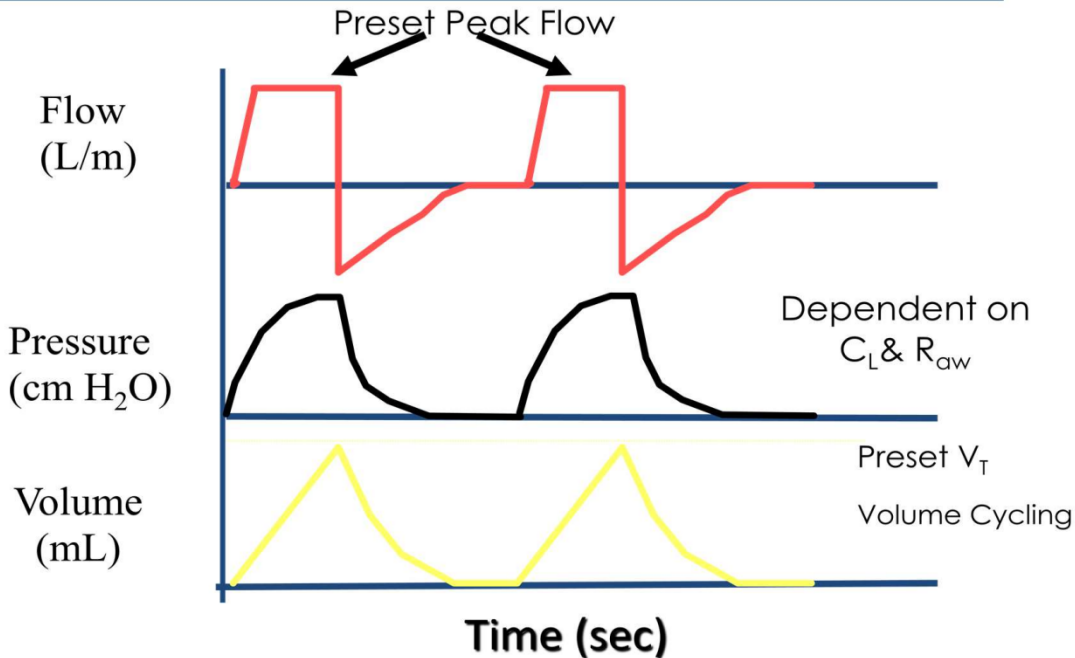Pressure   vs

Controlled Mode
(Pressure Control Ventilation)

Time Triggered, Pressure Limited, Time Cycled Ventilation

Flow (L/min)

Time-Cycled

Pressure (cm H₂O)

Set PC level

Volume (ml)

Time (sec)

Controlled Mode
(Volume Control Ventilation)

Time triggered, Volume limited, Time cycled Ventilation

Preset Peak Flow

Flow
(L/m)

Pressure
(cm H_2O)

Dependent on
C_L & R_{aw}

Volume
(mL)

Preset V_T
Volume Cycling

Time (sec)

Volume control

Goal is to ventilate and oxygenate adequately.Both pressure and volume control modes can achieve it.Important requirements include adequate movement of the chest, smooth gas flow and minimal barotrauma or volutrauma.

One must have a set up of high/low pressure alarms in volume cycling and, low expired tidal volume alarm when using pressure cycling.

Pressure limited ventilation
Ventilator stops the inspiratory cycle when set PIP is achieved.
Caution: Tidal volume changes suddenly as patient's compliance changes. Ventilator delivers a decelerating flow pattern (lower PIP for same Vt). This can lead to hypoventilation or overexpansion of the lung. If endotracheal tube is obstructed acutely, delivered tidal volume will decrease.This mode is useful if there is a leak around the endotracheal tube.

For improving oxygenation,one needs to control FiO2 and MAP,(I-time,PIP,PEEP)and to influence ventilation,one needs to control PIP and respiratory rate.

Volume limited ventilation

Ventilator stops the inspiratory cycle when set tidal volume has been delivered.One can control minute ventilation by changing the tidal volume and rate. For improving oxygenation primarily FiO2, PEEP, I-time can be manipulated. Increasing tidal volume will also increase the PIP, hence affecting the oxygenation by increasing the mean airway pressure.It delivers volume in a square wave flow pattern.Square wave (constant) flow pattern results in higher PIP for same tidal volume as compared to pressure modes.

**Caution:** There is no limit per se on PIP (so ventilator alarm will have to be set for an upper pressure limit to avoid barotrauma). Volume is lost if there is a circuit leak or significant leak around the endotrcheal tube,therefore an expired tidal volume needs to be monitored and set .Some ventilators will alarm automatically if the difference between set inspired tidal volume and expired tidal volume is significant(varies between the ventilators).

Initial ventilator settings

One should always have the general idea regarding what initial ventilatory settings to choose when initiating the ventilation.
Choose the mode: Control every breath (Assist control) if planed for heavy sedation and muscle relaxation or use SIMV when patient likely to breath spontaneously.

General Parameters to choose will include:

Rate: Start with a rate that is somewhat normal; i.e., 15 for adolescent/child, 20-30 for infant/small child, 30-40 for a neonate, 40-50 for a premature neonate.
FiO$_2$: 1(100%) and quickly wean down to level<0.5. Depending upon oxygen requirement 0.5 may be a starting point for the FiO2.
PEEP: 3-5 cm of H$_2$0 (higher to 6-7 if ARDS, or low compliance disease, lower (2-3cm) if Asthma, or high compliance disease.
Inspiratory time (I-time or I:E ratio): 0.3 to 0.4 sec for neonates, 0.5-0.6 sec for children,0.7-0.9 in older children. Normal I:E ratio=1:2-1:3

Then specifically choose if the modality of delivered breath will be pressure controlled or volume controlled (correct term is pressure limited or volume limited)

Pressure limited:
Peak inspiratory pressure (PIP) is set depending upon lung compliance and pathology
Neonates: Apnea 12-14cm, Hyaline membrane disease 18-22cm H$_2$O
Children: For normal lung 16-18cm, for low compliance 18-25cm H$_2$O, severe ARDS 25-35 cm may be required.
Volume limited
Tidal volume 8ml-10ml/kg with a goal to get 6-8 ml/kg expired tidal volume. Initial tidal volume at 10-12ml/kg may need to be set if leak present around endotracheal tube; in such

patients pressure limited ventilation may be preferred.Flow in most ventilators is set at 6-10 litres for the washout of the CO2 from the internal ventilator circuit, tubings etc. Flow less than 4L/min is not recommended. Following discussion includes cases and principles of ventilation based on disease specific pathophysiology.

Adjustments after Initiation:
Usually based on blood gases and oxygen saturations.
For oxygenation Fio2,PEEP,i Time, PIP (Tidal volume) can be adjusted(increase MAP)
For ventilation Respiratory rate,Tidal volume(In volume limited) and PIP(In presure limited mode) can be adjusted.

PEEP is used to help prevent alveolar collapse at end inspiration; it can also be used to recruit collapsed lung spaces or to stent open floppy airways

Gas exchange related problems

- **Inadequate oxygenation (Hypoxemia)**
- **Inadequate ventilation (Hypercarbia)**

Inadequate oxygenation

Important guidelines:

1. Don't just increase fiO_2
2. Increase tidal volume if volume limited mode, PEEP, Inspiratory time.
3. Increase peak inspiratory pressure (PIP)/PEEP/inspiratory time (ti) if pressure limited mode
4. If O2 worse, get chest xray to rule out air leak (treat!)/ if lung fields show worsening(increase PEEP further)
5. Do not forget other measures to improve oxygenation
       a. Normalize cardiac out put (if low output) by fluids and /inotropes
       b. Maintain normal hemoglobin
       c. Maintain normothermia
       d. Deepen sedation/consider neuromuscular block

High $paCO_2$
Common reasons include hypoventilation, deadspace ventilation (too high PEEP, decreased cadiac output, pulmonary vasoconstriction), increased CO2 production, hyperthermia, high carbohydrate diet, shivering. Inadequate tidal volume delivery(hypoventilation) will occur with endotracheal tube block,malposition, kink, circuit leak, ventilator malfunction.

Measures for normalizing high paCO2 guidelines:

1. If volume limited: Increase tidal volume (Vt), Increase frequency(rate)(f).

2. If asthma: Increase expiratory time, may need to decrease ratio to achieve an I:E ratio >1:3.
3. If pressure limited: Increase peak inspiratory pressure(PIP), decrease PEEP, increase frequency(rate).
4. Decrease dead space (increase cardiac output, decrease PEEP, vasodilator).
5. Decrease CO_2 production: Cool, increase sedation, decrease carbohydrate load.
6. Change endotracheal tube if blocked, kinked, malplaced or out, check proper placement.
7. Fix leaks in the circuit, endotracheal tube cuff, humidifier.

Measures to reduce barotrauma and volutrauma

Following concepts are being increasingly followed in most pediatric intensive care units.

1. **Permissive Hypercapnia:** Higher PaCO2s are acceptable in exchange for limiting peak airway pressures: as long as pH>7.2.
2. **Permissive Hypoxemia:**
PaO2 of 55-65; SaO2 88-90% is acceptable in exchange for limiting FiO2 (<.60) and PEEP, as long as there is no metabolic acidosis. Adequate oxygen content can be maintained by keeping hematocrit > 30%.

Patient ventilator dysynchrony:

Incoordination between the patient and the ventilator: Patient fights the ventilator!!Common causes include, Hypoventilation, hypoxemia, tube block/ kink/malposition, bronchospasm, pneumothorax, silent aspiration, increased oxygen demand/increased CO_2 production (in sepsis), inadequate sedation.

If patient fighting the ventilator and desaturating: Immediate measures
USE PNEMONIC : D O P E
D displacement,O obstruction,P pneumothorax,E equipment failure

1. Check tube placement. When in doubt take the endotracheal tube out, start manual ventilation with 100% oxygen.
2. Examine the patient: is the chest rising? Breath sounds present and equal? Changes in exam? Atelectasis, treat bronchospasm/tube block/malposition/pneumothorax? (Consider needle thoracentesis).
3. Examine circulation: shock? Sepsis?
4. Check arterial blood gas and chest x ray for worsening lung condition, and for confirming pneumothorax.
5. Examine the ventilator, ventilator circuit/humidifier/gas source.

If no other reason for hypoxemia: increase sedation/muscle relaxation, put back on ventilator.

Sedation and muscle relaxation during ventilation

Most patients can be managed by titration of sedation without muscle relaxation. Midazolam (0.1-0.2mg/kg/hr) and vecuronium drip (0.1-0.2mg/kg/hr) is most commonly used. Morphine or fentanyl drip can also be used if painful procedures are anticipated.
Do not muscle relaxants without adequate sedation.

Routine ventilator management protocol
Following protocol is commonly followed

1. Wean Fio2 for spO2 above 93-94.In ARDS 89-92 may be acceptable.
2. ABG one hour after intubation, then am pm schedule(12 hourly) ,and after major ventilator settings change, and 20min after extubation.
3. Pulse oximetry on all patients, End tidal carbondioxide (EtCO2) /graphics monitoring if available.
4. Frequent clinical examination for respiratory rate, breath sounds, retractions, color.
5. Chest x ray every day/alternate day /as needed.

Respiratory care protocol

1. Position changes every 2 hourly   right chest tilt/left chest tilt/supine position and try to maintain 30 degree head up position.

2. Suction 4 hourly and as needed (In line suction to avoid de recruitement/loss of PEEP/desaturation if available).
3. Physiotherapy 8 hourly: Percussion, vibration and postural drainage. NO physiotherapy if labile oxygenation such as ARDS (Acute respiratory distress syndrome), PPHN (persistent pulmonary hypertension of neonate).
4. Nebulization: In line nebulization is preferred over manual bagging. Metered dose inhalers (MDIs) can also be used.
5. Disposable Circuit change if visible soiling.
6. Humidification/In line disposable humidifier.

Ventilator care protocols, suctioning , physiotherapy ,positioning should all be under proper protocols for patient safety and to prevent adverse events such as unplanned intubation.

Weaning from mechanical ventilation

Process of weaning begins at the time of initiation of ventilation (i.e. minimal ventilatory settings to keep blood gases and clinical parameters within acceptable limits although these settings will be very high).

If such procedure is followed then ventilatory settings would be reduced once the primary pathology/condition that led to ventilation is improving.

How do we know if the condition is improving ?

- Improving general condition,fever etc.
- Decreasing FiO2 requirement
- Improving breath sounds
- Decreasing endotracheal secretions
- Improving chest X rays
- Decreased chest tube drainage,bleeding/air bubbles(as the case may be)
- Improved fluid and electrolyte status(no overload or dyselectrolytemia)
- Improving hemodynamic status
- Improving neurological status,muscle power,airway reflexes/control.Described weaning criteria such as maximal negative inspiratory force, vital capacity measurement are usually impractical. In pediatrics and neonatal age group weaning criteria are generally clinical.

Key messages

1. Remember shock and post resuscitation are important indications for ventilation,in addition to respiratory failure and neuromuscular disease.
2. Clinical monitoring of adequate chest rise and oxygen saturations is very important. (Regardless of mode volume, pressure or time cycled mode).
3. If ventilator fails, turn FiO2 to 1.(100%) and take over hand bag tube ventilation : Follow DOPE protocol and correct accordingly.
4. If and when in doubt regarding endotrachel tube status, don't waste time :Remove endotracheal tube and try bag mask ventilation.
5. Low tidal volume is recommended to prevent lung trauma (permissive hypercapnia and permissive hypoxemia).
6. Ventilator care protocols, suctioning, physiotherapy, positioning should all be under proper protocols for patient safety and to prevent adverse events such as unplanned intubation.
7. With or without weaning protocols, most pediatric patients can be extubated successfully.
8. Spontaneous breathing trial and clinical indicators for extubation readiness may be used in difficult situations of extubation failure, however none of the pediatric specific weaning protocols and guidelines are able to predict successful extubation.

Advanced modes and modalities of ventilation
Praveen Khilnani

1. Inverse Ratio Ventilation (IRV)

2. Airway Pressure Release Ventilation (APRV)

3. Pressure Support Ventilation (PSV)

4. Pressure-Regulated Volume Control (PRVC)

5. Proportional Assist Ventilation (PAV)

Introduction

Ventilatory strategies in the past evolved from surgical and anesthetic practice. In the immediate postoperative period, tidal volumes of 10 and 15 ml/kg ("sigh volumes") were used to prevent the microatelectasis that accompanied shallow or inadequate ventilation. In addition, blood gases were "normalized". It has become more apparent that this may place the injured lung at risk of further damage. Therefore, even more important than the technique or the machine, is the need to develop a strategy of treatment based on the pathophysiology and keeping with principles of lung protection.

Use of newer modes and different modalities of ventilation is rapidly evolving using a combination of following strategies in ventilation.

• Using decreased tidal volumes (Vt) less than 7 ml/kg.
• Decreasing peak inspiratory pressure (PIP).
• Increasing inspiratory time (T insp).
• Using increased mean airway pressure.
• Permissive Hypercarbia and Hypoxemia.
• Guaranteed tidal volume at lowest PIP.
• Using very small volumes at high frequency
• Non invasive ventilation

1. Inverse Ratio Ventilation (IRV)

• ***Strategy:*** Decrease PIP increase (T insp) increase mean airway pressure.

• Inverse ratio ventilation is a technique first popularized by Reynolds in England, who applied this method to newborn with hyaline membrane disease. He reported better oxygenation and less barotrauma. This method became the forerunner of pressure-limited ventilation. Basically, it is controlled by positive-pressure ventilation with an inspiratory time ratio >1:1 or a duty cycle of >0.5. Duty cycle (Ti/Ttot) is the ratio of the inspiratory time (Ti) to the total breath cycle duration (T tot).

Inverse ratio ventilation can be established by any of the following methods:

a. Timed-cycled, volume-cycled, or pressure controlled ventilation with the inspiratory time (Ti) adjusted to the desired level,

b. Pressure-limited ventilation (either timed or volume-cycled ventilators) with a prolonged inspiratory time, or

c. Volume cycling with a prolonged inspiratory hold.

Improved ventilation reported with this technique probably results from increased duration of the inspiratory phase. This allows areas with longer time constants to receive greater flow than would otherwise occur with conventional volume-controlled ventilation with the usual I:E ratio. Improvement in gas exchange during inverse ratio ventilation is attributed to more effective alveolar recruitment and better distribution of ventilation during the extended inspiratory phase, thus decreasing deadspace ventilation.

The short expiratory time invariably induces intrinsic PEEP. Peak airway pressure (is lower than that with continuous positive-pressure) and increased functional residual capacity are responsible for the improvement in oxygenation.

Inverse ratio ventilation imposes a nonphysiologic breathing pattern and requires sedation or paralysis. There is often little effect on cardiac output at I:E ratio of < 4:1 in the volume repleted individual, but the occurrence of pneumothoraces may be as high as 25 percent. This may correlate more with length of time on inverse ratio ventilation than the I:E ratio or the mean airway pressure.

Inverse ratio ventilation is often used in patients with respiratory distress syndrome with refractory hypoxemia (PaO < – 60 mm with an FiO, > 80%) despite the use of PEEP > I 5 cm H_2O with CPPV. There are no controlled studies to show that inverse ratio ventilation reduces morbidity and mortality rates. In fact the beneficial effect is inconsistent and the exact role played by changing the 1:E ratio, as opposed to alterations in mean airway pressure or the incorporation of a decelerating inspiratory flow, is difficult to ascertain. It is possible that it is the absolute duration of inspiration that is the most significant feature in the apparent success of inversed 1:E ratio ventilation. Recently, East *et al,* reported on their success with the use of a computerized protocol for pressure control inverse ratio ventilation in patients with acute respiratory distress syndrome (ARDS). Their goal was to provide the highest mean airway pressure at the lowest PIP. They used FiO_2, and intrinsic PEEP values to determine oxygenation. The computerized protocol followed a specific algorithm for ventilator manipulations based on very appropriate physiologic strategies. They attained their goals with no apparent increase in barotrauma. Perhaps more importantly, it proved the feasibility of this protocol to allow for a more systematic approach.

2. Airway Pressure Release Ventilation (APRV)

*Strategy*: Decreased PIP increased (T insp) mean airway pressure.

Airway pressure release ventilation is a form of continuous positive airway pressure (CPAP) that releases the airway pressure from one preset CPAP level to a lower CPAP level. This allows a spontaneously breathing patient to exhale to a lower lung volume. The unique feature of airway pressure release ventilation is the augmentation of alveolar ventilation by a decrease in airway pressure and lung volume.

Preset variables are the initial CPAP level, the frequency of airway pressure releases (similar in concept, to intermittent ventilation (IMV), the level to which CPAP reduces during release, duration of the airway pressure release. The duration of the expiratory release (expiratory time) is usually < 1.5 sec. Peak airway pressure during airway release ventilation is 30 to75 percent that during CPPV and may explain the finding that it has less of an effect hemodynamics than does CPPV. Peak airway pressure never, exceeds the CPAP level. Since airway pressure during airway release ventilation never exceeds the CPAP level, it may be that inverse ratio ventilation which reduces morbidity, clinically feasible to commence airway pressure release ventilation in a patient receiving mask CPAP therapy without the need for tracheal intubation.

Weaning occurs by lowering the frequency airway pressure release, until the patient is breathing with CPAP alone. From a synchronization standpoint, augmentation of ventilation during airway pressure release ventilation is similar to conventional IMV. Airway pressure release ventilation has not been shown to significantly improve oxygenation in studies of humans with acute lung injury. It appears more effective in improving alveolar ventilation than oxygenation. It results in a lower $PaCO_2$ value than conventional ventilation at same minute ventilation.

The rationale for the development of airway pressure release ventilation is similar to inverse ratio ventilation, i.e. to open, maintain and stabilize the collapsed alveoli without peak airway pressure and the hazard of barotrauma. Thus, the initial CPAP level (or insulation pressure in inverse ratio ventilation) is responsible for stabilization of lung units and the expiratory phase is responsible for ventilation. Although the pressure tracings of inverse ratio ventilation with pressure limit closely mimic airway pressure release ventilation. The former usually has higher peak and mean airway pressures and doesn't allow for spontaneous breathing. In contrast to inverse ratio ventilation, airway pressure release ventilation allows the patient to breathe spontaneously between ventilator breaths, without requiring sedation and paralysis. In addition, the increase in functional residual capacity (FRC) in inverse ratio ventilation depends more on intrinsic PEEP because of higher closing volumes in children and CPAP levels that are often below critical opening pressure of airways, it is doubtful that airway pressure release ventilation will be useful for prolonged ventilation in children with acute lung disease. Further studies would be necessary as the indications for its use remain unclear.

Airway pressure release ventilation APRV
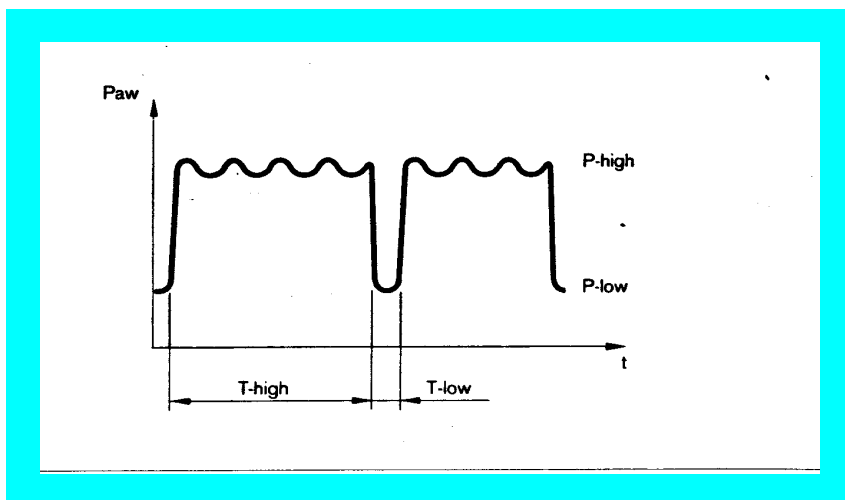
3. Pressure Support Ventilation (PSV)

Strategy: Decreased work of breathing.

This is a patient-triggered, pressure-limited mode of positive pressure ventilation that delivers a preset positive pressure during the inspiratory phase. Commonly, a pressure plateau is maintained until the patient's inspiratory flow decreases to a specified level at which time exhalation occurs. Unlike pressure limited ventilation, during pressure support the patient determines the initiation of inspiration, inspiratory times, flows and tidal volume, and the termination of inspiratory phase. The patients effort, the preset pressure limit, and the respiratory system impedance determines tidal volume (Vt). Inspiratory time and Vt can vary on a breath-to-breath basis.
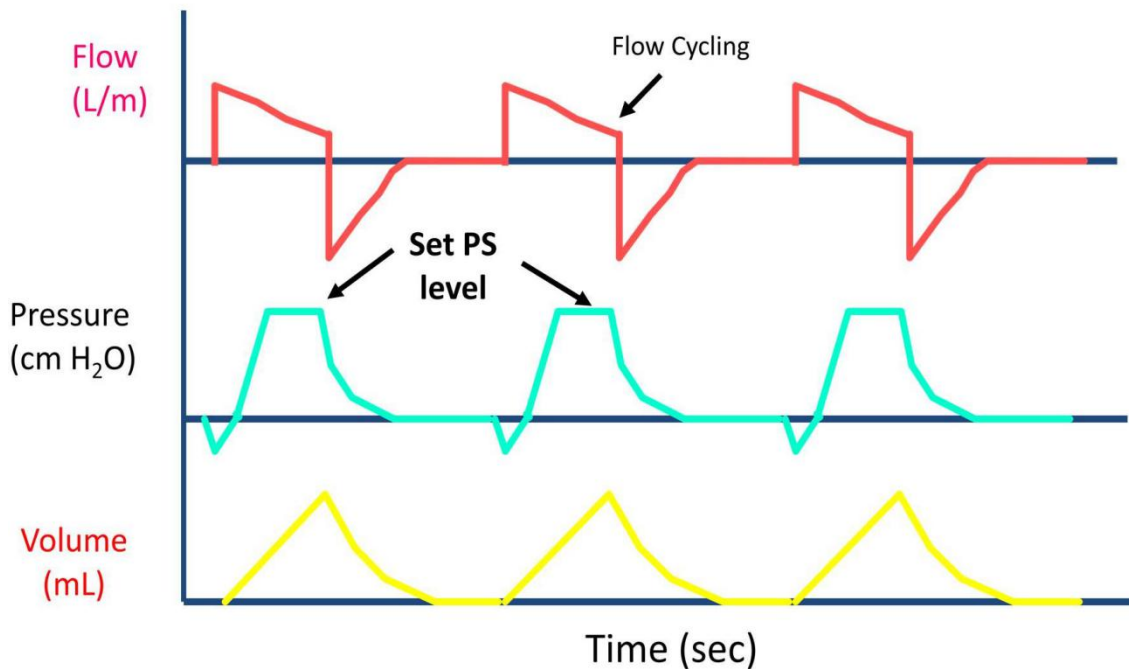
Pressure support ventilation is reported to have many benefits. Most agree that it does compensate for the increased work of breathing due to the endotracheal tube and the ventilator demand valve. Improved synchrony between the patient and machine may help to explain the increased sense of comfort that pressure support ventilation provides. The most important physiologic aspect of pressure support ventilation may be its ability to better match the patient's inspiratory flow demands, thereby minimizing respiratory muscle effort as compared with other forms of mechanical ventilation. Pressure support ventilation should allow patients with mechanical impairment to ventilation to acquire larger inspiratory tidal volumes at the same level of effort or the same level of Vt at a lower level of inspiratory work.

Other benefits of pressure support ventilation include the reduction in the activity of the inspiratory muscles during spontaneous breathing. Values of 20 cm H_2O of pressure support ventilation abolish the pattern of electromyographic activity indicative of muscle fatigue. It may decrease oxygen consumption by the respiratory muscles and improve respiratory muscle loading while decreasing the respiratory muscle work per liter of ventilation as well as total muscular work per minute. Decreased intrinsic PEEP, elastic work of breathing, and oxygen cost of breathing have also been reported. Although not documented, some feel that it improves endurance conditioning of the respiratory muscles.

Proponents suggest initial ventilation at maximum pressure support ventilation. This is the

Pressure Support Ventilation

Patient Triggered, Pressure Limited, Flow Cycled Mode

level of pressure support required to produce a Vt of 10 to 12 ml/kg, and corresponds to the level where inspiratory work is least. Gradually, the pressure support ventilation level is decreased to < 10 cm H_2O (above PEEP), when extubation can be performed.

Since each breath must be initiated by the patient. Pressure support ventilation should be used with caution in patients with an unstable respiratory drive or highly changeable respiratory impedance (e.g., a patient with reactive airway disease). In the latter case, as with all forms of pressure-limited ventilation, as the patient's impedance changes, delivered VT may be affected. Synchronized mandatory ventilation (SIMV) and pressure support ventilation may be preferable when one requires complete mechanical support.

Pressure Regulated Volume Control (PRVC)
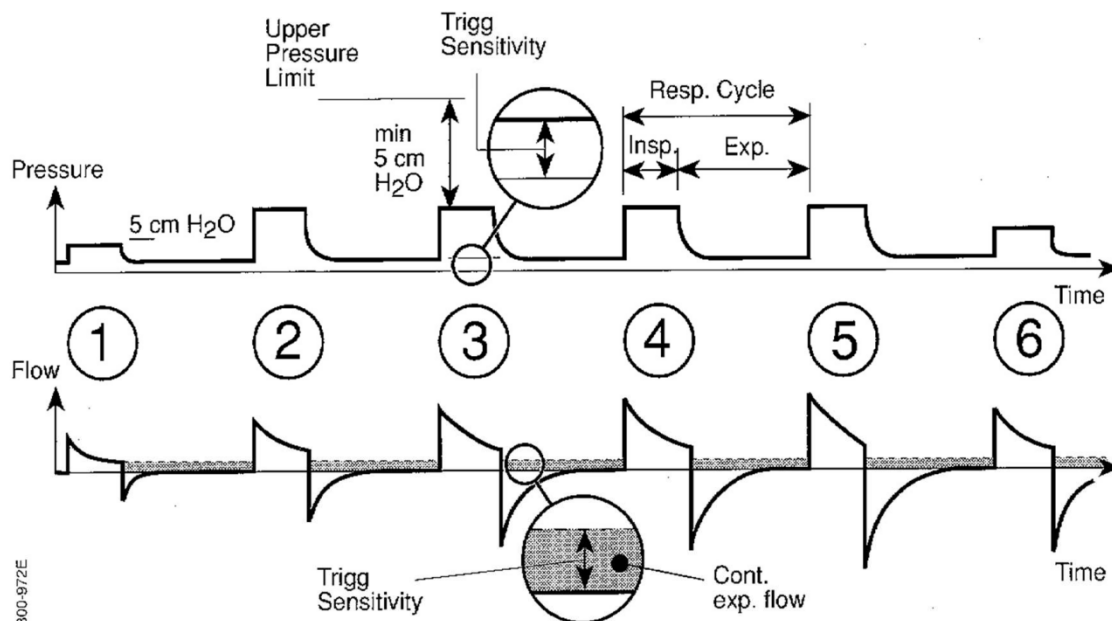
■ From Siemens prod. literature

4. Pressure-Regulated Volume Control (PRVC)

Strategy: Guaranteed Vt at lowest PIP and decreased work of breathing.

This is the new mode of controlled ventilation found on many ventilators (Servo300, Servo i). It uses a decelerating flow generator (as used in the Pressure Control mode on the 900C ventilator) as a pressure generator (pressure remains constant). The ventilator evaluates the exhaled volume on a breath-by-breath basis and will reset the pressure support level as needed to guarantee the Vt. The flow pattern often decreases the PIP (by approximately 5 to 7 cm H_2O) when compared with the same volume delivered in a volume control (constant flow) mode. Mean airway pressures may increase, averaging about 1 cm H_2O. Therefore, it overcomes the limitation of pressure support ventilation and mandatory minute ventilation by assuring a constant Vt, even with a lung with changing pulmonary mechanics.

5. Proportional Assist Ventilation (PAV)

Strategy: Decrease PIP and decrease work of breathing, increase volume of spontaneous breaths.

First described by Younes as a method to alter the mechanical load of the respiratory system, this method of positive pressure ventilation holds great promise for those patients with adequate respiratory drive. Essentially, the ventilator changes pressure at the airway in proportion to the inspired volume (elastic assist), the inspired flow (resistive assist), or both inspired volume and flow. The delivered volume or pressure varies according to the feedback

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signal the ventilator receives proportionate to the patient's elastic and/or resistive load. Thus, the ventilator simply reduces the total respiratory load and leaves the patient in total control of the breathing pattern. Conceptually, it is similar to the use of helium to decrease the respiratory load, or an intra-aortic balloon pump to decrease the load on the left ventricle. It also approximates what an individual may do while "hand bagging" a spontaneously breathing patient. As the patient takes a greater inspiration, the person bagging will squeeze the bag harder, i.e. the volume delivered will be proportional to the patient's inspiratory effort.

Newer techniques (such as NAVA: Neurally Adjusted Ventilatory Assistance) to reduce patient ventilator asynchrony, though promising, need further clinical trials to establish as a routine technology in Neonatal and Pediatric critical care units.

Neonatal Noninvasive Ventilation (CPAP/NIMV)
Kumar Ankur, Sanjeev Chetry

Continuous Positive Airway Pressure (CPAP) is positive pressure applied to the airways of a spontaneously breathing baby throughout the respiratory cycle. Advocates of non-invasive ventilation cite the reduced risk of trauma to the larynx and trachea, infection and acute and chronic lung disease with this form of support. The rationale for use of CPAP is to support the airways and avoid alveolar collapse to a level below FRC.

How does CPAP work?
CPAP supports the breathing of premature infants in a number of ways. The upper airway of the preterm infant is very compliant and therefore prone to collapse. CPAP splints the upper airway and therefore reduces obstruction and apnea. CPAP assists expansion of the lungs and prevents alveolar collapse. In doing so it reduces protein leak and conserves surfactant.
Physiology & how CPAP helps
Preterm: Inability to maintainfunctional residual capacity (FRC) due to various reasons:

- Not able to generate enough negative pressure to achieve an effective FRC
- Has low laryngeal tone to maintain PEEP by grunting
- Fluid clearance from lung depends on gestational age of baby because of immature amiloride sensitive Na channel
- Lack of fat laden superficial fascia in the neck   which stabilise the airway
- Not able to mobilise the genioglossus muscle effectively which normally stabilises the pharynx.
- Insufficient numbers of alveolar channels for collateral ventilation.
- Chest wall is soft and horizontal ribs are flatter reducing the potential for lung expansion.
- During REMsleep, intercostal muscle activity may be lost.
- PDA predisposing to pulmonary oedema.
- Deficient surfactant.

Effects of CPAP in the infant with respiratory distress:
- Reduces upper airway occlusion by decreasing upper airway resistance and increasing the pharyngeal cross sectional area.
- Increases the FRC.
- Reduces inspiratory resistance by dilating the airways. This permits a larger tidal volume for a given pressure, reducing the work of breathing.
- Increases the compliance and tidal volume of stiff lungs with a low FRC by stabilizing the chest wall and counteracting the paradoxical movements.
- Regularizes and slows the respiratory rate.
- Reduces the incidence of apnea.
- Increases the mean airway pressure and improves ventilation perfusion mismatch.
- By diminishing alveolar edema conserves surfactant on the alveolar surface**.**

Components of CPAP system

| Table 2	
Components of a CPAP system	
Factor	**Relevance**
Pressure-generating device	Constant vs variable pressure influencing potential for gas exchange and airway recruitment Constant vs variable flow influencing work of breathing
Heated and humidified circuit	Delivery of appropriate gas energy and saturation content at high flow to avoid mucosal injury and avoidance of condensation
Blended gas source	Avoidance of hyperoxia/hypoxia
Patient interface	Influences ease of application, rebreathing, extrinsic work of breathing, and potential for local injury
Safety pressure release and alarm	Avoidance of overpressurization with obstruction of expiratory tubing Alert carer to potential deprivation of fresh gas associated with inspiratory limb obstruction

CPAP delivery

CPAP interfaces:

Nasal CPAP devices fall into four groups:

1. **A long nasopharyngeal tube**
2. **A single nasal prong**
3. **Nose masks**
4. **Short bi-nasal prongs**

Short binasal prongs are more effective at preventing reintubation than single nasal prongs.
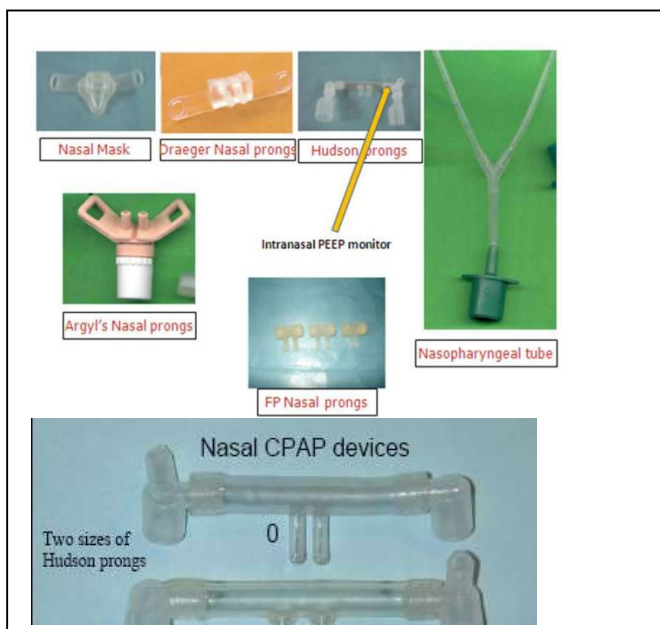

Different types of Nasal interfaces.

The way CPAP pressure is generated is distinct from the interface device. There are four techniques for generating nasal CPAP.

1. Bubble CPAP:With this technique gas flows past the nasal device and the pressure is generated in the circuit by placing the distal limb of the CPAP circuit under a known depth of water. Gas flow is increased until continuous bubbling is achieved.

Expiratory limb in water column
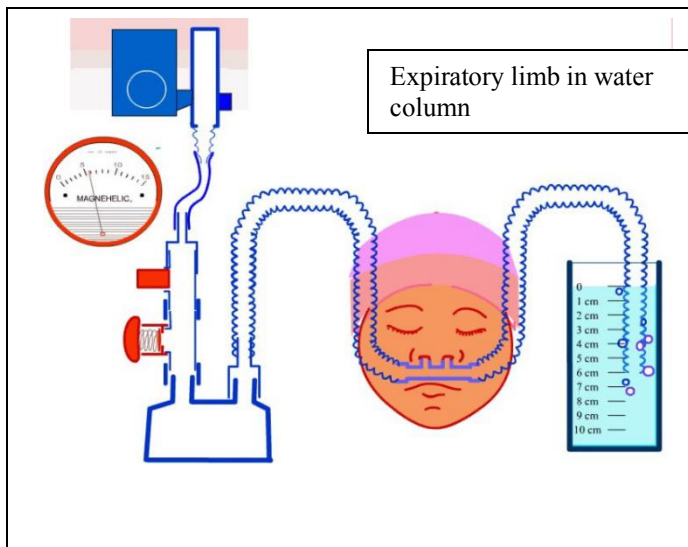
Buuble NCPAP

2. Ventilator CPAP: The ventilator PEEP (end expiratory pressure) valve controls the CPAP delivered.
3. Variable flow nCPAP devices: These devices have an integrated nasal interface and pressure generator. They use a higher gas flow than other devices and pressure is generated by increased resistance as the gas leaves the nasal device. The pressure is determined by altering the flow of gas into the device.
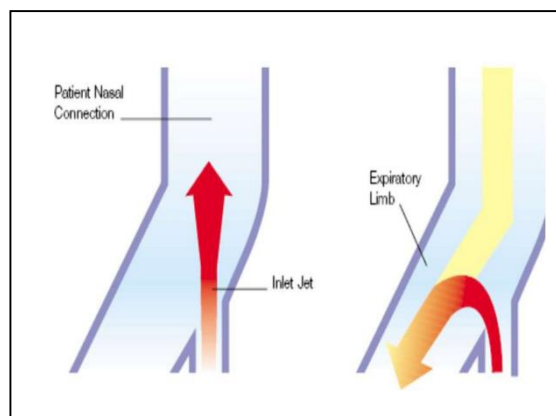
VARIABLE FLOW/fluidic flip

4. High flow nasal cannulae: High flow cannulae deliver gas flows >2 L/min into the nostrils through small prongs which are loose in the nostrils. Low flow cannulae are used to deliver supplemental oxygen whereas high flow cannulae are used because it is thought that they provide some CPAP.

Problems of nCPAP:
1. Leak at the nose and mouth: It has been reported that the pharyngeal pressure drops markedly when the CPAP supported infant opens his mouth. The use of chin strap or pacifier is recommended to reduce mouth leak for effective CPAP support. However, it should not be so tight as to prevent the infant from yawning or crying but tight enough to prevent leaking at rest
2. Nasal trauma: It is mostly caused by incorrect positioning of the prongs. To prevent injury the nasal device must not be pushed up against the columella. Injury can also **occur inside the** nose and erode the nasal septum if the prongs are not positioned straight into the nostrils.
3. Gastric distension: Gastric distension is common in the CPAP supported infant (CPAP Belly Syndrome). Frequent decompression of the stomach with an oro-gastric tube is necessary to promote comfort, preventing the distended stomach from splinting the diaphragm and compromising respiration.

How to prevent nasal septal injury:
Nasal septal Injury is absolutely preventable by checking following things:
- Snugly fitting nasal prongs
- Secure caps
- Light weight interface
- Correct positioning and attachment of corrugated tubing(bubble)
- Velcro moustache (Hudson prong ,bubble CPAP)
- Careful, frequent observation: give rest
- Careful positioning of the infant

Indications for nCPAP:
1. Delivery room NCPAP
2. Respiratory distress syndrome
3. Prophylactic CPAP for very preterm
4. Post-extubation

Nasal intermittent positive pressure ventilation (NIPPV)
Any mode of assisted ventilation that delivers positive pressure throughout the respiratory cycle with additional phasic increases in airway pressure, without the presence of an endotracheal tube. These additional phasic increases in airway pressure may either synchronized or nonsynchronized depending on the delivery system used. NIPPV provides the benefits of CPAP with the addition of positive pressure breaths.

How does NIPPV work?

The mechanism of action of NIPPV remains uncertain. It is unclear whether mechanical inflations during NIPPV are transmittedto the lungs. Moretti showed that some synchronised breaths were transmitted, but asynchronous breaths were not.

Delivery of NIPPV

Devices to generate NIPPV:Ventilators can be used to deliver nonsynchronised NIMV. The only specialised devices which attempt to provide synchronised NIPPV are the Infant Flow SiPAP and Infant Infant Flow Advance – IFDa (Viasys Healthcare, Conshohocken, PA, USA).

Nasal interface: Mask or Prong

How to start NIMV:Ventilator settings

- Changed from CPAP mode to IMV mode
- PEEP: 5 or more as determined by lung disease
- PIP: 2– 4 cm H2O higher than pre-extubation PIP 'to see the chest rise'or specific target pressures (16–20 cm H2O)
- Rate: 35 to 50 /min
- I time : according to diseased lung
- Monitor work of breathing, apnea, SPO2, PcO2(if required), CXR

However, the delivered pressure may be lower than the set pressure due to leak at the nose and mouth.

Indications:

- o RDS as a primary treatment
- o Apnea of prematurity
- o Post extubation

Criteria to discontinue NIMV:

- o Blood gas: if pH < 7.2 & Pco2 > 65 mm
- o Inability to improve gas exchange or increased work of breathing
- o Hemodynamic instabilit

Heated Humidified High flow nasal cannulae (HHHFNC)

Background

High flow nasal cannulae (HFNC) are small, thin, tapered cannulae (usually less than 1 cm in length) that sit just inside the nostrils without occluding them[10], and are used to deliver oxygen or blended oxygen and air at flow rates of > 1 L/min. HFNC can be used to provide high concentrations of oxygen and may deliver positive end-expiratory pressure. Oxygen delivered by 'low flow' nasal cannulae (LFNC) typically refers to the use of flow rates of less than or equal to 1 L/minute. It uses unblended, unheated and non-humidified gas (that is 100% oxygen). LFNC are commonly used in convalescing preterm infants, often with chronic lung disease[11], and does not provide significant support to the infant's pulmonary function (apart from the provision of oxygen). In contrast, 'high flow' nasal cannulae (HFNC) have been used to for administration of oxygen or blended oxygen and air to newborn infants via nasal cannulae at higher flow rates. The use of high flow rates in preterm infants may provide positive end-expiratory pressure (PEEP)[12,13]. In HFNC systems, circuit flow is adjusted according to clinical effect and, although a pressure relief valve is often used, the circuit pressure is not routinely measured.

Advantages

1. HHF provides a warmed and humidified flow of air and/or air-oxygen mixture (via a blender) to the infant where FiO2 can be monitored.
2. There is some degree of end distending pressure involved in HHF.
3. HHF may be better tolerated by infants becoming unsettled with HCPAP.
4. Reduced gastric distension[14].
5. Sucking feeds and Kangaroo care are more easily attempted with HHF than HCPAP.

Indications

HHF is utilised in NICU for infants with mild respiratory dysfunction. Main indications include

1. Neonates with bronchopulmonary dysplasia (ability to wean flow over FiO2).
2. Neonates who are CPAP (6cmH2O) dependent
3. Neonates with an FiO2 requirement of <0.3
4. Neonates 34-36 weeks corrected gestational age
5. Neonates not deemed stable enough to be trialled self ventilating in Air
6. Respiratory support post extubation
7. Apnea of prematurity
8. Post-op respiratory support.
9. Babies with nasal trauma from NCPAP

Contraindications

1. Blocked nasal passage (choanal atresia)
2. Trauma/ surgery to nasopharynx

Setting up of HHHFNC

1. Select appropriate size optiflow nasal cannula. (fig 1)
2. Assemble humidifier, base, filter, temperature probe, oxygen tubing and oxygen analyzer and RT330 set.
3. Prepare the optiflow nasal cannula for application.
4. The swivel connector of the Optiflow Nasal Cannula may be connected (clicked into) to the blue tubing before application. This allows humidified high flow oxygen/air to be given while the cannula is being applied.
5. Remove the paper protector from the nasal aspect of the wiggle pads (base tape).
6. Ensure baby's skin is dry and check septum's integrity.
7. Insert one prong into each nostril so that the bridge is just touching the septum.
8. Holding the cannula/wiggle pad wings, apply slight tension so as to straighten the bridge of the cannula then fix the nasal aspects of the wiggle pads onto the baby's cheeks.
9. On one side lift the outer edge of the wing and holding the paper tab remove the paper and press the wiggle pad into place. Do the same on the other side. (Fig 2)
10. On one side lift the outer edge of the wing and holding the paper tab remove the paper and press the wiggle pad into place. Do the same on the other side.
11. Once the cannula is applied check that the bridge has naturally moved 2mm away from the septum.
12. Be sure that the baby, particularly baby's ears are not lying on the Optiflow Cannula's coil tubing.
13. Change the cannula and circuit every 7 days.

Recommended Settings

• Prongs: Must be smaller than 50% of patient's nares (tight fit of nasal cannulae may generate pressure of 6-10cm H2O at flow as low as 1,5-2 L/min)
• Flow 4-8 L/min (lower flow 5-6 L/min may be sufficient for smaller babies.)
• Fio2 <40%
• Operating temperature set at 34 - 35º C for flow rate < 5 L/min and 36 - 38º C at > 5 L/min (to prevent condensation- manufacturer's recommendation)
• Use appropriately sized nasal cannula. The following is a guide but the diameters of nares may vary.

Weight	Cannula type	Outer diameter
< 1.4 Kg	Premature	0.14 cm
1.4 to 2.6 Kg	Neonatal	0.19 cm
> 2.6 Kg	Infant	0.27 cm

Maintenance of HHFNC

1. Do not use a chin strap with High flow. Active mouth closure is not required.

2. Watch for "rain-out" as this can cause a lavage to the infant resulting in apnea.

3. The baby may be nursed prone, skin to skin (kangaroo cuddle), supine or side to side lying.

4. All infants on high flow should have a nasogastric tube in place

5. Infants may be offered breast or bottle feeds whilst on HHF.

Monitoring of a baby on HHFNC

Continuous monitoring of Temperature, heart rate, respiratory rate and SaO2

Nasal and/or oro-pharyngeal suction may be necessary if baby has a lot of secretions.

Blood gases if on supplemental oxygen or on clinical grounds

Weaning

It may not be possible to wean flow rate if FiO2 > 0.3

Attempt to reduce by 1 L/min 24 hourly if FiO2 < 0.25-0.3 in babies >1.5Kg

Attempt to reduce by 0.5L/min 12 hourly if FiO2 < 0.25-0.3 in babies < 1.5 Kg

Attempt to stop if requiring 2.0 L/min or less

Clinical instability, increased work of breathing or significant increase in FiO2 consider pneumothorax

Simplified weaning

• 1 L/min every 24h if FiO2 < 30%

• 1 L/min every 12h if FiO2 = 21%

If cycling of HHFNC and CPAP is being utilised:

- HHF should be administered during the day allowing for increased parental interaction and sucking feed attempts. HCPAP should continue at night time.

- HCPAP and HHF have different tubing and pressure relief valves (HCPAP white/HHF blue), so the entire system needs to be alternated.

- The same humidifier base may be utilized.

Failure of HHFNC

If the baby is requiring FiO2 > 0.5 or has CO2 retention, acidosis or apnoea s/he is likely to need alternative support.

Complications

1. Potential for asynchrony in breathing may result in the infant becoming tired over long periods; therefore, good assessment of work of breathing is required.

2. Potential for nasal erosion (although less than with HCPAP) remains.

3. There is some concern about unknown end distending pressure and varied results gained in research studies; therefore ensure that the prongs do not seal the nares and reduce flow as able.

4. "Rainout" in circuit resulting in lavage and apnoea. Use designated circuit (RT330) and check for "rainout" regularly, draining circuit as required


Fig 1

Fig 2

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Neonatal Patient triggered ventilation (PTV)

Kumar Ankur, Avneet Kaur

Before we move to patient trigger ventilation and various trigger mechanisms, we should know critical events during a mechanical breath:

A. What initiates the breath?
B. What limits the breath?
C. What cycles the breath?
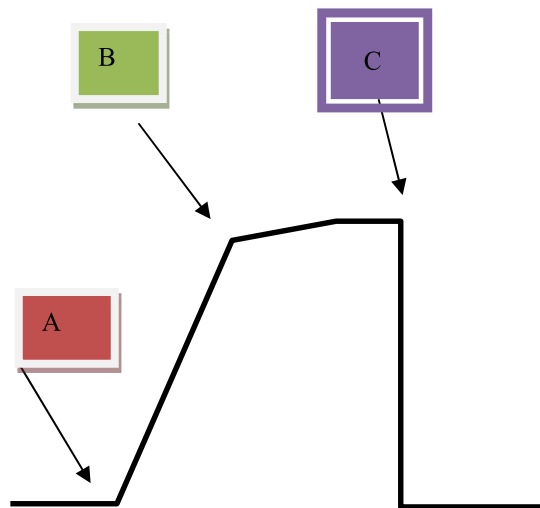
A In convention intermittent mandatory ventilation (IMV), breath is initiated once a preset time (as decided by set ventilatory rate) has elapsed. This is called time triggering. In more sophisticated ventilators, change in flow or pressure in the ventilatory circuit or realization of abdominal movements, as induced by patient's inspiratory effort is detected by ventilator and a breath is initiated. This is called patient triggering.

B In conventional ventilation 2nd point i.e. limit is pressure, that is the breath is limited to a point when preset pressure is achieved. This is called pressure-limited ventilation. More recently, volume guarantee ventilation is being used increasingly, in which breath is limited, once preset volume is achieved.

C Third point is what cycles the breath? This can be time, in which breath is terminated, once a preset inspiratory time is elapsed and expiration is allowed. This is called time cycling. Other mechanism is flow cycling, in which ventilator senses decelerating inspiratory flow and breath is terminated, once flow rate falls to a critical point. This is called flow cycling. This will be discussed in more detail later.

Initial assisted ventilation in 1960s was time cycled pressure limited ventilation, where ventilator delivered intermittent mechanical breath at preset interval (IMV), irrespective of infant's spontaneous effort.

As expected there is asynchrony between ventilatory breath and patient's spontaneous breath in such type of ventilation. This asynchrony may have many untoward effects. Asynchrony may impair gas exchange as well as might lead to air trapping and pneumothorax. There is also association of asynchrony with alteration in blood pressure, cerebral blood flow and intra ventricular haemorrhage.

This asynchrony can be avoided if patient's spontaneous effort and the onset of mechanical inspiration could be coordinated. Patient-triggered ventilation (PTV) is one of such innovation, which achieves synchronization between spontaneous and mechanical breaths. These synchronized ventilatory modes are characterized by the delivery mechanical breaths in response to a signal derived from the patient's inspiratory effort. Synchronized intermittent mandatory ventilation (SIMV), assist/control (A/C) ventilation and pressure support ventilation (PSV) are examples of patient triggered ventilation.

Signal detection: Flow sensor

Different ventilators have different trigger signal mechanisms for detection of patient's inspiratory efforts with some advantages/disadvantages over one another.
Following is tabulated list of these methods:

Synchronized intermittent mandatory ventilation (SIMV)

In this mode mechanical breaths are delivered in synchrony with patient's inspiratory effort. In between the mandatory breath, patient can breathe spontaneously from continuous flow in the circuit. In this mode onset of mechanical breath coincides with patient's inspiration but expiration is not synchronized as inspiratory time is preset and might be different from patient's spontaneous inspiratory time (Ti). As a result patient might start expiration, while ventilator is still in inspiratory phase. This might lead to fight against ventilator during expiration. This situation can be avoided by optimizing the inspiratory time according to patient's Ti.
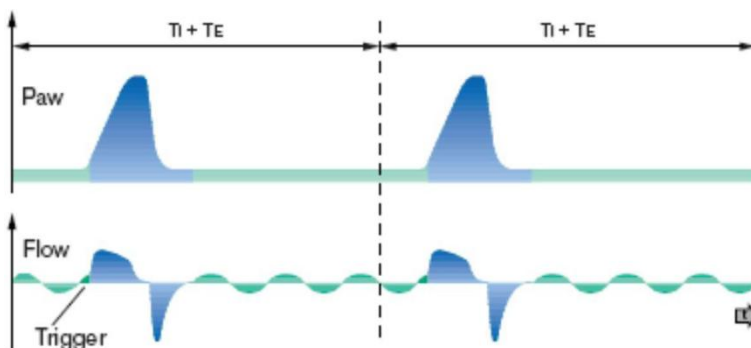
Assist/control ventilation (A/C)

This is also time cycled pressure limited ventilation, where every breath triggered by patient is supported. In case patient doesn't have spontaneous effort, ventilator delivers mandatory breaths as preset rate. Here also inspiration is synchronized but possibility of expiratory asynchrony still remains there. Merit over SIMV is that all of patient's breaths are supported.
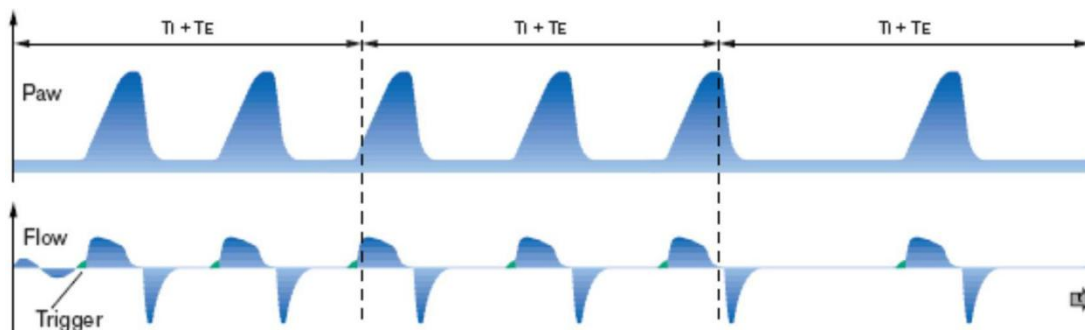
Flow cycled ventilation

In this mode of ventilation, every inspiratory effort of the patient triggers the ventilator. But unlike other modes, Ti is not pre-selected. Rather Ti is decided by time constant of the lung unit, so inspiration and expiration both are synchronized. Inspiration is initiated with patient's spontaneous efforts. During decelerating phase of inspiratory flow breath is terminated when flow rate falls to certain percentage of peak flow rate, which is called termination sensitivity. Termination sensitivity usually varies from 5 to 20%.
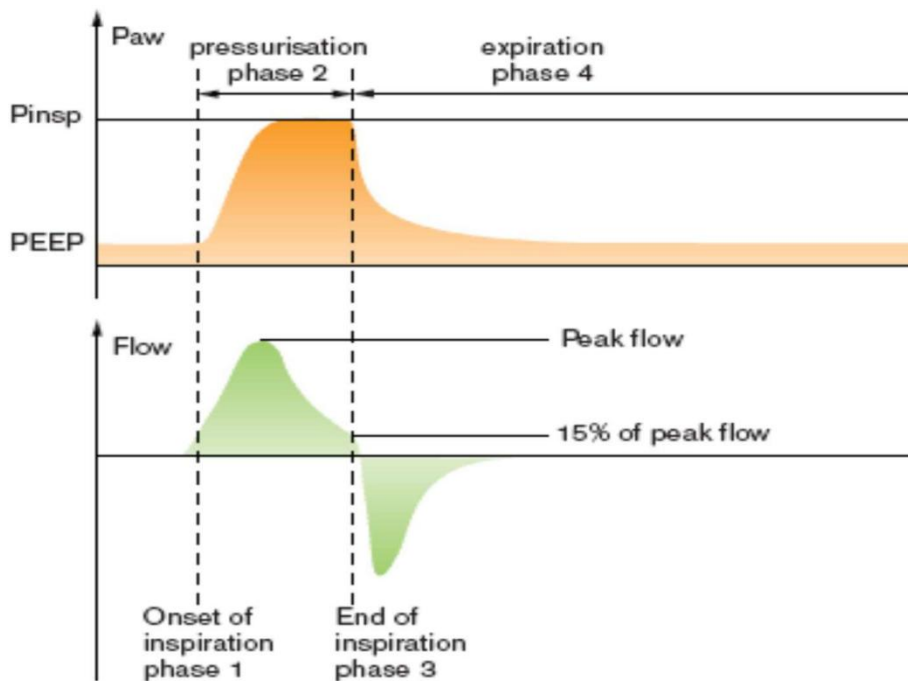
Pressure Support Ventilation: It is a pressure limited ventilatory mode in which each breath is patient-triggered and supported. It provides breath-by-breath ventilatory support by means of a positive pressure wave synchronized with the inspiratory effort of the patient, both patient-initiated and patient-terminated. Thus, during a cycle of Pressure Support Ventilation four phases seen:

- Recognition of the beginning of inspiration
- Pressurization
- Recognition of the end of inspiration
- Expiration

If flow cycling is used instead of time cycling, inspiration is terminated at a percentage of peak flow rather than time, thus providing full synchronisation between patient and ventilator. Thus A/C ventilation can be provided either as a time cycled or flow cycled mode. Flow-cycling is also incorporated in PSV which provides synchrony during both inspiration and expiration. Flow cycled A/C and PSV on surface look the same but PSV has additional features designed to regulate inspiratory gas flow in such a way that it provides a 'boost' to spontaneous breaths to help overcome the work of breathing imposed by the narrow lumen endotracheal tube, ventilator circuit, and demand valve.

As inspiratory flow in PSV is servo controlled, patient can breathe as much, as fast and for as long as he or she wants. In that sense it resembles spontaneous breaths and appears to be more physiologic as the patient has full control on his or her breathing.

Advantages of PSV:

- Better synchrony between patient and ventilator
- Increased patient comfort
- Reduced need for sedation
- Decrease in work of breathing
- Shorter duration of weaning process

SIMV+PC/PSV:

Alternative is to put baby on SIMV+PSV mode, if available. In this mode full support is provided through set SIMV rate. For rest of spontaneous breath a partial pressure support (PS) is added.

Summary:

During conventional ventilation neonates are ventilated with continuous flow, pressure limited, time cycled ventilators. Various triggered ventilation modes have been developed for neonates: Synchronous Intermittent Mandatory Ventilation (SIMV), Assist/Control Ventilation (A/C), and more recently Pressure Support Ventilation (PSV).

Among these, PSV gives the patient optimum liberty during ventilation. The patient decides over start of inspiration and start of expiration and therefore controls inspiration time, breathing frequency and minute volume. It's not only baby friendly but also doctor friendly.

How to initiate nasal CPAP& CMV
Kumar Ankur, Manoj Modi

- Try to put baby on CPAP as early as possible, before marked atlectasis sets in. A preterm neonate with RDS is better started on CPAP, right at delivery area and transported to NICU on same mode, as waiting for CPAP till arrival to NICU may cause unnecessarily delay and alveolar collapse.
- Don't rely on oxygen delivery tubing sets for CPAP. Use conventional CPAP circuits, either bubble CPAP or ventilator driven CPAP. Preferably use binasal short prongs.
- Start with PEEP 6-8 cm of H_2O and adjust FiO_2 to maintain SPO_2 in target range (85-95%).
- Success of CPAP is defined by decrease in work of breathing (WOB) to acceptable level, which is usually noticed within 30-60 min. Grunting should disappear and retractions should decrease to acceptable limits.

Maintaining and Weaning from CPAP:
- If baby stabilizes on CPAP with acceptable WOB and FiO_2<50%, CPAP may be continued.
- Pressure and FiO2 may be reduced gradually as per clinical condition and response.
- Once FiO2 is <25-30% and CPAP level <5, baby may be weaned off CPAP to O2 by nasal cannula, though many wait till baby come to 21% FiO2 before weaning off.
- Duration of CPAP may be be as short as couple of hours in mild disease to as long as several weeks in severe lung disease.
- While ventilating the neonate, take care that baby isn't too agitated. It may induce pneumothorax. Try to eliminate the reason of irritation and pacify the baby.

Failure of CPAP:
- Persistent severe retractions and/or grunting or FiO_2 need >40-50% even after 30 min of CPAP may be considered as indications of intubation and mechanical ventilation. Other indications of intubation are repeated episodes of apnea (despite xanthenes), or marked acidosis (pH<7.2). Failure of CPAP:
    - Persistently high WOB despite adequate CPAP (6-8)
    - FiO2 need >40-50%
    - Repeated episodes of apnea, nonresponsive to xanthine therapy(Caffeine citrate or Theophylline)
    - Severe acidosis (pH<7.2)
- Before declaring failure of CPAP, check whether CPAP is being delivered effectively. If baby is too agitated or there is significant leak of flow through mouth, enough pressure will not be generated. Try to pacify the baby by

massaging/stroking/caressing. Look if nasal prongs/assembly are hurting the baby. May consider soother or gentle strapping of chin.
- If baby gets repeatedly apneic on CPAP:
  Look for airway obstruction
  Start/escalate xanthines if apnea of prematurity.
  Rule out other causes of apneas.

Nasal IPPV:
- Many clinicians consider nasal IPPV as the missing link between CPAP and ET ventilation.
- In this mode, assembly is same as for nasal CPAP, only difference is that on top of PEEP, positive breaths are added at a certain rate.
- Usual ventilator settings are as follows: PIP of 12-18 cm of water, inspiratory time 0.3-0.5 second, rate 20-30.
- This further augments infants spontaneous efforts and Nasal IPPV has been shown to reduce the intubation rate in infants who were failing on CPAP.
- An attempt of nasal IPPV may be warranted in any infant, who is failing on nasal CPAP, before a decision of intubation is taken, particularly if infant is not very preterm (in very preterm infant trial of nasal IPPV may delay the benefit of surfactant therapy.

Initial mode of invasive ventilation

- In most neonates with respiratory distress, initial mode of ventilation should be CPAP. However if respiratory drive is not sufficient or CPAP fails, baby should be intubated and put on mechanical ventilation.

Intubation and mechanical ventilation:

- Threshold for intubation may vary among neonatal units. Usually we decide to intubate a neonate once he fails on CPAP (as mentioned above).
- In a preterm neonate, decision of intubation may be taken even earlier to give benefit of early surfactant therapy. Subsequently baby may be rapidly extubated after surfactant delivery *(INSURE: INtubation-SUrfactant administration-Rapid Extubation)*. In this regimen, as soon as infant is symptomatic, infant is intubated, surfactant instilled into trachea and infant is then extubated to CPAP within few minutes. This practice is claimed to shorten subsequent ventilation.
- In most of infants >28 wks it may be worthwhile to give a fair trial of CPAP before considering intubation and mechanical ventilation/INSURE, as intubation itself might destabilize them and most of them do with CPAP alone. In smaller infants (<28wks), benefits of INSURE may outweigh any harm incurred.

<u>Intubation:</u>

 – Select appropriate size wndotracheal tube (ET) tube. Tube should neither be very tightly fitting, nor having high peri-tubal leak (>20-25%). Usual guidelines are based on infants weight:

Weight	Inner diameter of ET
<1 kg	2.5 mm
1-2 kg	3 mm
2-3 kg	3.5 mm
>3 kg	4 mm

 – Keep appropriate size mask, bag, oxygen tubing, reservoir, suction catheter ready before proceeding. An appropriate size mask should cover chin, mouth and nose but not eyes. Maintain thorough aseptic precautions. If baby is too agitated, sedate him with IV midazolam (0.1 mg/kg). If intubation attempt is getting prolonged or baby has severe desaturation, interrupt the procedure, rexogenate with supplemental oxygen or bag and mask. Be gentle, don't panic. If needed call for help, rather than taking it to your pride.
 – Don't place the ET too deep into the trachea. Always look at vocal cord guide on ET tube (dark line at the tip), and keep it at level of vocal cord. Look for B/L equal air entry, before securing it. Optimal tube position should be later reconfirmed on chest X-Ray. If tube is closer to carina, pull it out. Note the cm marking at the corner of mouth. This reading will be a future guide for reintubation and tube fixation. A rough rule for length of tube from corner of mouth is: 6 + birth weight in Kg.

Tip to lip distance =	6 + birth weight in kg

 – However this formula overestimates the distance at times. Always check tube position by auscultation and on X-Ray.

Selecting "Mode of ET ventilation

 – *<u>We usually use either SIMV or PSV mode</u>*

 – Many clinicians prefer volume guarantee modes (VG-Volume Guarantee; VAPS-Volume Assured Pressure Support; PRVC-Pressure Regulated Volume Control) etc. In these modes caregiver sets the desired tidal volume (4-6 ml/kg) and then peak inspiratory pressure (PIP) is constantly manipulated by the ventilator so as to keep the tidal volume VT (VT) into target range. Whereas in pressure modes (SIMV, PSV), care giver sets a pressure, so as to generate desired tidal volume. In this case VT will keep changing as per changing lung compliance. If compliance improves, delivered VT will increase and vice versa.
 – Message is that we shall not get blindly carried away by any particular mode. Keep looking if your set mode and settings are suitable for baby or not. Basic fundament

should be to provide adequate support to the baby so as to assist in his WOB, without taking total control of the situation.

Optimizing "Ventilatory settings":

- PEEP (Positive End Expiratory Pressure): set a PEEP of 4-6 cm H2O to start with. Later adjustments can be made by looking at FRC on chest X-Ray. Optimal FRC will be dome of right diaphragm at 7-9ᵗʰ posterior ribs. If X-Ray shows hyperinflation, decrease PEEP by 1-2 and vice versa.
- PIP (Peak Inspiratory Pressure): Adjust the PIP so as to achieve a tidal volume 3 to 5ml. This will cause visible chest rise (but not too much of chest excursions).
- Inspiratory time (Ti): Adjust theI time so that there is minimal gap between inspiratory and expiratory flow waveforms. Ti is usually kept short in hyaline membrane disease (HMD) i.e close to 0.25 sec and little longer in MAS (0.35 sec). In normal lung ventilation, Ti may be as high as 0.5 sec. It's better to look at flow waveforms for final adjustment or switch to PSV mode.
- Rate: Adjust the rate to provide optimal support. In severe disease set higher rate, you may require to set a rate as high as 70-80 per minute. In mild disease a rate of 30-40 might be sufficient. One way of dealing the situation could be shifting to PSV mode and allowing all the breaths be supported. Or in case of SIMV, we may set ventilatory rate so much that unsupported breaths are not more than 25-35% of total. Final manipulation of rate is done on looking at PCO2 in blood gas. If there is hypocapnia, decrease the rate (and PIP, if very high); if there is hypercapnia, increase the rate. As a lung protective strategy, many clinicians will tolerate a PCO as high as 55-60, as long as pH is >7.2.
- FiO2: Adjust FiO2 so as to maintain saturation 85-95%. In meconium aspiration syndrome MAS (± PPHN-Persistent Pulmonary Hypertension) target SPO2 may be 90-95%.
- Flow rate: set flow rate 6-8 liter/min.
- An X-Ray should be obtained within ½ to 1 hr of initiating ventilation, which will define the position of ET tube, FRC as well condition of lung parenchyma.
- Maintain a ventilatory chart, on which all important parameters are noted every hrly.

Other supportive measures:
- Investigate and Treat the underlying disorders, as indicated by clinical scenario: sepsis, pneumonia, airleak, pulmonary hemorrhage, shock, PDA, PPHN etc etc.

Monitoring of a ventilated neonate:
- Includes hourly vitals HR, RR temp, SPO2, BP, perfusion (CRT), respiratory distress and synchrony with ventilator, abdominal and neurological status.

- Ventilatory parameters both *set parameters* (PIP, PEEP, I time, rate, flow rate, FiO2) as well as *measured parameters* (PIP, PEEP, FiO2, MAP, tidal volume, minute ventilation (MV), compliance and resistance) should be noted periodically.
- Intermittent arterial blood gas should be obtained while patient is on ET ventilation, frequency of which will vary according to the severity of disease and fluctuations in ventilatory settings.
- If disease is severe and condition of baby is unstable and/or there are significant alterations in ventilatory parameters, blood gas should be repeated frequently (every 2-4 hrly).
- Once condition stabilizes, frequency of gases can be decreased to 2 to 3 gases per day or even lesser. Minor manipulations in settings can be done by looking at measured parameters alone(VT & MV).
- In case of any acute deterioration systematic examination should be done in following order:*DOPE*
  - Tube displacement (D)
  - Tube obstruction (O)
  - Pneumothorax (P)
  - Equipment malfunction (E)
  - Sepsis/pulmonary hemorrhage
  - PDA etc.

INITIATION OF **Ventilation IN RDS**
Ashish Jain

Resuscitation Strategy:

Most of these babies may require some form of resuscitation at birth. The use of a strategy as per the NRP guidelines would have a impact on the ventilation later on (if needed) and alter the outcome in a significant manner.

- T piece resuscitator is recommended as this provides a consistent controlled PEEP
- Initial FiO_2 should be between 30 to 40% in most of the preterms. This can be promptly increased to attain the target saturations according to published 2010 NRP guidelines
- Intubation solely for the purpose of administrating surfactant is generally not recommended at this stage in all babies

Delivery Room (DR) Strategy:

The initiation of a ventilator strategy should be done right at the time of birth. There is now a strong evidence to support the DR strategies to aim at facilitating lung fluid clearance and helping preterm infant with insufficient chest wall rigidity to establish FRC (Functional residual capacity).

27 weeks and less	27 to 32 weeks	More than 32 weeks
Infants whose mothers received ANS and who exhibit good respiratory efforts : • Initiate NCPAP immediately • Avoid sedation • If RD occurs and the FiO2 >40% give early rescue SF (within 2 hrs of birth) • Administer caffeine for babies less than 1250 grams ***Infants whose mothers have not received ANS*** • Give prophylactic Surfactant • Immediately put the baby on NCPAP if good respiratory efforts	• Initiate 5-7 cm Nasal CPAP • Avoid Sedation • If RD progresses and FiO2 > 40% while on 6-8 cm of CPAP, place infant on SIMV and give rescue Surfactant • Monitor ABG & clinical status of the neonates every 30 min. • As lung function improves (may be rapid). Wean PIP & FIo2, followed by rate. • When the ventilator support is weaned to minimal levels , attempt extubation on to N-CPAP	If no specific intervention is needed at birth the following graded strategy can be used • Initiate O2 in spontaneously breathing neonates (warm humidified oxygen) by hood • If the infants exhibits respiratory distress and requires 40% oxygen , place the infant on Nasal CPAP and adjust the pressures • If oxygen requirement remains above 40% despite CPAP of 6-8 cm, then intubate and place the infant on SIMV and give rescue Surfactant

CPAP Initiation Guidelines:

CPAP is generally indicated in all preterm with respiratory distress and a oxygen requirement of 40% or more to maintain a appropriate saturation

Type: Delivered by any of the delivery systems, viz Bubble CPAP, Constant flow ventilator or a variable flow system, however the ease of use of ventilator CPAP and the ease to conversion to invasive or non-invasive positive pressure ventilation make the ventilator CPAP a pragmatic option

Interface: Nasal CPAP should be initiated with short bi-nasal prongs and a interface to which the staff and the baby is most comfortable

Settings: Initiate the CPAP at 5 cm and step up to 7-8cm. optimal effects occur between 6-8 cm. The CPAP failure is considered when the (1) O2 requirement persists above 40% (2) severe apnea (3) severe hypercarbia

Initial Ventilator settings for RDS

During the conventional ventilation, attempts should be made to minimize volutrauma to the lungs by limiting PIP and delivered tidal volume to the lowest possible levels. This is best achieved using an "open lung strategy" using adequate lung recruitment with PEEP combined with permissive hypercarbia. In neonatal mechanical ventilation this is best achieved by applying adequate levels of PEEP (or MAP during HFOV). Optimal PEEP must be tailored to the lung compliance of each individual patient. In infants without lung disease, appropriate PEEP may be in the 3 to 5 cm range. For those with poorly complaint or severely atelactic lungs, PEEP levels as high as 8 cm H2O may be necessary.

| Mode | One may use the Volume ventilation (However ensure that the sensor is optimal and the sensing is near the patient end)

SIMV, PSV (with SIMV)

AC +VG or PSV +VG

SIMV + Volume Guarantee for babies less than or equal to 1500, who need surfactant | Pressure support ventilation (PSV) is preferred in small preterm infants because small ET tubes impose high resistance and work of breathing during weaning process. The PSV and the AC results in better synchronization

There is a disadvantage of the pressure limited ventilation as , there would be an inadvertent delivery of the volume when the compliance of the baby is rapidly changing , e.g. the HMD and surfactant administration

One should understand that the available universal ventilators (neonatal to adult population), control the VT delivered into the proximal end of the ventilator circuit, not the VT delivered to the patient. The loss of volume to compression of gas in |

		the circuit and the humidifier and the ETT leak may be 755 or more of the total, making standard VT ventilation difficult to use effectively in small newborn babies.
Rate	35 to 45 (Synchronized IMV breaths indicated)	This should be based on the severity of the illness, and the respiratory effort of the neonate. In infants with severe disease and low respiratory efforts , the rate should be 40-50/min Spontaneously breathing infants with less severe disease may be set at a respiratory rate of 40/min
PIP	15 to 25	Base your PIP on the chest rise , should be like than in a normal breathing neonate .Adjust, as needed to achieve a TV measured of 4-6 ml/Kg Pressure control above PEEP : 10-15
PEEP	5 cm H2O	Or a PEEP sufficient to deliver the desired TV , another method to obtain a optimal PEEP is to increase PEEP stepwise , till the FIO2 requirement falls (May increase upto 8-10 cm)
Ti	0.30 to 0.35 Maximum of 0.4 in PSV mode	This should be decided on the Time constant (time taken by the air to get in and out of the lung). The Time constant is less in preterm with RDS.
Flow	8 to 10 liters/ min	A pressure rise time that is 30 to 50% of the Ti , is appropriate in most of the times
Fio2	Start with 50%	This is later adjusted as per requirement
PS above PEEP	Pressure support above PEEP , 60% of the Pressure control above PEEP in case baby on SIMV + PS	The same may be reduced , when the baby is being weaned, especially when the surfactant is administered

Continued vigilance is necessary to detect improving lung compliance to avoid lung over distention and alveolar rupture. This may occur rapidly after a dose of exogenous surfactant. If oxygenation remains poor, or severs Hypercarbia occurs on SIMV, alternative management may be required. If PIP of 30 cm H_2O) or greater or MAP 12 to 14 cm H_2O is necessary with conventional ventilation, or if severe hypercarbia persists, the patient is a candidate for HFOV.
As lung compliance improves, wean FiO_2 and PIP followed by ventilator rate. When support has been weaned to FiO_2 less than 40%, PIP 18 to 20 or less, rate 25 and PEEP 5cm or less, and there is a good respiratory effort, the infant may be extubated. The nasal CPAP when

used post extubation is seen to reduce the extubation failures. The use of synchronized ventilation and the volume guarantee may enhance the weaning process.

INITIATION OF VENTILATION IN MAS

There is little evidence from clinical trials on ventilator treatment of MAS; conventional management is primarily based on pulmonary patho-physiology. However, the complicated pulmonary patho-physiology resulting from areas of atelectasis and areas of hyperinflation , in association with ventilation-perfusion mismatch and airway compromise , makes ventilator management of MAS one of the neonatologists greatest challenges.

Oxygen alone:

Many clinicians will treat initially oxygen alone without positive pressure of any type. An FiO_2 of 100 has been advocated in the past. But, studies have shown that the administration of high O2 may produce free radicals resulting in the constriction of the pulmonary arteries, and also result in negative biochemical and clinical response. Hence, one should not exceed a FiO_2 of 80 %, and if concentrations higher than these are required, one should quickly switch to some form of positive pressure ventilation. Hence the following initial guidelines may be followed

Amount of FiO_2 to be given as standalone therapy	Maximum of 80% , later one should shift to some form of PPV
Mode of administration of O2	Oxygen hood as, this is easily tolerated and a higher FIO2 can be administered.
Targets	SPO2 of 90 to 95% PaO2 of 70 to 90 As intermittent hypoxia may result in PPHN
Shift to CPAP or IMV	PaO2 <50mm Hg, PaCO2 of >60mm Hg or Acidosis of <7.25 in an enriched environment with FiO2 >0.80.

Infants with MAS without PPHN:

Infants with MAS without associated PPHN may be managed in relatively standard fashion with slightly higher oxygen targets than used in the preterm babies with RDS. The guidelines for the initiation of the ventilation in such babies are summarized as below;

Targets	pH to be maintained above 7.3 $PaCO_2$ 40 to 50 mm Hg PaO_2 70 to 80 mm Hg
PIP	Not exceeding 25 cm H_2O
RR	Relatively rapid ventilator rate of 40 to 60
PEEP	Moderate of 4 to 6 cms H_2O If the diaphragm on the chest x ray are flat and the gas trapping is suspected ; the PEEP should be decreased to minimum of 3 to 4 cm of H2O

IT	Relatively short inspiratory time of 0.3 to 0.4 seconds
	If the diaphragm on the chest x ray are flat and the gas trapping is suspected ; the IT should be decreased to minimum of 0.25
FIO2	To maintain the Spo2 90 to 95 (generally up to 80%)

One should consider shifting the baby to HFOV (High frequency oscillatory ventilation) in case higher ventilator settings are required, as chances of air leak is very high in these cases.

Infants with MAS complicated with PPHN:

In infants whose MAS is complicated with significant PPHN, a different ventilator strategy should be followed. Some of the guidelines for ventilation of such a case are as follows

Targets	Pao2 to be as high as 80 to 100
	PCo2 30 to 35 mm Hg (this results in mild alkalosis that acts as a buffer to acidosis that may trigger PPHN
	pH above 7.35 (avoid aggressive alkalosis)
Mode	One may use a patient triggered / Assist control ventilation with sedation (especially in a very active baby)
Avoid	Chest physiotherapy
	Noise
	Handling
	Bright Light
PIP	15-25 cm H2O
	Target VT 4-6ml/kg
	Institute HFOV for PIP >25
Rate	50-70 breaths / min
PEEP	3-4 cms (avoid higher PEEP especially where the MAS is associated with leaks)
I Time	0.4 to 0.5 (shorter Ti may be used for predominant atelactic MAS)
Fio2	Aggressive oxygenation to maintain Pao2 >80

Neonatal Disease specific ventilation: Protocol
Kumar Ankur, Manoj Modi

In this chapter we are going to discuss the ventilatory strategies in common neonatal diseases like hyaline membrane disease, meconium aspiration syndrome, bronchopulmonary dysplasia, PPHN, apnea of prematurity and hypoxic ischemic encephalopathy.

Respiratory distress syndrome

Optimal management of mechanical ventilation requires astute bedside clinical assessment and accurate interpretation of blood gas data and chest radiographs.

Recommended initial settings:

1. Always give fair trial of NON INVASIVE VENTILATION --nasal CPAP (Bubble or Ventilator CPAP)
    - Set flow rate
    - Set fiO_2 with blender as per target SpO_2
    - Target SPO2
        o Preterm: 88-92%
        o Term: > 90%
    - Attach with humidifier
    - SET PEEP (5 cm or more)
    - If you are using bubble CPAP
        o Look for bubbling & set flow rate accordingly (Note: PEEP also increases if you increase the flow rate)
        o If no bubbling or ventilator showing leak: Look for any leak in tubing's or from mouth, so if possible wrap mouth with splint.
    - Look for work of breathing, spo2, CXR, ABG (SOS)
    - Now how to proceed
        o **STEP 1:** if distress is still there & fio2 requirement is increasing & CXR revealed low FRC & white lung then increase the PEEP up-to 8 cm of water to <u>RECRUIT</u> the lung.
        o Signs of adequate recruitment:
            Decreased work of breathing (RR, grunting, & sub costal retraction)
            Gradual decrease in fio2 requirement to 21% over few minutes
            Gradual decrease in PEEP from 8 to 5 cm of H2O over few hours
            CXR: good FRC

After fair trial of CPAP if (PEEP is > 7 cm of H_2O along with fiO_2 requirement > 0.40) if work of breathing is still there & baby is barely maintaining SpO_2 then consider for intubation & surfactant administration.

2. INVASIVE VENTILATION: Conventional mechanical ventilation
   - Intubate the patient.
   - Consider for **SURFACTANT-INSURE (Intubatin Surfactant Administration & Rapid extubation)**
   - Mode: SIMV, SIMV+PC(PSV), PSV or Volume guarantee mode as per your ventilator
   - Set parameter
     - Flow: 6-8 litre/min
     - PEEP: > 5cm of water (change as CXR –FRC)
     - PIP: look for chest rise during inspiration
     - Rate: if SIMV/SIMV+PC: 30-50 /min. If PSV: only purpose of setting is back-up rate.
     - Fio2: as per your target SpO_2
     - Ti: 0.3 to 0.4 second
     - If you are using SIMV, SIMV+PC, PSV; then regulate PIP & PEEP till you to achieve the target Vt.
     - If you are using Volume guarantee mode; then set target Vt & ventilator will achieve with change in pressure.
     - Always look for

       Work Of Breathing (RR & retraction)

       SPO_2: decrease or increase fio2

       If spontaneous breathing rate is very high then most of the time it appears that you are not giving adequate pressure.

       TARGET TIDAL VOLUME(Vt): 4-6 ml/kg

       DONOT give any sedation even if RR is very high, provided if tidal volume, SpO2 & ABG is within <u>ACCEPTABLE</u> range.

> **Inspiratory time:** Adjust the I time so that there is minimal gap between inspiratory and expiratory flow waveforms. Ti is usually kept short in HMD (close to 0.25 sec) and little longer in MAS (0.35 sec). In normal lung ventilation, Ti may be as high as 0.5 sec. Its better to look at flow waveforms for final adjustment or switch to PSV mode.

Rationale for these settings is derived from clinical trials and physiologic principles.

Rapid ventilator rates and short Ti values are generally tolerated because of the characteristically low pulmonary compliance and short time constant in neonatal RDS. Always try to avoid volutrauma, as it is most dangerous.

Situation- I
   - Baby stable on ventilator. CXR: good FRC. ABG: pH: 7.25, pCO2-up-to 65 mm.
In this condition tolerate hypercapnia if pH is $\geq$ 7.2 & baby is hemodynamically stable.

Because if will target to correct this, that means you have to increase pressure which may lead to more tidal volume delivery & can cause volutrauma.

Situation- II

- Baby stable but blood gas is showing hypocapnia, then reduce PIP first and use high rates to maintain adequate minute ventilation. Also tolerate permissive hypercapnia means pCO_2 in range of 55 – 65 cm H2O as long as pH remains above 7.25.

Situation- III post surfactant

After surfactant delivery, reduce their settings fast especially PIP and try to shift them to nasal CPAP. This technique is known as INSURE meaning Intubation, Surfactant administration and Rapid extubation. Our ultimate aim is to reduce the lung injury and in other way to prevent development of bronchopulmonary dysplasia.

Surfactant administration

Kumar Ankur, Manoj Modi

Surfactant is complex of phospholipids and proteins, which reduces collapsing force in thealveolus, conferring stability and maintaining alveolar surface free of liquid. Surfactant changes surface tension as surfaces expand and contract.

Classification of surfactants:

1. Synthetic non-protein- EXOSURF
2. Animal-derived, protein-containing- SURVANTA,
   CUROSURF, NEOSURF

Indication:
1. Any infant at risk of RDS (Preterm or Late Preterm or Term)
2. If CPAP fails (PEEP > 7 cm of H2O & fio2 > 40%).
3. If CXR is white out but baby on CPAP (PEEP 6-7 cm of H2O & fio2-30 40%) barely maintaining saturation.
4. Baby requiring intubation in v/o respiratory distress (HMD).

Timing of administration:
1. Preferably during 1st 12 hours
2. But can consider after 24 hrs to get help in early extubation.

Prerequisites before administration of surfactant:
1. Vitals should be stabilized before administration of Surfactant.
2. Always confirm endotracheal tube position before instilling surfactant.
3. It should be given without disconnecting the ventilator through a special surfactant tube with a side port
4. Pulse oximeter should be connected to the patient.

Materials required
1. Surfactant vial stored in a refrigerator
2. Two 10 ml syringes and a 18 gauge needle
3. Sterile dressing sheet
4. Scalpel blade
5. Sterile gloves, cap, mask and gown
6. Appropriate size surfactant administration endotracheal tube

Endotracheal tube with side port for administration of surfactant

Dosages:
Survanta- 4ml/kg (100mg/kg of phospholipids) vial available as 8 ml vials
Curosurf- 2.5 ml/kg (200mg/kg of phospholipids) initial dose and later 1.2 ml/kg available as 1.5ml and 3 ml vials
Neosurf- 5 ml/kg (135 mg/kg of phospholipids) vial available as 3 and 5 ml vials

Methodology:

1. Warm the surfactant ampoule before administration by holding in the hand for 8 minutes or by leaving at room temperature for 20 minutes.
2. Never use artificial warming methods.
3. *Donot shake the vial* and donot use the solution if discolored.
4. Divide total dose into four equal aliquots and administer each aliquot as a bolus through the side port of the surfactant tube at an interval of 5 minutes between subsequent doses with total time of administration being 15-20 minutes.
5. Position of the infant does not need to be changed during administration of the surfactant.
6. Frequent ventilatory adjustments may be needed by decreasing the pressures to prevent air-leaks.

Continuous SpO2, IBP (Invasive blood pressure) / NIBP(Noninvasive blood pressure), HR monitoring should be done. No routine X-ray to document improvement.

7. No suction for 6-8 hours

Complications:

- Transient hypoxia / bradycardia
- Reflux into pharynx
- Plugging of endotracheal tube
- Hemorrhagic pulmonary edema
- Pneumothorax
- Opening of the PDA due to sudden decrease in pulmonary pressure

X-ray of baby with HMD before surfactant (Low FRC Lung)
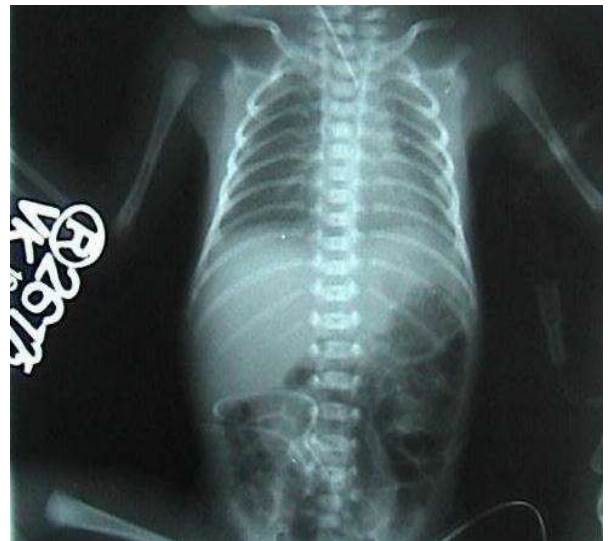
X-ray after post surfactant showing better lung aeration (Good FRC)

High Frequency Ventilation

Kumar Ankur, Sanjeev Chetry

Introduction:

Much progress has been made in the treatment of neonatal respiratory failure over the past few decades. However, lung injury and pulmonary morbidities secondary to mechanical ventilation remain an ongoing problem in the care of premature infants. High frequency ventilation(HFV) is a form of mechanical ventilation that uses small tidal volumes and extremely rapid ventilator rates. Whatever he HFV system, the presumed linear relationship between ventilator rate and CO_2 elimination improves with decreasing ventilator frequency as long as the inspiratory to expiratory time ratio (I:E) is held constant.

Principles of gas exchange:

Chang described the multiple modes of gas transport that occur during HFV (Fig 1 and 2)

- Bulk Convection: Direct ventilation of the alveoli that are lying close to the trachea is called the bulk convection. With a decreased tidal volume (as in HFO), a small fraction of the lung receives fresh gas with each inspiration, therefore allowing sufficient gas exchange to occur in that particular lung region. As long as the tidal volume remains above a certain limit, direct alveolar ventilation will contribute in some way to gas exchange during HFO.

- Taylor dispersion: Augmented diffusion occurs because of turbulent air currents that result from interaction between axial velocity and the radial concentration gradient in the airway.

- Pendelluft Effect: The alveoli are ventilated asynchronously, as opposed to synchronously in conventional ventilation. This asynchronous ventilation occurs when small neighbouring regions of the lung are different in compliance, air resistance or the time constants of their filling or emptying. This method o gas transport is termed as "pendelluft", which is defined as transient movement of respiratory gases out a certain and into neighbouring alveoli either at the instant of Zero flow at the end of inspiration, or in the opposite direction at the end of expiration.

- Asymetric Velocity Profiles: Convective gas transport is enhanced by asymmetry between inspiratory and expiratory velocity profiles that occur at branch points in the airway. The velocity profile of the inhaled gas is initially parabolic, but on exhalation is flat. After a full cycle, gas particles initially near the centre of the flow are displaced to then right while those near the wall are displaced to the left.

- Molecular diffusion: Diffusion occurring near the alveolo-capillary membrane with the random thermal oscillation of the gas molecule. This suggests that as long as the gas molecules are of a temperature that is above absolute zero, then molecular diffusion will always occur.
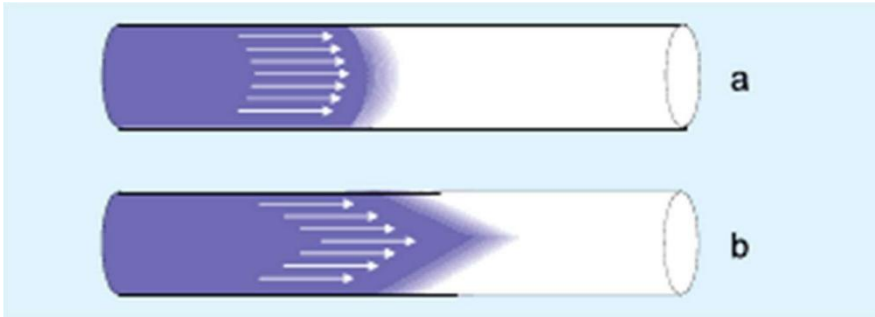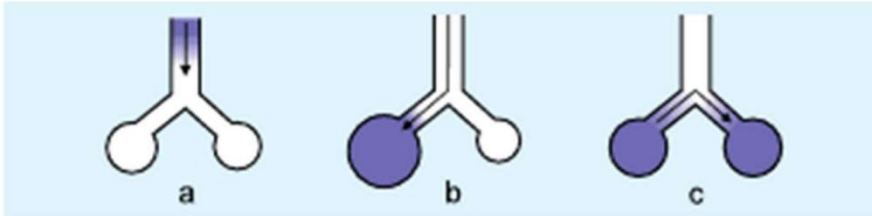
Fig 1, Taylor dispersion

Fig 2, Pendulum shift

Currently, there are three general types of HFV:
- High Frequency Positive Pressure Ventilation (HFPPV), which is produced by conventional or modified CMVs operating at rapid rates;
- High Frequency Jet Ventilation (HFJV), which is produced by ventilators that deliver a high –velocity jet of gases directly into the airway;
- High Frequency Oscillatory Ventilation (HFOV), which is produced by a device that moves air back and forth at the airway opening and produces minimal bulk gas flow.

High frequency Oscillators

High frequency oscillators (HFOs) are a type of HFV that use piston pumps or vibrating diaphragms, operating at frequencies ranging from 180-2400 breaths/min (3-40 Hz), to vibrate air in and out of the lungs. During HFOV, inspiration and expiration are both active (proximal airway praesures are negative during expiration). Oscillators produce little bulk gas delivery. A continuous flow of fresh gas rushes past he source, generating or powering the oscillations. The amplitude of the pressure oscillations within the airway determine the tiny tidal volumes that are delivered to the lungs around a constant mean airway pressure. This allows avoidance of high peak airway pressures for ventilation as well as maintenance of lung recruitment by avoidance of low end –expiratory pressures. Abasia flow system supplies fresh gas. (Fig 3)
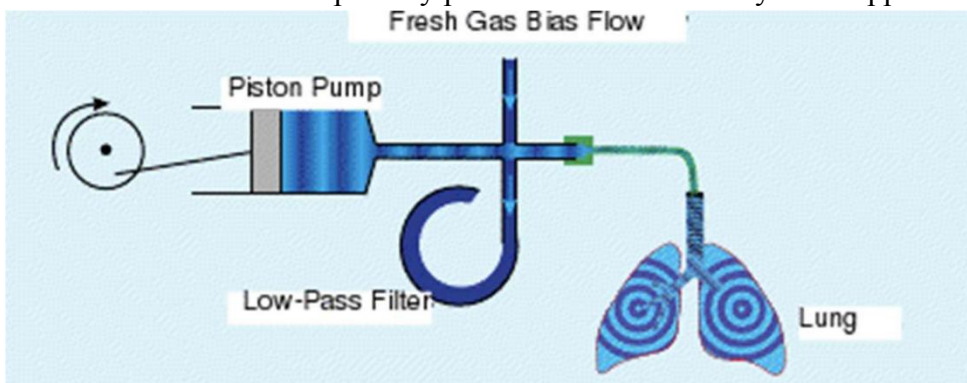

Figure 3 operating principle of HFOV

Variables on HFO

Three parameters determine oscillatory ventilation (Fig 3): mean airway pressure (MAP) around which he pressure oscillates; secondly, the oscillatory volume, which results from pressure swings and essentiallydetermines the effectiveness of this type of ventilation; thirdly, he oscillatory frequency denotes the number o cycles per unit of time.

Oxygenation:

During HFO, oxygenation is controlled by the inspired oxygen concentration and the MAP which controls lung volume. Oxygenation during HFO is independent o frequency and tidal volume, except at very low values. Two 'volume or pressure strategies'can be employed during HFO; low volume/low pressure or high volume/high pressure. The former strategy is used with the aim of reducing barotraumas and the latter to maintain lung volume above its closing pressure and ensure lung recruitment. The high volume strategy can improve pulmonary mechanics and is associated with a reduction in inspired oxygen concentrationand diminished structural injury. Increasing MAP on transfer from conventional ventilation to HFO (high volume strategy) improves oxygenation; whereas transfer to HFO at the same MAP as used on conventional ventilation had a variable effect on oxygenation. The MAP necessary to optimize oxygenation during HFO is directly correlated with disease severity. Infants with severe RDS with poor gas exchange on conventional ventilationhave very low lung volumes. Not surprisingly, then, such infants require the largest changes in MAP to optimize their lung volume adherence oxygenation. Such a strategy, however, is not appropriate to the healthy lungs as this may lead to over distension.

For any infant, there is a MAP level at which lung volume is optimum and hence so is oxygenation. Increasing MAP above that optimum level will impair oxygenation, as well reducing it below that level. The optimum MAP level will depend onthe infants disease severity and hence lung volume. The relationship between the mean airway pressure displayed on any given oscillator and the mean alveolar pressure in the infants lung will vary with the device. In the Sensor Medics at 30 % inspitrati0on the mean alveolar pressure is lower than displayed; the same is true for the InfantStar. For the Humming Bird and possibly other Sine Wave generators with an I/E ratio of 1:1 the displayed and actual pressures are about equal but, if using a Drager Baby Log 8000, the intra pulmonary pressures will be higher than the displayed pressure. This has practical implications if a hospital has more than one type of oscillators. For eg. a switch from a Sensor Medics 3100 A to a Drager ventilator at a display pressure of 12 cm H2O could result in a 5-8 cm H2O abrupt increase in distending pressure.

Ventilation:

Ventilation in CMV is calculated by the product of respiratory rate (f) times tidal volume (VT). In contrast ventilation in HFV is calculated by the equation $f^a X VT^b$ where 'a' is found to be between 0.75 and 1.24 and 'b' is between 1.5 and 2.2. For clinical application the equation is simplified to $f X VT^2$. Thus Co2 elimination is more strongly affected by changes in VT than in frequency. His explains why even small changes in VT would produce big effect on ventilation. Furthermore due to the characteristics of ventilator machine, the delivered tidal volume is inversely proportional to frequency. To avoid wide variations in delivered tidal volume the oscillatory rate is generally held constant during the clinical application of HFV.

Frequency:

During HFO, the delivered volume might be expected to be greatest at the resonant frequency of the respiratory system. The resonant frequency of the preterm lung varies between 15-20 Hz. The problem with the using high frequency to match the resonant frequency of the lung is that volume delivered by the commercially available oscillators decreases as frequency is increased. Thus in clinical setting while using HFO in very preterm babies one should use a frequency between 12-15 Hz so as to facilitate the oxygenation as tidal volume recruitment moving in and out of he lung is less but in cases of term babies with respiratory acidosis the frequency of 10 Hz or less is optimum as the volume delivery to the lung increases.

Indication for high frequency ventilation:

- Failure of conventional ventilation in the term infant (PPHN, MAS)
- Air leak syndromes (pneumothorax, PIE)
- Failure of conventional ventilation in the preterm infant (severe RDS, PIE, pulmonary hypoplasia) or to reduce barotraumas when conventional ventilator settings are high.

Elective HFOV

Some authors are of the view that starting high frequency ventilation as a primary mode of respiratory support results in less pulmonary injury. A meta analysis of 11 trials has shown HFOV had no significant effect on mortality or on short term neurological abnormality. Although, a reduction in BPD survivors at term was highlighted, he effect of HFOV was modest. The "HFOV" trials have, however been different in the details o their study design and the results obtained. The data are limited and the results are mixed as to whether HFJV may reduce incidence of CLD. Keszler and colleagues, in a multicenter controlled trial of 130 babies who had RDS, demonstrated a decreased incidence of CLD at 36 weeks corrected gestational age, as well as a decreased need for home oxygen therapy in the HFJV treated group.

HFOV as a rescue mode

HFOV is frequently used to "rescue" infants who has respiratory failure, due to a variety of conditions as has been mentioned above. Most of this is from non randomised trials or case series. For eg there are reports of infants with congenital diaphragmatic hernia and refractory hypoxemia on conventional ventilation improving when transferred to HFOV and similarly oxygenation in infants with pulmonary hypertension, but no long term benefits have been highlighted. Oxygenation in infants with severe pulmonary interstitial emphysema has also been documented to improve when infants are transferred from convention ventilation to HFOV. Here have been two randomised trials of HFOV in infants with severe respiratory failure, both report only short term outcomes. In term born infants, HFOV was shown to be more effective rescue support than conventional ventilation, but there were no significant differences in the requirement for ECMO or duration of ventilator or oxygen dependency between the two respiratory support modes. In preterm infants, although HFOV use was associated with a significant reduction in new pulmonary air leak, intracranial hemorrhage was increased.

Transfer from conventional Ventilation

- Use appropriate sized ET: Down the length of endotracheal tube there is attenuation of the oscillatory amplitude signal. The degree of attenuation was greatest with smaller ET and lower oscillatory amplitudes with appx 20% loss of amplitudes with smaller tubes.
- Make sure that hypotension is corrected before starting high frequency as the hypotension is exacerbated by HFO
- Use a higher MAP by 2 cm H2O than the child was on conventional ventilation. MAP should be increased by 1 cm every 10-15 min as the improvement in oxygenation because of the increased lung volume can be seen in that time only.
- The oscillatory amplitude is increased until the chest wall is seen vibrating. Vibrations til the umbilicus are considered adequate but beyond that are supposed to be excessive. However, this is a rough starting guideline and should always be correlated with blood gas examinations.
- Perform a chest Xray soon after commencing HFO to ensure adequate lung expansion has occured and there is no evidence of over expansion.
- Once lung volume is established, any procedure which requires the infant to be disconnected from the oscillator should be done as infrequently and as quickly as possible so as to prevent frequent loss of volume eg suctioning. It is preferable to use a closed system suctioning device.
- If the child is not improving despite increasing MAP on ventilator, then other causes should be sought before labelling as failure of HFO like- anemia, PPHN, hypotension, pneumothorax etc.

Weaning from high frequency

It is important to maintain lung volume to optimise oxygenation, thus, as recovery from RDS begins; the inspred oxygen concentration should be turned down before altering the MAP level. Once the inspired oxygen concentration has been reduced to 30%, the MAP is the reduced in 2 cm H2O steps at a rate dictated by the blood gases. If weaning is performed too rapidly, atelactesis will occur and it is necessary to increase the MAP level above that at which the weaning process occurred to optimise respiratory status once more. Once the MAP level has been reduced to 8 cm H2O there are two options for further weaning. One option is to extubate the infant directly from HFO. The second option is to change the infant to a shorter period of PTV prior to extubation; such a policy is facilitated by the machines that offer a choice of oscillation, triggered and conventional ventilation. A period on PTV can be useful to confirm that the infant has adequate respiratory drive to ensure successful extubation.

Adverse effects of high frequency ventilation

1. Gas trapping: leading to lung over inflation which may cause adverse cardiopulmonary function or air leak. This has not been found to be a significant problem during HFOV in premature infants in experienced centers.
2. Pulmonary interstitial emphysema: There have been few cases of PIE developing in babies on Hummng Bird Ventilator. Author postulated that the unusually low MAP used might be the main cause of PIE. At low MAP and low compliance gas can not be

transported into the atetlactatic alveoli. This led to an increase in the amplitude of oscillations in the peripheral airway resulting in overexpansion of the airways and hence airway injury.

3. IVH and PVL: A meta analysis of studies on the association of IVH and PVL with HFV in premature infants with RDS showed that a significant association was present only when the HIFI study was included, while analysis of the more recent studies without the HIFI study did not show any association. So possibly the current load of ventilators using the high volume strategy is not associated with increased IVH and PVL.

4. Noise pollution: The noise produced by Infant Star 500 and Sensor Medics 3100A were 53 (49-54) and 59 (56-64)dB respectively. These noise levels were lower than the recommended highest level of 85 dB by the occupational safety and health administration. However we have to be cautious in using HFV in patients receiving aminoglycosides. These infants are recommended not to be exposed to noise levels of > 58 dB.

5. Tracheal damage: This is a well documented complication of HFJV in neonates, but fortunately not described in HFOV. It is essential to adequately humidify (90% RH) the breathing gas otherwise severe irreversible damage to the trachea may result. Viscous secretion could obstruct bronchi and deteriorate the pulmonary situation. Excessive humidification on the other hand can lead to condensation in the paten circuit,the ET tube and the airways, completely undoing the effect of HFV.

Conclusions

HFOV used with a high lung volume strategy is at least as safe as CMV with the current lot of ventilators and may even be life saving and used as a rescue therapy. Experience is an important element in the safe and efficient use of HFOV particularly in premature infants. The long term risk-benefit ratio for HFV is not well documented. Follow up studies should be performed to investigate the long term survival, lung function and neurodevelopment of infants who have been treated with HFV in the neonatal period before this exciting form of ventilation is more widely accepted and used.

High frequency ventilation : IOWA PROTOCOL

Management Strategies with High Frequency Ventilation in Neonates and children Using the SensorMedics 3100A High Frequency Oscillatory Ventilator

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Sensor Medics Oscillatory Ventilator 3100A

A true high frequency oscillator with a diaphragmatically-sealed piston driver. Theoretically capable of ventilating patients up to 30 kg. Tidal volume typically delivered approximately 1.5-3.0 cc/kg (< dead space). Extremely efficient ventilation secondary to active expiratory phase, but not capable of delivering sigh breaths.

I. INITIAL SETTINGS:

A. FREQUENCY: Set initially at 10 Hz (600 BPM) for term infants and 15 Hz (900 BPM) for premature infants (< 2.5 kg). For children between 6-10 kg, use 8 Hz, between 10-20 kg, use 6 Hz and between 20-30 kg, use 4 Hz for an initial setting.

B. INSPIRATORY TIME (I.T.): Set initially at 33% (e.g. 22 msec at 15 Hz, 41 msec at 8 Hz, 55 msec at 6 Hz).

1) Warning - The percent of I.T. should never be increased because it will lead to air trapping and fulminant barotrauma. Total I.T. should only be increased by decreasing frequency, thus leaving the I:E ratio constant. I.T. can be decreased to 30% to heal airleaks.

2) I:E ratio: 1:2 (3-15 Hz), at 33% I.T.

C. POWER: A rough representation of the volume of gas generated by each high frequency wave. Range (1.0 - 10.0). Maximum true volume of gas generated by the piston is 365 cc. Maximum amplitude or volume delivered is highly variable and depends on the following factors: circuit tubing (compliance, length and diameter), humidifier (resistance and compliance - water level), ET tube diameter and length (FLOW is directly proportional to r^4/l, where r = radius of airway and l = length of airway), the patient's airways and compliance.

1) Set the POWER initially 2.5 if wt <2.0 kg, 3.0 if wt 2-2.5 kg, 4.0 if wt 2.5 - 4.0 kg, 5.0 if wt 4.0 - 5.0 kg, 6.0 if wt 5-10 kg, 7.0 if wt 10-20 kg. Chest wall needs to be vibrating. If not vibrating, increase power.

* Check ABG's every 15-20 min until PaCO2 is 40-60, i.e., titrate POWER setting based on PaCO2 desired. Many HFOV centers have you order amplitude or delta P to regulate ventilation instead of power. We have decided that the Power setting is a more reliable way of adjusting this ventilator and thus we order changes in power in order to regulate ventilation.

2) Alveolar ventilation is directly proportional to POWER, therefore the level of PaCO2 is inversely proportional to the power.

3) During HFOV, alveolar ventilation $(Ve) = (TV)^2 f$ as compared to CMV where $Ve = TV(R)$. Thus we primarily adjust the power (amplitude) to change tidal volume in order to manipulate ventilation.

4) Management of ABG's (Ventilation - Ve):

a) Change POWER by 0.2-0.3 to change CO2 ± 2-4 mm Hg

b) Change POWER by 0.4-0.7 to change CO2 ± 5-9 mm Hg

c) Change POWER by 0.8-1.0 to change CO2 ± 10-15 mm Hg

d) Warning - It is extremely important to normalize PaCO2 rapidly by weaning Power in order to avoid volutrauma from excessive tidal volumes. Thus check ABG's frequently (Q15- 20 min) and decrease POWER accordingly until PaCO2 > 35. PaCO2 < 35 correlates with an increased risk of pneumothorax. Thus to minimize the risk of volutrauma, it is important to use the least amount of TV (POWER or AMPLITUDE) possible to achieve ventilation.

e) If PaCO2 still remains elevated at high POWER setting (>8.0), decrease FREQUENCY by 2 Hz every 15-20 min until maximum tidal volume is reached (3-4 Hz at a POWER of 10.0). The lower frequency leads to a longer I.T. which results in a larger tidal volume of gas displaced towards the infant. This increased TV leads to increased alveolar ventilation (on HFOV, $Ve = (TV)^2 f$.

5) Manual Ventilation: Hand bagging while on the SensorMedics Ventilator should be avoided secondary to the risk of barotrauma due to over distention. Suctioning should be performed using just the ventilator breaths alone (an inline suctioning adapter would be best). If bagging has to be done, the PIP while bagging should not exceed 8-10 cm above the MAP and a PEEP of 6-8 cm should be maintained as tolerated.

D. MAP: Oxygenation on HFOV is directly proportional to MAP which is similar to CMV, however with the SensorMedics HFOV all of the MAP is generated by PEEP. Thus during HFOV:MAP=PEEP.

1. Initial Settings:
a) Neonates - Initial MAP should be 2-4 cm above the MAP on CMV.

b) Infants/Children - Initial MAP should be 4-8 cm above the MAP on CMV.

c) If starting immediately on HFOV use a MAP of 8-10 cm in neonates and 15-18 cm in infants/children.

2. Management of ABG's (Oxygenation a MAP):

a) If not oxygenating adequately at initial MAP (12-18 cm) obtain CXR to assess lung volume. If lung is not hyperinflated (flattened diaphragm) or is below optimal lung volume 9-10 ribs then increase MAP by 2-4 cm every 20-30 min until adequate oxygenation is achieved or lung starts to become overinflated (e.g. FiO2 0.6-0.7 increase by 2-4 cm, FiO2 1.0 increase by 4-8 cm).

b) Maximum potential MAP = 40-45 cm

c) Warning - If oxygenating adequately, but the lung is hyperinflated immediately decrease MAP by 1-2 cm every 2-4h until lung volumes return to normal. If the lung is allowed to remain hyperinflated for prolonged periods of time the risk of barotrauma increases dramatically.

d) If not oxygenating with lung becoming hyperinflated, you can decrease frequency as a way to increase I.T. while keeping I:E ratio constant.

II.   MANAGEMENT   STRATEGIES   FOR   THE   SENSORMEDICS   3100: The SensorMedics HFOV is usually used for premature infants, term infants or young children with respiratory failure not responsive to CMV. The Infant Star HFV with its ability to oxygenate using less MAP than the SensorMedics due to the use of an occasional sigh breath (1-4 per min) is also recommended for premature infants with respiratory failure.

A. TERM INFANT WITH SEVERE RESPIRATORY FAILURE (PPHN, MAS, GBS pneumonia, RDS): Start at a frequency of 10 Hz and a Power of 3.0 to 5.0. Initial MAP 4 cm above MAP while on CMV. Check CXR 2 hrs after converting to HFOV, then adjust MAP to achieve optimal lung volume (9-10 ribs expanded). If not oxygenating, increase MAP by 2 cm every hour until oxygenation improves. Adjust Power to keep PaCO2 45-55.

B. AIRLEAKS: Pneumothorax or PIE - The goal is to minimize both tidal volumes and peak pressures generated by any given TV. Tolerate increased FiO2 requirements (0.6 - 1.0) in order to keep MAP at a minimum. Practice permissive hypercarbia and accept high PaCO2's to minimize the TV. Use a FREQUENCY of 12-15 Hz in order to minimize both total I.T. and TV in order to heal the airleak. Also decrease I.T. to 30%.

C. ARDS: The goal is to minimize volutrauma, barotrauma and oxygen toxicity. Thus use the minimum POWER possible at the appropriate FREQUENCY in order to keep PaCO2 adequate (e.g. 55-65 mm Hg). Increase MAP as high as necessary to keep FiO2 < 1.0. Also decrease frequency to increase I.T. to improve oxygenation.

D. RDS: Give surfactant replacement therapy using manual bagging. Start with frequency of 15 Hz, I.T. of 33%. Use MAP of 8-10 cm or 2 cm above MAP on CMV. Wean FiO2 until <0.50 then MAP as tolerated to avoid overinflation. Wean power/amplitude to keep PaCO2 45-60 mmHg. Follow blood gases q30-60 min after surfactant until stable and wean appropriately to avoid hypocarbia.

E. Rescue Therapy For Premature Infant With RDS: To be used for premature neonates who can't ventilate on either CMV or the Infant Star HFV or who require a MAP > 20 cm to achieve oxygenation while on the Infant Star. Use initial frequency of 15 Hz, Power of 3.0 - 4.0, MAP 2 cm above MAP on the Infant Star HFV, or MAP 4 cm above the MAP on CMV.

F. BPD: The goal is to minimize volutrauma, barotrauma, atelectatrauma, biotrauma and oxygen toxicity. Use minimum power/amplitude to keep $PaCO_2$ adequate (e.g., 50-70 mmHg). Increase MAP as necessary to keep FiO_2 <0.50 if possible. Use I.T. of 33%. Use frequency of 10-15 Hz: use the lower frequencies if having difficulty with oxygenation, use the higher frequencies if having problems with hypocarbia or PIE.

G. Other Potential Indications: CHF/Pulmonary Edema, Chest Physiotherapy, Hypoplastic Lungs and Post-op Heart Patients

H. Not Beneficial For Asthma: Increased risk of air trapping with reactive airway disease.

III. WEANING:

A. OXYGENATION: Once oxygenation is adequate and the patient is ready to be weaned follow these steps:

1) First only wean FiO_2 until < 0.60 unless hyperinflated.

2) Once FiO_2 < 0.60 or hyperinflated, decrease MAP by 1 cm Q4-8h; if OXYGENATION is lost during weaning then increase MAP by 3-4 cm to restore lung volumes and begin weaning again, but proceed more slowly with decreases in MAP.

3) Minimal MAP is 8-16 cm with FiO_2 < 0.50, at this point one can convert to CMV or remain on HFOV while the patient continues to heal (e.g., 8-12 cm < 2.5 kg, 13-16 cm > 2.5 kg).

B. VENTILATION: Reduce POWER by 0.2-0.3 units per change whenever $PaCO_2$ decreases below threshold, until minimal POWER is reached (1.5-4.0) depending on the size of the patient. If frequency is below the standard frequency for the patient's weight, then first wean by increasing frequency back to baseline which also decreases the tidal volume, then decrease power as described.

1) Extubation - Neonates are ready to be extubated for a trial of NPCPAP when they meet the following criteria:
a) MAP < 10 cm, FiO_2 ≤ 0.40 and power < 2.5 then use NPCPAP of 7-9 cm.

2) Conventional Ventilation - Term neonates are ready for conversion to conventional mechanical ventilation (IMV or SIMV) when they meet the following criteria:

a) MAP < 16-17 cm, FiO_2 < 0.40 - 0.45 and power < 4.0

b) To convert to conventional mechanical ventilation (CMV) use MAP 3-4 cm less than the MAP on HFV [e.g., MAP = 16-17 on HFV, use a MAP of 12-13 on CMV (PIP = 26, PEEP = 8, Rate = 40, IT = 0.4)]

III. COMPLICATIONS ASSOCIATED WITH HFOV:

A. Hyperinflation or Barotrauma: increased MAP

B. Secretions: a suctioning

C. Hypotension: increase MAP, and rule out other causes (e.g., pneumothorax, sepsis, dehydration, etc.)

Respiratory monitoring on ventilator

Rachna Sharma

Why monitor?

Monitoring of the respiratory indices has the potential for predicting catastrophies and providing an opportunity for the timely institution of lifesaving measures. Along with physical examination which remains clinically relevant, non invasive (pulse oximetry and end-tidal CO_2) and invasive(Arterial blood gas and derived indices) monitoring is important and routinely done in most PICUs. The appropriate integration and interpretation of all data are essential for efficient, high-quality, cost-effective, pediatric critical care.

Physical Examination

Initial assessment begins by observing the child's position (will assume a most comfortable posture like "sniffing position" during upper airway obstruction or splinting of the chest in pts of pneumonia), respiratory pattern and body habitus (shape of the chest wall). The respiratory pattern will provide information about respiratory rate(tachypnea: an early sign of distress) which varies with age(table-1), increased work of breathing which can be assessed by the presence grunting, flaring of alae nasi, suprasternal, intercostal and subcostal retractions, use of accessory muscles of respiration and paradoxical breathing.

Evaluation of cyanosis, clubbing, friction rub, breath sounds (wheezes and crackles) through the tracheobronchial tree are reliable and reproducible.

Normal Respiratory Rate (Table1)

Age	Respiratory rate
Infant (birth–1 year)	30-60
Toddler (1–3 years)	24-40
Preschooler (3–6 years)	22-34
School-age (6–12 years)	18-30
Adolescent (12–18 years)	12-16

Radiography

A very commonly ordered investigation in PICU which has diagnostic, therapeutic and prognostic value is x-ray chest

Non Invasive Monitoring

Transcutaneous oxygen and carbondioxide monitoring

Transcuataneous measurements reflect both gas exchange and skin perfusion. in this technique , a probe composed of heater , an electrode , and a thermistor is applied to the patients skin> The skin is warmed and softened toimprove diffusion and permeability. This also causes capillaries to dilate, resulting in better approximation of arterial oxygen values.

Limitations:

Several disadvantages limit the use of transcutaneous monitoring to the newborn population. Skin thickness increases with age , making transcuataneous measurements less predictable. Frequent electrode site are changes are required to prevent local burns. relatively frequent calibration and comparison with arterial blood gases are necessary.

Pulse oximetry - Pulse oximetry is an important noninvasive monitoring technique that allows continuous evaluation of arterial oxygen saturation *and now widely accepted as the fifth vital sign*. The two basic requirements of commercially available pulse oximeters are the presence of a pulsatile tissue bed (arterial vessel) and the spectrophotometric analysis(governed by **Beer-Lambert law)** of oxygenated hemoglobin and nonoxygenated hemoglobin. Oxygenated hemoglobin primarily absorbs infrared(940nm) light, whereas nonoxygenated hemoglobin primarily absorbs red (660nm) light. A microprocessor in the pulse oximeter determines the relative proportions of red and infrared light to calculate the percentage of oxygenated versus nonoxygenated hemoglobin in the tissue bed. In addition to the digital readout of O_2 saturation and pulse rate, most pulse oximeters display a plethysmographic waveform which can help clinicians to distinguish an artifactual signal from the true signal.

Factors That Affect the Performance of Pulse Oximetry

- Poor cardiac output/low perfusion states- Low perfusion states, such as low cardiac output, vasoconstriction and hypothermia may impair peripheral perfusion and made it difficult for the pulse oximeter sensor to distinguish true signal from background;
- Motion artifact - Excessive motion of the photosensor causes intermittent contact with the skin and mechanically modulates the path length of the transmitted light and the amplitude and intensity of the received light , producing spurious saturation values.
- Optical interference from environment - Falsely low and high SpO_2 readings occur with fluorescent and xenon arc surgical lamps, bilirubin lights, infrared heating lamps, and direct sunlight.
- Dyshemoglobinemias: carbon monoxide, methemoglobinemia, fetal hemoglobin- Since pulse oximeters use only two wavelengths of light and, thus, it can distinguish only two substances, Hb and HbO_2. When Carboxyhemoglobin (COHb) and methemoglobin (MetHb) are also present, four wavelengths are required to determine the 'fractional SaO_2': i.e., $(HbO_2 \times 100)/ (Hb + HbO_2 + COHb + MetHb)$ and this can be measured by Co-oximetry. In the presence of elevated COHb levels, oximetry consistently over- estimates the true SaO_2 by the amount of COHb present since it has got same absorption spectrum as of HbO_2. Elevated MetHb levels also may cause inaccurate oximetry readings. Anemia does not appear to affect the accuracy of pulse oximetry even in non-hypoxemic patients with acute anemia; pulse oximetry was accurate in measuring O_2 saturation. Severe hyperbilirubinemia (mean bilirubin, 30.6 mg/dl) does not affect the accuracy of pulse oximetry.
- Dyes and pigments: Intravenous dyes such as methylene blue, indocyaninegreen, and indigocarmine can cause falsely low SpO_2readings. Nail polish, if blue, green or black, causes inaccurate SpO_2 readings, whereas acrylic nails do not interfere with pulse oximetry readings.

It is important to remember that pulse oximeters assess oxygen saturation only and thereby Oxygenation status and gives no indication of the level of CO_2 and thereby Ventilation status. For this reason they have a limited benefit in patients developing respiratory failure due to CO_2 retention.

The pulse oximeter may be used in a variety of situations that require monitoring of oxygen status and may be used either continuously or intermittently. It is not a substitute for an ABG,

but can give clinicians an early warning of decreasing arterial oxyhemoglobin saturation prior to the patient exhibiting clinical signs of hypoxia. The pulse oximeter is a useful tool but the patient must be treated--not the numbers. As with all monitoring equipment, the reading should be interpreted in association with the patient's clinical condition.

Masimo pulse oximetry - a new promising way of measuring SpO2...!!

What makes Masimo pulse oximetry different from conventional pulse oximetry?

Conventional pulse oximetry assumes that arterial blood is the only blood moving (pulsating) in the measurement site. During patient motion, the venous blood also moves, which causes conventional pulse oximetry to under-read because it cannot distinguish between the arterial and venous blood. Masimo signal technology identifies the venous blood signal, isolates it, and cancels the noise and extracts the arterial signal, and then reports the true arterial oxygen saturation and pulse rate.

Following setbacks of Conventional Pulse Oximetry for inaccurate monitoring or signal dropout during the reading are rectified by Masimo technology

> Patient Motion or Movement
> Low Perfusion (low signal amplitude)
> Intense Ambient Light (lighting or sunlight)
> Electrosurgical Instrument Interference

Capnography (End Tidal CO_2 Monitoring)

Capnography is a noninvasive monitoring tool that measures CO_2 concentration in exhaled gas, displayed continuously as a wave form through the respiratory cycle. Typically, this measurement is made using infrared light absorption based on the concept that CO_2 strongly absorbs infrared light with a wavelength of 4280 µm.

Normal Capnogram (fig 3)

The Capnogram is c

1. Phase I (most of the anatomical dead spa
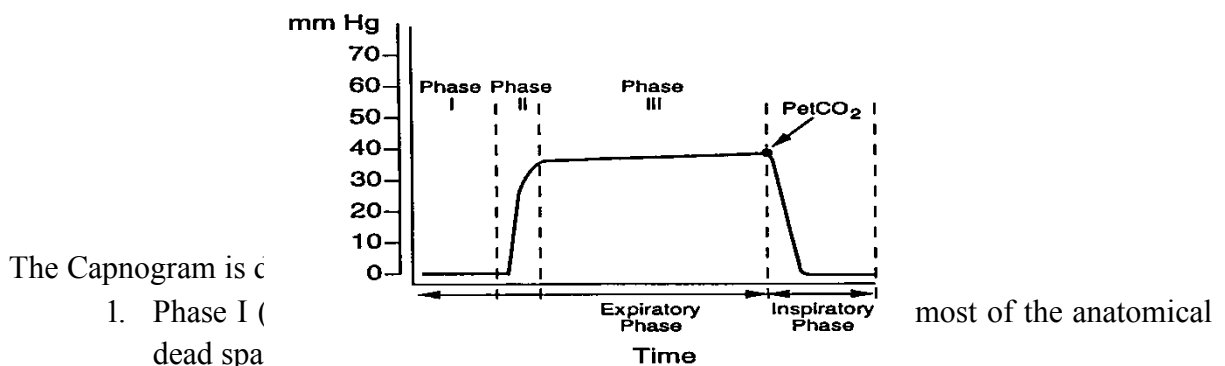
2. Phase II (B-C) is where the alveolar gas begins to mix with the dead space gas and the CO_2 begins to rapidly rise.

3. Phase III (C-D) represents the alveolar gas, usually has a slight increase in the slope as "slow" alveoli empty. The "slow" alveoli have a lower V/Q ratio and therefore have higher CO_2 concentrations. In addition, diffusion of CO2 into the alveoli is greater during expiration. *This is more pronounced in infants.* EtCO2 is measured at the maximal point of Phase III...... (D)

4. Phase IV (D-E) is the inspirational phase

Note that the presence of the alveolar plateau confirms that the measurement is End-tidal. Without a Capnography you cannot be sure that a measured CO_2 value is really end-tidal.

A normal value for ETCO2 is approximately 38-40 mm Hg. Under normal conditions, the end tidal CO_2 is usually slightly less than the $PaCO_2$, with a normal difference of 2–5 mm/Hg. Note that this gradient may be considerably higher in situations where there is an increase in dead space. Sampling of exhaled carbondioxide can be at the patient - ventilator interface (mainstream) , diverted to a monitor (side stream0 , or an intermediate connection. Sidestream canbe used with in intubated or non-intubated patients thus have wider applications but it is less accurate at higher respiratory rates.
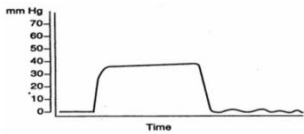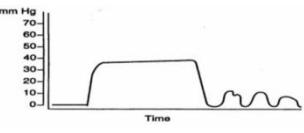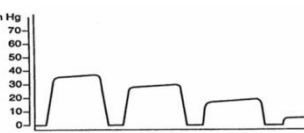
Indications for Capnography are:
1. Confirm and verify tracheal intubation placement.
2. Evaluate ventilator settings and circuit integrity.
3. Assess cardiopulmonary status and changes in pulmonary blood flow.
4. Assess airway management and changes in airway resistance.
5. Monitor effectiveness of CPR.
6. Monitor ventilatory status of the respiratory impaired patient.
7. Monitor ventilation of a nonintubated patient during sedation/analgesia.
8. Monitor the effectiveness of ventilator weaning process, and response to
changes in ventilator settings (i.e., respiratory rate, flow and/or volume).
9. Reduce the number and/or frequency of arterial blood gas drawings.
10. Aids in the treatment of neurological patients and the possibility of increasing intracranial pressures.
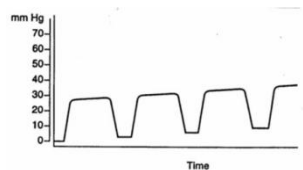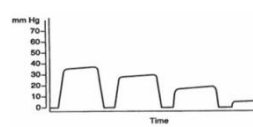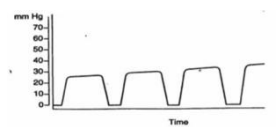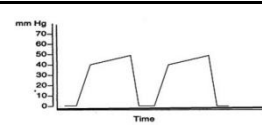
Other uses
- Metabolic
  - Assess energy expenditure
- Cardiovascular
  - Monitor trend in cardiac output
  - Can use as an indirect Fick method, but actual numbers are hard to quantify
  - Measure of effectiveness in CPR
  - Diagnosis of pulmonary embolism by measuring measure gradient

Differential Diagnosis of Abnormal Capnogram (Table)

Symptom	Possible Cause	
Sudden drop of EtCO2 to zero	Esophageal intubation Ventilator disconnection or malfunction Defect in CO2 analyzer Dislodged OR obstructed endotracheal tube	
Sudden fall of EtCO2 (not to 0)	Leak in ventilator system, obstruction Partial disconnect in ventilator circuit Partial airway obstruction (secretions)	
Exponential fall of EtCO2	Cardiac Arrest Hypotension (sudden) Severe hyperventilation Cardiopulmonary bypass	

	Pulmonary Embolism	
Change in CO_2 Baseline	CO2 absorber saturation (anesthesia) Calibration error Water droplet in analyzer Mechanical failure (ventilator)	
Gradual lowering of $EtCO_2$	Hypovolemia Decreasing Cardiac Output Decreasing body temperature, hypothermia, drop in metabolism	
Gradual increase in $EtCO_2$	Rising body temperature Hypoventilation CO_2 absorption Partial airway obstruction (foreign body), reactive airway disease	
Constantly high $EtCO_2$	Respiratory depression duo to drugs Metabolic alkalosis (respiratory compensation) Insufficient minute ventilation	

Limitations:

1. Critically ill patients often have rapidly changing dead space and V/Q mismatch
2. Higher rates and smaller tidal volumes can increase the amount of dead space ventilation
3. High mean airway pressures and PEEP restrict alveolar perfusion, leading to falsely decreased readings
4. Low cardiac output will decrease the reading

Microstream technology

It is 3rd generation technology which can be used with intubated or non-intubated patients and requires low sample flow rate - 50 ml/min. It allows its use in neonate & pediatric patients. In this technology sampling lines not flooded with moisture. Microstream improves upon conventional Sidestream sampling based upon the principle that CO_2 molecules absorb IR radiation at specific wavelengths

Advantages

1. No sensor at airway
2. Intubated and non-intubated patients (neonatal through adult)
3. No routine calibration
4. Automatic zeroing
5. Accurate at small tidal volumes and high respiratory rates
6. Superior moisture handling

INVASIVE MONITORING

Arterial blood gas monitoring

Evaluation of arterial blood gas provides information on the uptake of oxygen and disposal of carbondioxide by the lung. In diseased lungs, indexes of oxygenation have been developed that use the data obtained from a blood gas to better define the effeciency of gas exchange and the causes of hypoxemia.

Let's elaborate now, how to determine oxygenation, and then evaluate the acid-base status systematically.

Oxygen Homeostasis

The most important interpretation in ABG is to look for the adequacy of oxygenation status. The normal partial pressure of O_2 in the arterial blood PaO_2 is 80-100 mm of Hg on breathing at atmospheric oxygen (which contains 21% of oxygen = FiO_2 21%). When the fraction of the inspired O_2 (FiO_2) concentration increases, the arterial PaO_2 increases by five times (PaO_2 = $Fio2 \times 5$). When PaO_2 falls below 80 mm of Hg ,**hypoxemia** results which can be classified as either mild, PaO2 of 60-80 mmHg, moderate Pao2 of 50-60 mmHg or severe PaO2 < than 50 mmHg. This arterial inspired oxygen concentration ratio (Pao2/fio2) is the easiest index to calculate and it also is the basis for the definition of acute lung injury/acute respiratory distress syndrome (ARDS).

The alveolar-arterial (A-a) O2 gradient, PAO2 - PaO2 helps to differentiate hypoxemia caused by hypoventilation from diffusion abnormalities , ventilation/perfusion mismatch, or shunt. Unlike oxygen (for which alveolar concentrations are higher than arterial concentrations) CO_2 freely diffuses across the lung such that the arterial and alveolar concentrations are identical. As a patient hypoventilates, CO_2 will accumulate in the body (more CO_2 is produced through metabolism than can be eliminated) and thus in the blood (where we measure it as $PaCO_2$). The carbon dioxide displaces the oxygen in the alveolus. It does require PAO2,which is difficult to measure. A normal A-a gradient is 10-20 mm Hg, with the normal gradient increasing within this range as the patient ages. An increased A-a gradient identifies decreased O_2 in the arterial blood compared to the O_2 in the alveolus. This suggests a process that interferes with gas transfer, or in general terms, suggests ventilation- perfusion mismatch. A normal A-a gradient in the face of hypoxemia suggests the hypoxemia is due to hypoventilation and not due to underlying lung disorders.

A-a gradient = $\{(FIO_2) (760-47) - (1.25) (PaCO_2)\}$ - PaO_2

Where does 1.25 come from?

This is a fudge factor which is derived from the respiratory quotient. The formula actually requires that the $PACO_2$ be divided by the respiratory quotient, which is defined as the ratio of CO_2 produced to O_2 consumed (and which depends on diet and metabolism). We estimate the RQ to be 0.8, and the reciprocal of 0.8 is 1.25

Stepwise Approach to Diagnosing Acid-Base Disorders

Interpreting an arterial blood gas (ABG) is a crucial skill for physicians, nurses, respiratory therapists, and other health care personnel. ABG interpretation is especially important in critically ill patients. The following six-step process helps ensure a complete interpretation of every ABG. **Normal values of ABG……………………………**

Step 1: Assess the internal consistency of the values using the Henderseon-Hasselbach equation:

$[H+] = \dfrac{24(PaCO_2)}{[HCO_3-]}$

Step 2: Is there alkalemia or acidemia present?

pH < 7.35 acidemia

pH > 7.45 alkalemia

This is usually the primary disorder. *Remember:* an acidosis or alkalosis may be present even if the pH is in the normal range (7.35 – 7.45).You will need to check the $PaCO_2$, HCO_3- and anion gap

Step 3: Is the disturbance respiratory or metabolic? What is the relationship between the direction of change in the pH and the direction of change in the $PaCO_2$? In primary respiratory disorders, the pH and PaCO2 change in *opposite* directions; in metabolic disorders the pH and $PaCO_2$ change in the same direction.

Acidosis	Respiratory	pH ↓	$PaCO_2$ ↑
Acidosis	Metabolic&	pH ↓	$PaCO_2$ ↓
Alkalosis	Respiratory	pH ↑	$PaCO_2$ ↓
Alkalosis	Metabolic	pH ↑	$PaCO_2$ ↑

Step 4: Is there appropriate compensation for the primary disturbance? Usually, compensation does not return the pH to normal (7.35 – 7.45).

Disorder	Expected compensation	Correction factor
Metabolic acidosis	$PaCO_2 = (1.5 \times [HCO_3-]) +8$	± 2
Acute respiratory acidosis	Increase in $[HCO_3-]= \Delta\ PaCO_2/10$	± 3
Chronic respiratory acidosis (3-5 days)	Increase in $[HCO_3-]= 3.5(\Delta\ PaCO_2/10)$	
Metabolic alkalosis	Increase in $PaCO_2 = 40 + 0.6(\Delta HCO_3-)$	
Acute respiratory alkalosis	Decrease in $[HCO_3-]= 2(\Delta\ PaCO_2/10)$	
Chronic respiratory alkalosis	Decrease in $[HCO_3-] = 5(\Delta\ PaCO_2/10)$ to $7(\Delta\ PaCO_2/10)$	

If the observed compensation is not the expected compensation, it is likely that more than one acid-base disorder is present.

Step 5: Calculate the anion gap (if a metabolic acidosis exists):

$AG = [Na+] - ([Cl-] + [HCO_3-]) - 12 \pm 2$

- A normal anion gap is approximately 12 meq/L.
- In patients with hypoalbuminemia, the normal anion gap is lower than 12 meq/L; the "normal" anion gap in patients with hypoalbuminemia is about 2.5 meq/L lower for each 1 gm/dL decrease in the plasma albumin concentration (for example, a patient with a plasma albumin of 2.0 gm/dL would be approximately 7 meq/L.)
- If the anion gap is elevated, consider calculating the osmolal gap (OSM) in compatible clinical situations.
    - Elevation in AG is not explained by an obvious case (DKA, lactic acidosis, renal failure, Toxic ingestion is suspected)

$OSM\ gap = measured\ OSM - (2[Na+] - glucose/18 - BUN/2.8$

- The OSM gap should be < 10

Step 6: If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in [HCO$_3$-]. Assess the ratio of the change in the anion gap (ΔAG) to the change in [HCO3-] (Δ[HCO$_3$-]): $\Delta AG/\Delta$[HCO$_3$-]

This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present. If this ratio falls outside of this range, then another metabolic disorder is present:

- If $\Delta AG/\Delta$[HCO$_3$-] < 1.0, then a concurrent non-anion gap metabolic acidosis is likely to be present.
- If $\Delta AG/\Delta$[HCO$_3$-] > 2.0, then a concurrent metabolic alkalosis is likely to be present.

It is important to remember what the expected "normal" anion gap for your patient should be, by adjusting for hypoalbuminemia (see **Step 5**, above.)

SUMMARY

Respiratory monitoring is an essential tool in the care of critically ill pediatric patients. With the physical examination, radiographic studies, blood gas analysis, pulse oxymetry, capnography and respiratory mechanics - the deleterious effect of respiratory distress in the pediatric population can be greatly ameliorated. It is important, therefore, that such tools are used carefully, with their limitations in mind, and in the light of other findings.

Monitoring of neonates on ventilator

Kumar Ankur, Sanjeev Chetry,

The aim of monitoring is simple and clear to follow in real time specific physiological values that can change rapidly and alter the patient's clinical status. Especially in the intensive care setting, in which the vast majority of patients are admitted because of a primary respiratory problem or of respiratory complications during their illness, monitoring of the cardiorespiratory system alerts the clinician to sudden untoward events, aids in diagnosis, helps manage diagnosis, facilitates prognosis, and enables assessment of therapeutic response.

On routine NICU round daily monitoring includes the following things

1. Clinical monitoring
2. Non invasive monitoring
3. Invasive monitoring

Clinical monitoring:

- o Physical examination:
  - − HR, RR, WOB (work of breathing), SPO2, perfusion, temperature etc
  - − Quantify WOB (downe score or silverman score)

Downe score for assessment of severity of respiratory distress			
	Score 0	Score 1	Score 2
Cyanosis	No cyanosis	Cyanosis on FiO2<40%	Cyanosis on FiO2>40%
RR	<60	60-80	>80
S/C retractions	No	Mild	severe
Air entry	Normal	Diminished	absent
Grunting	No grunting	Audible with stethoscope	Audible without stethoscope

Non invasive monitoring:

1. Pulse oximetry
2. Transcutaneous –Tc pO_2/TcP pCO_2
3. ET CO_2-Capnography
4. Pulmonary graphics
5. ECG
6. Apnea monitor- chest wall movement
7. CXR/Cold Light for air leaks

Invasive monitoring:

- Blood gas-arterial, venous, capillary

Capillary blood gas:

The capillary blood is sampled by heel stab. The method of arterialization is heel warming, usually by immersing the heel in warm water (40°C to 45°C) for five to 10 minutes prior to heel stab, or using a warmed towel or surgical gloves(with hot water).

– There is clinically acceptable agreement between capillary and arterial **pH & pCO₂.** With regard to pO_2 values significantly lesser than arterial blood.

Venous:

– **Central Venous:** in comparison with arterial blood gas, pH 0.03 to 0.05 pH units lower & the PCO_2 is 4 to 5 mmHg higher, with little or no increase in serum HCO_3.

– **Peripheral Venous:** in comparison with arterial blood gas, pH is 0.02 to 0.04 units lower than the arterial pH, HCO_3 is 1 to 2 meq/L higher, and the PCO_2 is 3 to 8 mmHg.

Pulse oximeter:

In premature neonates, oxygen toxicity is associated with the development of retinopathy of prematurity (ROP) and bronchopulmonary dysplasia (BPD). Reducing the levels and time of oxygen exposure in this patient population will likely decrease these morbidities.

The goal of SpO2 level targeting for premature neonates is to adequately deliver oxygen to the tissue without causing the complications of oxygen toxicity. Premature neonates' SpO2 readings are often labile and difficult to keep within a narrow range. The care-team must aim to maintain SpO2 levels within this range.

> - *For the high risk premature neonate the SpO2 target range is 88-92%, with monitor alarm limits set at 85% and 94%.*
> - *For neonates in the high risk group that are in air, the upper alarm limit can be set at 100%*

Permissive Hypercapnia:

Ventilator–induced lung injury remains an important cause of neonatal morbidity. Permissive hypercapnia is a ventilatory strategy that may reduce injury to the developing lung through a variety of mechanisms.

> - *Baby stable on ventilator. CXR: good FRC. ABG: pH: >7.2, pCO_2-55 to 65 mm.*
> - *Because if will target to correct this, that means you have to increase pressure which may lead to more tidal volume delivery & can cause volutrauma.*

PULMONARY GRAPHICS

Sanjeev Chetry, Anup Thakur, Vivek Choudhury

INTRODUCTION

The use of neonatal bedside real-time pulmonary graphic monitoring has been recently added to neonatal ventilation to optimize ventilation and to seek and notice the changes in disease pathophysiology. Till late 90's to early 21st century the assessment of the appropriateness of neonatal invasive ventilation was determined subjectively by noting colour, observing chest excursions, and listening to breath sounds, and objectively by intermittent assessment of gas exchange and radiography when pulmonary mechanics technology was finally made available in the neonatal intensive care unit (NICU). The objective was to

1. Adjust the ventilator parameters to achieve optimal ventilation and oxygenation by altering
    1. Peak inspiratory pressure (PIP)
    2. Positive end expiratory pressure (PEEP)
    3. Inspiratory and expiratory tidal volume (VTI or VTE)
    4. Inspiratory time (TI)
    5. Expiratory time (TE)
    6. Flow rate
    7. Synchronization
    8. Compliance

2. Assess disease.

3. Evaluate response to treatments such as bronchodilators, surfactant diuretics,steroids.

To achieve this end initially, pneumatocograph was brought whose major disadvantage was that it provided snap shots of the events rather than a continuous process. In addition, the instrument introduced significant dead space and increased the work of breathing in neonates. This disadvantage was overcome with real time pulmonary graphics.

Real-time pulmonary graphics are the graphical display of measured and derived values captured during the process of mechanical ventilation. These visual representations of the interaction between the mechanical ventilator and the baby receiving support for respiratory failure are critical in understanding both the support used and its effectiveness.

PRINCIPLE

With the development of sensor technology real time pulmonary graphics was brought to NICUs where a microprocessor-based technology is integrated to the intended function of the ventilator.

The sensor technologies fall into one of two categories: thermal or differential pressure type. The sensor detects either flow or pressure and converts the signal to a clinically useful analogue value. For example, the flow signal can be integrated to obtain a volume measurement. The sensor also is used to detect patient effort to facilitate or ''trigger'' synchrony between the patient's own effort and the delivery of a mechanical breath by the ventilator. The information is presented in real-time and is a continuous display

ADVANTAGES

Graphic monitoring assists the clinician at the bedside in several ways.

1. It can be helpful in fine-tuning or adjusting ventilator parameters.
2. Its monitoring may help to determine the patient's response to pharmacologic agents such as surfactant, diuretics, or bronchodilators.
3. The clinician also has the ability to trend monitored events over a prolonged period of time.

DISADVANTAGES

1. The understanding of graphic monitoring may at times be considered complex.

2. There are many clinical situations that may be identified at the bedside and each patient is different and provides unique learning experiences.

BASICS OF PULMONARY GRAPHICS

The three major wave forms are pressure, volume, and flow. These wave forms are displayed versus time

Pressure wave form
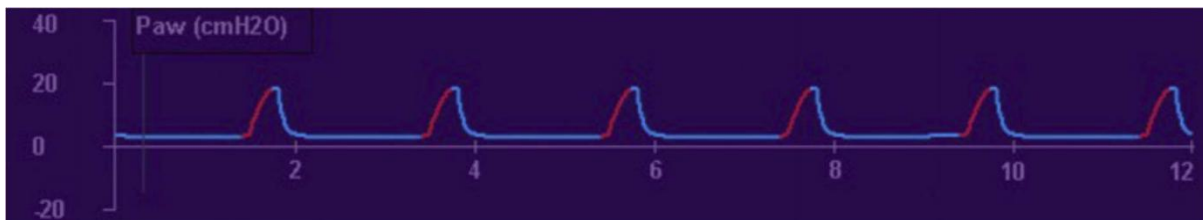
The pressure wave form has upward (inspiration) and downward (expiration) scalars. PEEP is the baseline pressure level. If PEEP is used, the wave form will begin and end at this value and not reach zero. The uppermost point of the wave form represents PIP, whereas the area under the curve is the mean airway pressure. The inspiratory time can be measured from the point of upward deflection until PIP is reached; expiratory time begins at PIP and lasts until the next positive deflection. The shape of the curve represents the breath type, e.g., volume (triangular) or pressure (square).
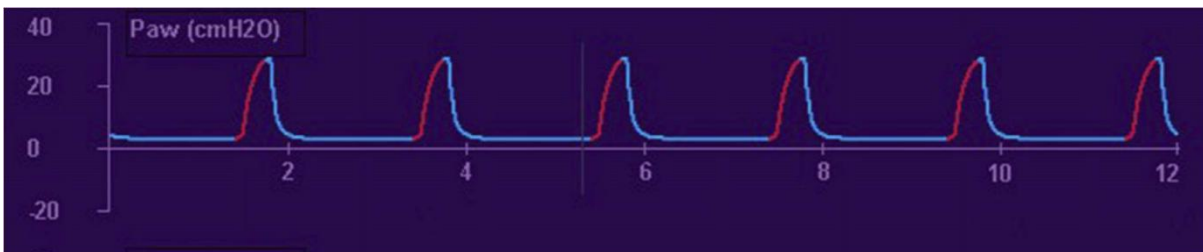
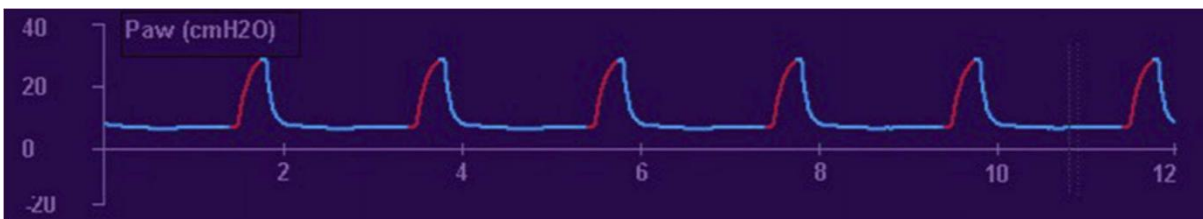Oxygenation is a function of mean airway pressure.
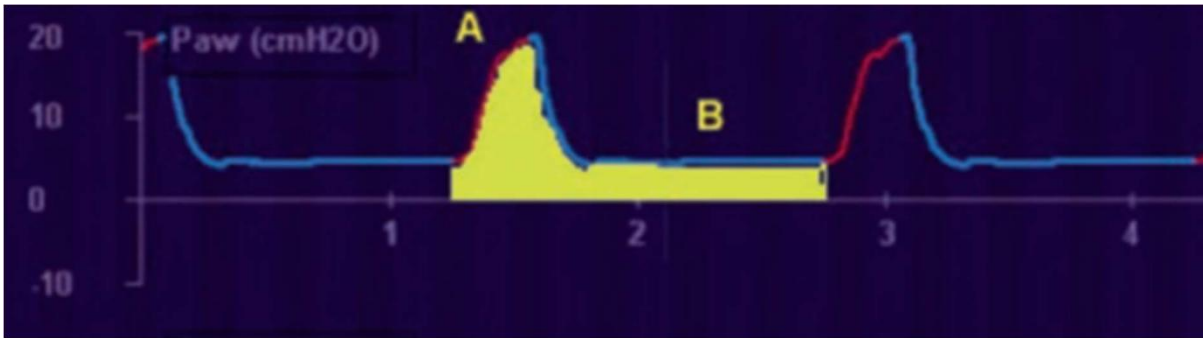
Graphic display of Mean Airway pressure

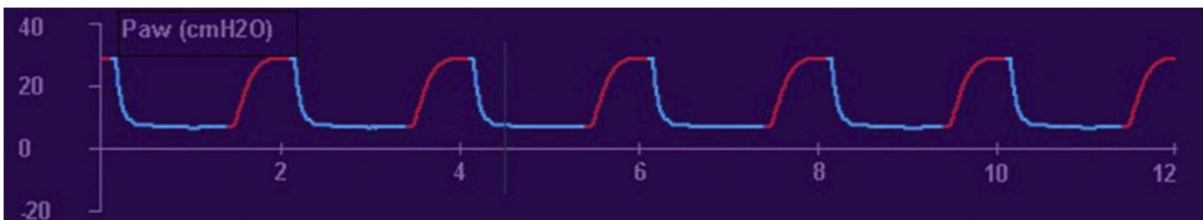Thus, increasing the area under the curve will improve oxygenation. This can be accomplished by increases in
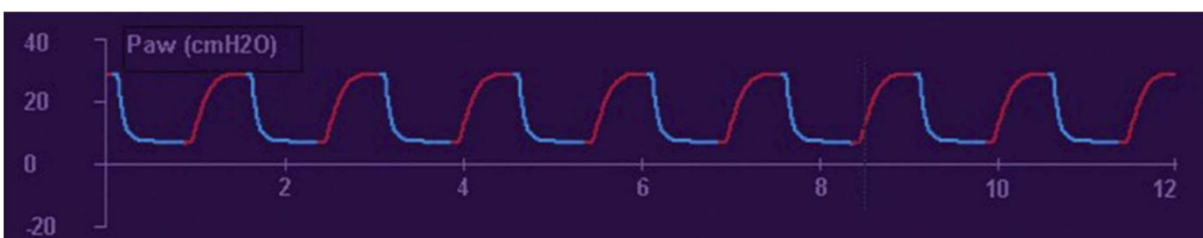
1. PIP
2. PEEP
3. Inspiratory time
4. Rate

1. MAP increased by increasing PIP


2. MAP increased by increasing PEEP

3. MAP increased by increasing inspiratory time.

4. MAP increased by increasing rate

Ventilation is a function of tidal volume and frequency. The primary determinant of tidal volume is amplitude, the difference between PIP and PEEP, often referred to as Delta P(DP). So, ventilation can be enhanced by changes in amplitude (higher PIP, lower PEEP, or both) and increases in ventilator frequency and/or expiratory time

Flow wave form
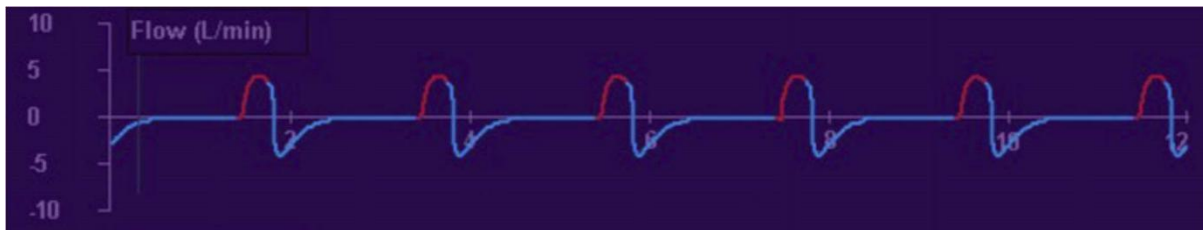
The flow wave form has two separate components. Anything above the zero baseline represents positive flow, or in other words, gas flow into the patient(inspiration). Inspiratory flow has two components: accelerating flow (at the start of inspiration), and decelerating flow (velocity slows as the lung approaches capacity). The highest point of the positive point of the wave form is peak inspiratory flow. Anything below the zero baseline represents negative flow, or gas flow from the patient(expiration). The expiratory flow wave form similarly has two components: accelerating flow (at the start of expiration), and decelerating flow (velocity slows as the lung empties to functional residual capacity). The lowest negative point of the wave form is peak expiratory flow.
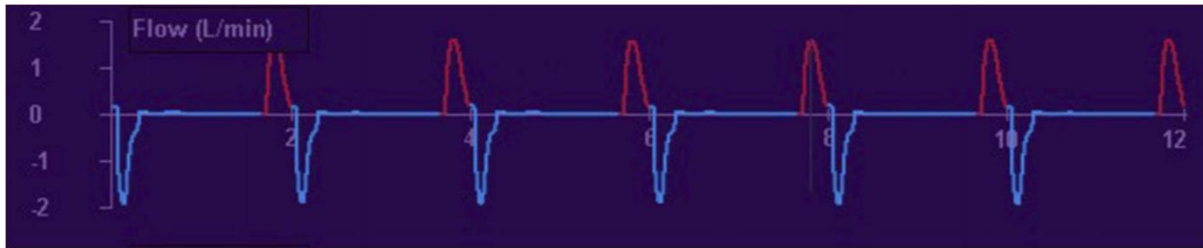
1. The flow waveform can help to detect air trappings.
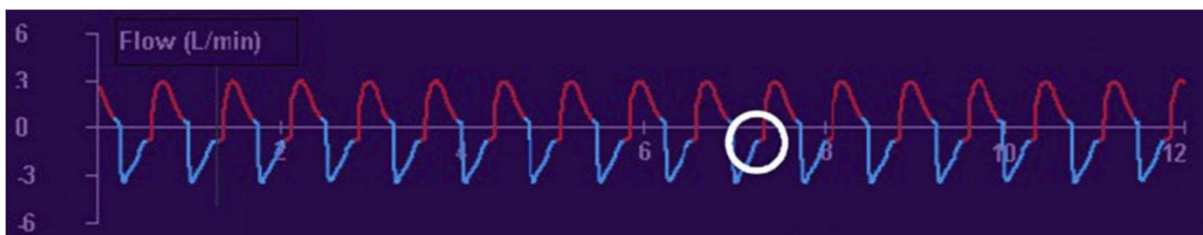
Flow waveform showing air trappings. The decelerating expiratory limb fails to reach the baseline before the next breath begins (circled), preventing complete emptying of the lung.
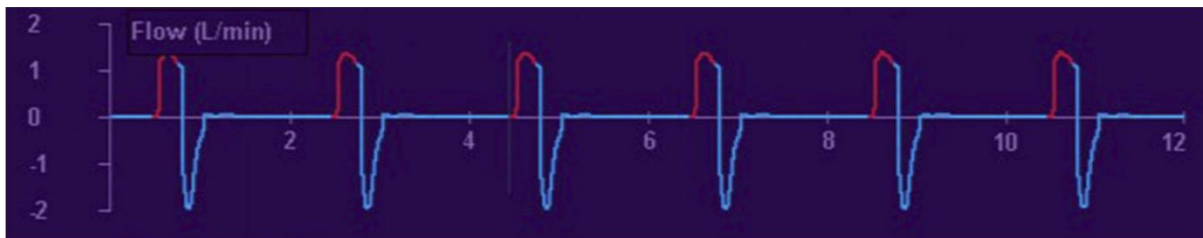
2. The flow wave form may help distinguish breath types.

Pressure-targeted ventilation produces a spiked or sinusoidal wave form.

Flow waveform showing sinusoidal pressure wave form is characteristic of pressure-targeted ventilation

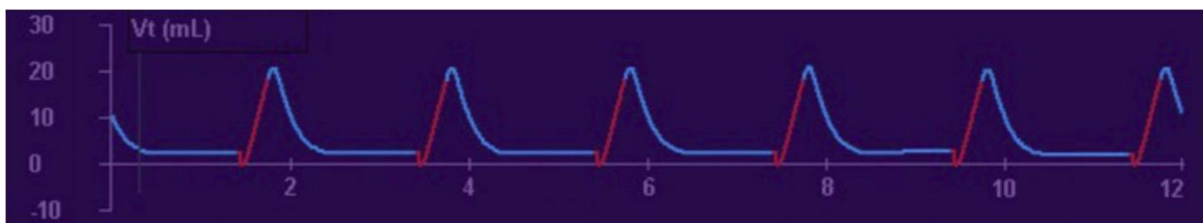Volume-targeted ventilation produces a characteristic square wave whereby flow plateaus and is held constant

Volume-targeted ventilation produces a square wave, with a flow plateau.
Flow waveform can also demonstrate an endotracheal tube leak.
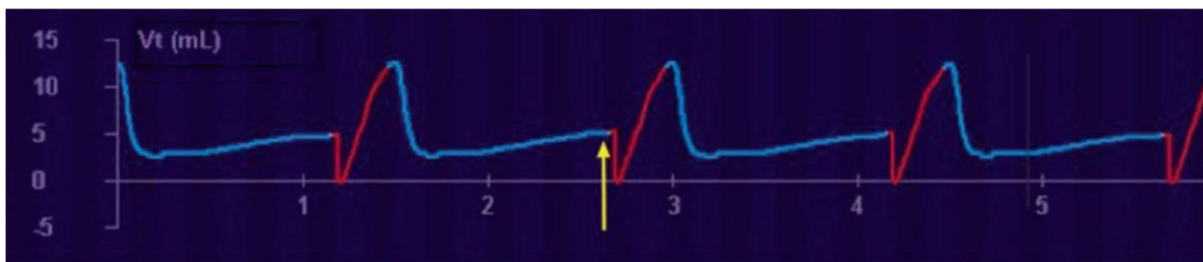
The expiratory flow does not completely return to baseline before the next breath
**Volume wave form**

The volume wave form is similar in appearance to the pressure wave form, except that it starts and end on the baseline. The shape of the pressure wave form demonstrates how volume is delivered to the baby. During pressure-targeted ventilation, peak volume delivery occurs early in inspiration, then decreases. This is in contrast to volume-targeted ventilation, which creates a ''shark's fin'' pressure wave form, whereby peak volume delivery occurs at the end of inspiration. Leaks may be suspected by looking at the volume wave form, whereby the expiratory portion also fails to reach the baseline.

Turbulence can be be detected in all these waveforms and is most commonly due to secretions in the tube.
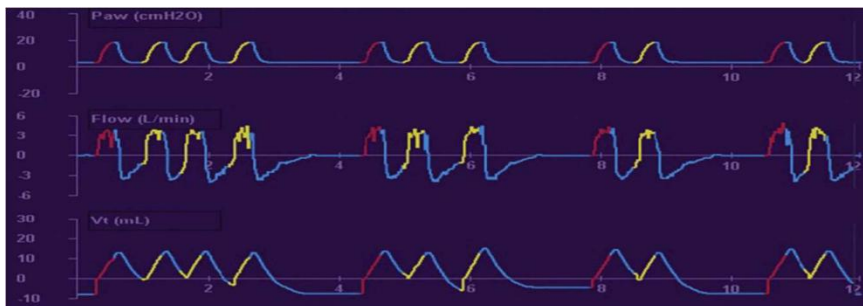
Irregular waveforms indicating turbulence

The pulmonary waveforms(all three forms) also indicates autocycling as the graph below shows
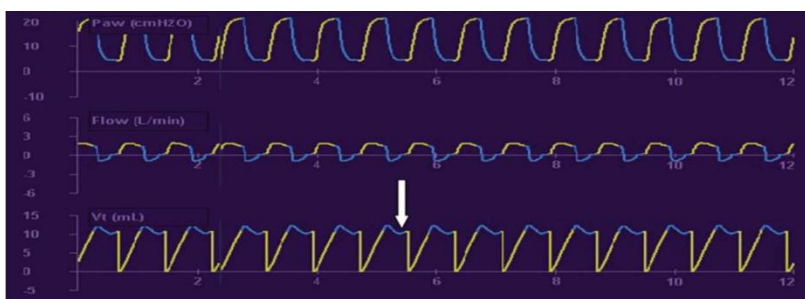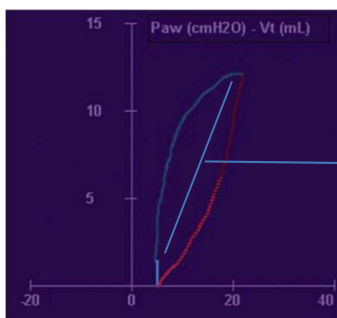

In autocycling the rhythmic breaths comes without a pause as well as the large leak

Loops

Pulmonary mechanics can also be assessed when changes in pressure versus volume or flow versus volume are graphed over time and these are called loops.
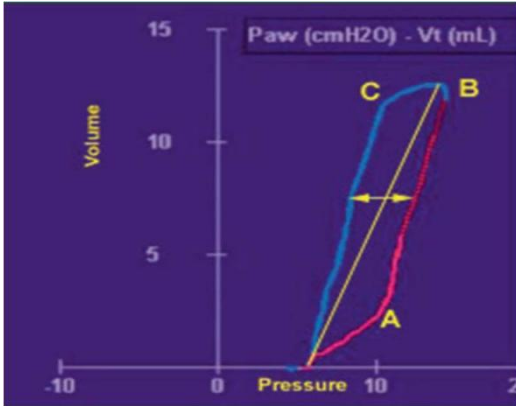
**Pressure volume loop**

Pressure–volume loop

Pressure–volume loop (*P-V* loop) displays the relationship of pressure to volume. Pressure is displayed along the horizontal axis and volume is displayed on the vertical axis. Inspiration is represented by the up-sweep from the baseline (PEEP) terminating at PIP. Expiration is the down-sweep from PIP back to baseline. The shape of this loop is referred to as hysteresis. A line drawn from each endpoint represents pulmonary compliance.

The *P-V* loop may be used to assess adequacy of PEEP, used to maintain end-expiratory lung volume. If the inspiratory limb of the *P-V* curve demonstrates a lower inflection point, identifying opening pressure, PEEP is inadequate.

PEEP assessment using the P-V loop

The *P-V* loop may help identify lung overdistension. If the inspiratory limb flattens at the top, this indicates pressure exposure without further volume delivery.
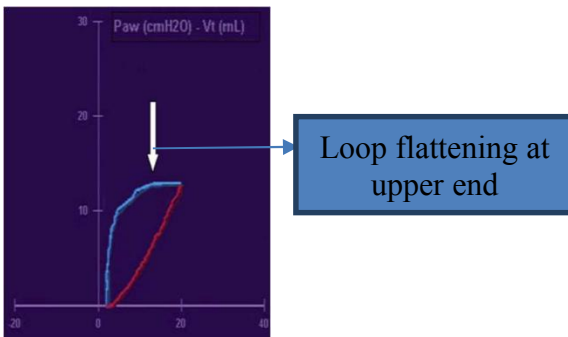
Penguin beak appearance

P-V loops can help evaluate whether flow delivery from the ventilator is adequate to meet the needs of the patient. Inadequate flow is represented by cusping of the inspiratory portion of the curve. Severe flow limitation may appear as a "figure-8" on the *P-V* loop.
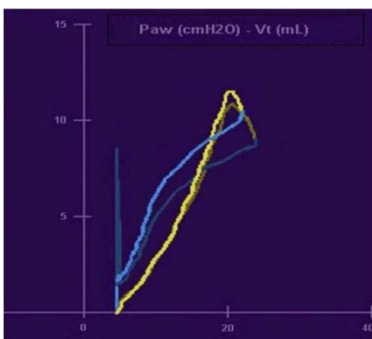

Figure of 8 appearance indicating air hunger
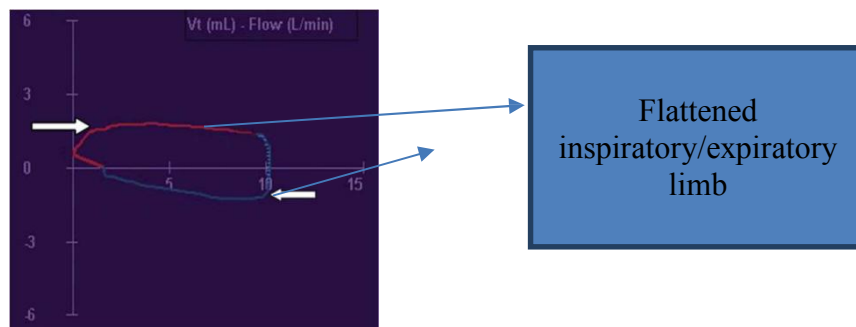
Flow–volume loop

The flow–volume loop displays the relationship between volume and flow. Volume is plotted on the horizontal axis and flow is plotted on the vertical axis. The breath starts at the zero axis and moves upward and to the right on inspiration, terminating at the delivered inspiratory volume and downward, to the left, back to zero on expiration.

A Normal flow volume loop
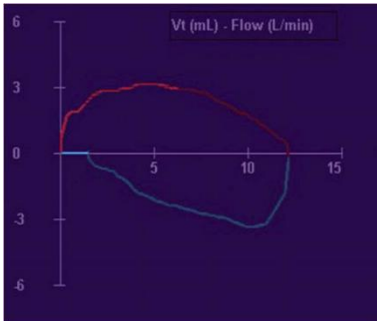
Flow volume loop with increased airway resistance

The flow volume loop changes shape when either inspiratory resistance (flattened inspiratory limb) or expiratory resistance (flattened expiratory limb) is increased. The response to bronchodilators can also be assessed from observing the improvement in peak inspiratory/expiratory flow in the flow volume loop.

Flow volume showing inspiratory and expiratory resistance
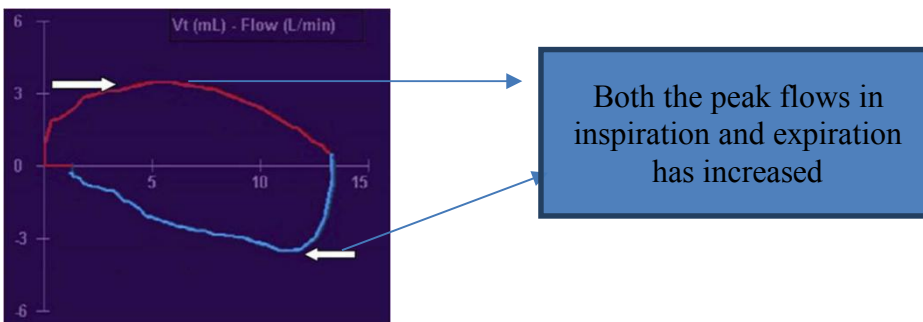
Flow volume showing improvement after a dose of bronchodilator

Loops can also help in diagnosing

1.   Endotracheal tube leak

Flow-volume loop showing large endotracheal tube leak where the expiratory portion fails to reach the origin

2. Optimum positive end expiratory pressure

Loops can aid in the determination of the best PEEP
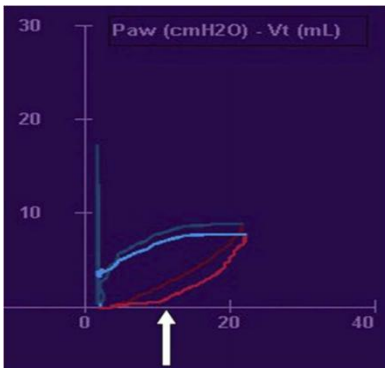
Abnormality in the pressure–volume loop, characterized by a need for a higher opening pressure. The loop looks ''box-like'' rather than elliptical. There is improvement when the PEEP (and concomitantly the PIP) is raised
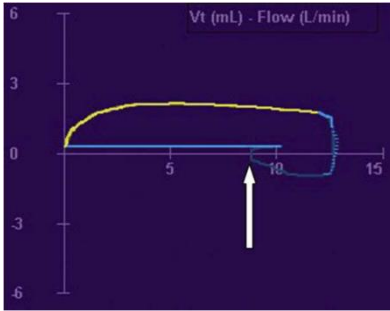
3. Turbulence

Can be noted in both flow volume or pressure volume loop
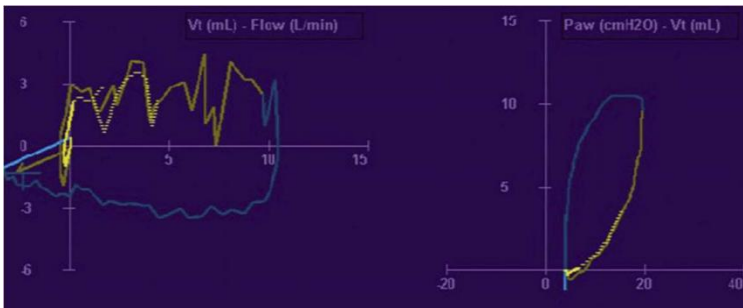
the ''noisy,'' irregular appearance to the loops in turbulence

CONCLUSION

- Real-time pulmonary graphics do provide useful information regarding the breath-to-breath performance of the ventilator and its interaction with the baby.
- Some complications of mechanical ventilation, such as gas trapping and hyperinflation, may be detected by graphics before they are clinically apparent.

- Fine-tuning of ventilator settings based on pathophysiology and patient response allows for customization of settings.
- It can also decrease the frequency of blood gas analysis and radiography, reducing the cost of care and increasing the comfort of the patient.

Suggested Readings

1. Real-Time Pulmonary Graphic Monitoring. Michael A. Becker, Steven M. Donn Clin Perinatol 34 (2007) 1–17
2. Real-time pulmonary graphics. Mark C. Mammel, Steven M. Donn. Seminars in Fetal & Neonatal Medicine Issue 3, July 2015.
3. Manual of Neonatal Respiratory care. 4th edn, Steven M Donn, Sunil K.Sinha. Springer publication

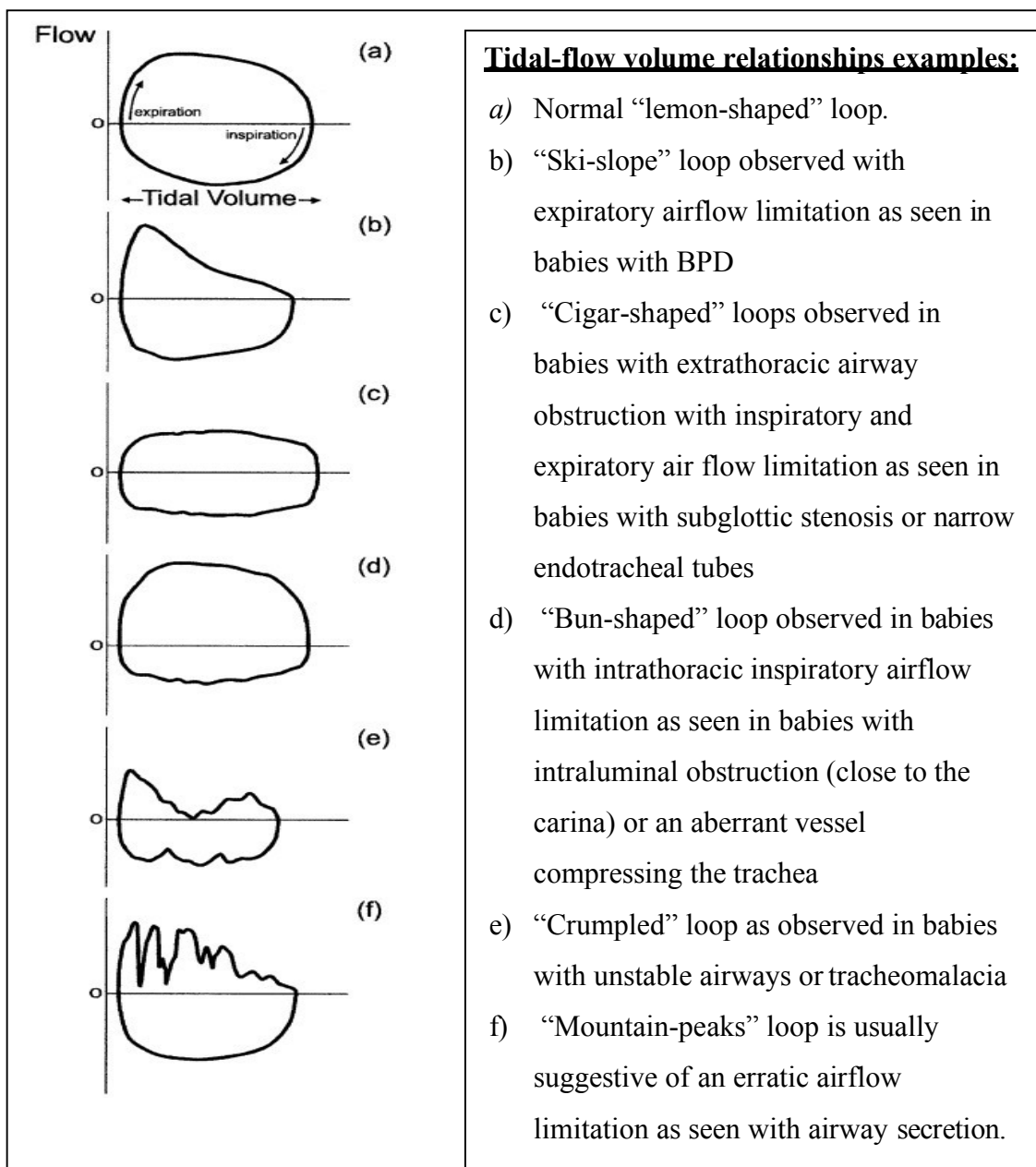**Tidal-flow volume relationships examples:**

a) Normal "lemon-shaped" loop.

b) "Ski-slope" loop observed with expiratory airflow limitation as seen in babies with BPD

c) "Cigar-shaped" loops observed in babies with extrathoracic airway obstruction with inspiratory and expiratory air flow limitation as seen in babies with subglottic stenosis or narrow endotracheal tubes

d) "Bun-shaped" loop observed in babies with intrathoracic inspiratory airflow limitation as seen in babies with intraluminal obstruction (close to the carina) or an aberrant vessel compressing the trachea

e) "Crumpled" loop as observed in babies with unstable airways or tracheomalacia

f) "Mountain-peaks" loop is usually suggestive of an erratic airflow limitation as seen with airway secretion.

Weaning a Neonate from ventilator
Sanjay Wazir

Despite the growing use of non invasive ventilation in NICUs across the globe, mechanical ventilation (MV) use is quite common with almost 75-83% of extremely low birth weight babies (ELBW) requiring invasive ventilation during the course of stay in NICU. MV although is life saving in such tiny babies is not without its attendenant risks including mortality and neurodevelopmental handicap.

Weaning is not equivalent to pulling out endotracheal tube but a process of slowly decreasing the amount of ventilatory support, with the patient gradually assuming a greater proportion of overall ventilation. While there is a relative consensus as to when MV mechanical ventilation should be initiated in a particular child, the management of babies during recovery from respiratory failure remains largely subjective and is predominantly determined by institutional or individual practices or preferences. This can lead to babies either being left on the ventilator too long, or extubated too hastily, thus requiring repeated re-intubation. Only two thirds of the ELBW babies can be successfully extubated exposing rest to periods of hypoxia, hypercapnia and trauma, infection and atlectasis during the reintubation attempts.

In physiologic terms, effective spontaneous breathing is dependent on a delicate balance between the loads imposed on the respiratory system and its capacity.The inability to tolerate extubation may be the result of poor effort, increased load on the respiratory muscles, and/or decreased inspiratory drive. Weaning attempts that are repeatedly unsuccessful usually indicate either incomplete resolution of the underlying illness or the development of new problems. Causes of extubation failure in neonates are presented in table

1. Factors increasing the infant's respiratory workload must be examined and optimized.

Table 1 Common causes of extubation failure in neonates

↑Respiratory load	↓Respiratory capacity
Increased elastic load	**Decreased respiratory drive**
● Unresolved lung disease	● Sedation
● Secondary pneumonia	● CNS infection
● Left to right shunt (PDA)	● PVH/PVL
● Abdominal distension	● Hypocapnia/alkalosis
● Hyperinflated lungs	
Increased resistive load	**Muscular dysfunction**
● Thick/copious airway secretions	● Muscular catabolism and weakness (malnutrition)
● Narrow/occluded endotracheal tube	● Severe electrolyte disturbances
● Upper airway obstruction	● Chronic pulmonary hyperinflation (BPD)
Increased minute ventilation	**Neuro-muscular disorders**
● Pain and irritability	● Diaphragmatic dysfunction
● Sepsis/hyperthermia	● Prolonged neuromuscular blockage (in renal failure, concomitant use of aminoglycoside and phenobarbitone)
● Metabolic acidosis	● Myotonic dystrophy
	● Cervical spinal injury

Weaning strategies
- Decrease the most potentially harmful parameter first -Once the infant has 'stabilized' clinically and blood gas values suggest that ventilatory needs are decreasing, the general principle should be to decrease the most potentially harmful parameter first e.g. in case of SIMV mode of ventilation, one should reduce the PIP as this is one responsible for the barotrauma more than other variables
- Limit changes to one parameter alone at one time so that if the process fails we know which parameter was responsible.
- Avoid changes of large magnitude - limit changes to 1-2 in PIP and rate changes to less than 10 at one time depending on the mode being used.
- Document response to all the changes

Mode of ventilation and weaning
- Assist-control
  - Decrease PIP but provide adequate V_T (4 ml/kg)
  - Decrease back up rate to 25–30
  - May increase trigger sensitivity to condition respiratory muscles
  - Extubate directly from assist-control or switch to SIMV
  - (BW) <1000 g: MAP < 7 cm H_2O and Fi O2 <0.30, BW >1000 g: MAP > 8 cm H_2O and Fi O2 <0.30
- SIMV
  - Decrease PIP but provide adequate V_T (4 ml/kg)
  - Decrease rate
  - Extubate when stable at low rate (i.e. 15 bpm) or combine with PSV
  - PIP < 14 cm H_2O, PEEP < 50 cm H_2O, rate <20, Fi O2 <0.30

- SIMV plus PSV –
  - Add PSV when SIMV rate below 30/min
  - Adjust level of PSV to give adequate V_T (4 ml/kg), reduce SIMV slowly
  - Extubate when stable at low SIMV rate (i.e. 15 bpm)
  - (BW) <1000 g: MAP < 7 cm H_2O and Fi O2 <0.30, BW >1000 g: MAP > 8 cm H_2O and Fi O2 <0.30
- HFOV –
  - Decrease both mean airway pressure and amplitude.
  - As the patient improves, and as amplitude decreases, the patient will do more spontaneous breathing.
  - When achieving most of the CO_2 elimination with spontaneous breathing, and the mean airway pressure has decreased sufficiently, patient can be extubated. General guidelines for extubation.
    - Patient <1000 g: mean airway pressure <8 cm of H_2O and FiO2<0.25

🖐 Patient >1000 g: mean airway pressure <9 cm of H_2O and FiO_2<0.3

Available data do not clearly document superiority of one mode over another in terms of their effect on lung injury, but there is good evidence, as well as a sound physiologic rationale, for using modes that support every breath of the patients. Important physiologic considerations why SIMV does not provide optimal support in very premature infants. For example, with SIMV, the spontaneous breaths in excess of the set IMV rate are not supported, resulting in uneven tidal volumes and potentially a high work of breathing, especially true during weaning, when the number of unsupported breaths increases, issue is most important in extremely small infants with correspondingly narrow ETT, because resistance to flow is inversely proportional to the fourth power of the radius. Consequently, in order to achieve adequate alveolar minute ventilation, a relatively large V_T is required for the limited number of mechanical inflations provided by the ventilator. Despite these considerations, many clinicians still prefer SIMV for weaning from MV, based largely on tradition and the belief that fewer mechanical breaths are inherently less damaging. Another misconception is that supporting every breath does not provide the infant with an opportunity for respiratory muscle training. with compliance of the respiratory system, determines the V_T. During weaning, as ventilator peak inflation pressure is decreased, the infant gradually assumes a greater proportion of the work of breathing and in the process achieves training of the respiratory muscles. The major disadvantage of SIMV can be mitigated by the use of pressure support (PS) for the spontaneous breaths.

Clinical data : comparing PTV versus SIMV, there are two small trials suggesting that weaning is faster in the PTV mode than SIMV. One trial comparing SIMV +PS vs SIMV alone also showed shorted duration of weaning in the SIMV + PS group. In pressure versus volume ventilation Cochrane review which included 6 studies on the secondary outcome of duration of IPPV, suggested that volume ventilation resulted in 1.5 days less on ventilator than the pressure limited group. Effect however was not significant for the less than 1000 grams babies. Twenty six center, randomized, noncrossover, controlled clinical trial comparing HFOV with SIMV showed age at successful extubation was significantly lower for infants assigned to high-frequency oscillatory ventilation. In conclusion, using other modes than the conventional SIMV mode may result in faster weaning but user friendliness to the new mode must be the prime reason for using a particular mode.

What should the newborn be extubated to

There are three options while removing from the ventilator

- Nasal Intermittent mandatory ventilation NIMV
- CPAP
- Head box oxygen

Cochrane review of extubation to CPAP vs Head box showed benefit of extubating to CPAP with NNT being 5. The appropriate duration of treatment with NCPAP however, remains uncertain, as does the method of its weaning. Cochrane review comparing extubation to NIMV versus CPAP has shown decreased reintubation rates with use of NIMV after extubation. NIPPV delivery was synchronised in all trials using the Infant Star ventilator with Star Synch abdominal capsule. Ventilator settings applied after extubation varied between studies. IMV rates varied between 10 and 25 per minute, and PIP from that used pre-

extubation to 2 to 4 cm water above that used pre-extubation. A recent abstract presented at PAS meeting 2012 PAS a multinational trial of non synchronized NIMV vs CPAP and showed no difference in the outcome. Hence, in our setting where we have non synchronized ventilation during nasal ventilation present data does not show superiority of one over the other and post extubation care would depend on the expertise of the care giver.

Other practices
Post natal steroids -Endotracheal intubation is used to provide intermittent positive pressure ventilation (IPPV) for a number of neonatal conditions. The presence of a foreign body in contact with delicate upper airway mucosa can lead to injury. This may take the form of laryngeal oedema, vocal cord injury or subglottic stenosis, all of which may present clinically as upper airway obstruction after extubation. This may in turn lead to increasing respiratory distress requiring reintubation of the trachea. Factors that may increase the likelihood of damage include repeated passage of an endotracheal tube, prolonged intubation and the presence of a large tube relative to the size of the glottis.

In a high risk population, the equation of treating six infants with dexamethasone in order to prevent one reintubation seems to favour treatment. The use of exogenous surfactant, increased use of antenatal steroids and the trend to extubate early to NCPAP have reduced the duration of endotracheal intubation. In addition, an increasing number of survivors at 22 to 24 weeks forms a new population in whom this treatment may have a different safety/efficacy profile. Dexamethasone reduces the need for endotracheal reintubation of neonates after a period of IPPV. In view of the lack of effect in low risk infants and the documented and potential side effects, it appears reasonable to restrict its use to infants at increased risk for airway edema and obstruction, such as those who have received repeated or prolonged intubations.
Caffeine - A systematic Cochrane reviewhas indicated a relative risk of failed extubation of 0.48 for infants exposed to methylxanthines before extubation, and on that basis, caffeine is virtually always administered before extubation in ELBW infants. A higher dose of 20mg/kg is more beneficial in extubation than a smaller dose.
Post extubation X ray – This idea was borne out of high incidence of PEA to the tune of 40% in 1970's which resulted in almost 1/3rd getting intubated again. But now the humidification and ventilation techniques have changed significantly in the last 3 decades and 1990's study have shown an incidence of 2.5% PEA. None of the babies with PEA required reintubation but some babies who required CPAP manifestated with increased Fio2 requirements and work of breathing. Hence, X ray is required only in patients with increasing Fio2 requirements.
Chest Physiotherapy-Cochrane review on this topic showed no clear benefit of peri- extubation active chest physiotherapy. Active chest physiotherapy did not significantly reduce the rate of postextubation lobar collapse. Applicability of the results of the review to current practice may be compromised due to advancements in neonatal care which have occurred over the interval since the earlier trials were performed
Nebulized racemic epinephrine - There is no evidence either supporting or refuting the use of inhaled nebulised racemic epinephrine in newborn infants. Similarly there is no evidence for the practice of saline nebulization after extubation.

Conclusion

Weaning and extubation from MV remain an inexact science. Current practice in most units is a trend towards non invasive ventilation and if that is not feasible then using MV for as short a time as possible. Alternate primary modes of ventilation apart from the conventional SIMV is likely to result in faster weaning. There is a strong evidence base for using caffeine and distending airway pressure after extubation.

Protocolised approach to Weaning a neonate from ventilator
Kumar Ankur

Definition: process by which ventilator-dependent patient is removed from ventilator. Weaning from MV is usually achieved by the gradual reduction of ventilatory support until the settings are judged to be low enough to remove support.

Prerequisite:
- No s/o encephalopathy – alert, active.
- Off sedation & good respiratory effort.
- Spo2- as per target with fio2 requirement ≤ 0.40.
- Maintaining tidal volume with minimal settings.
- Consider for caffeine before extubation if premature.
- After extubation electively consider for nasal ventilation (CPAP or NIMV).

Post-extubation monitoring

- Look for work of breathing: usually we tolerate some amount of tachypnea if CPAP requirement is not high (PEEP & fio2).
- ABG:Also tolerate PCO2 in range of 55 – 65 cm H2O as long as pH remains above 7.25 which is known as permissive hypercapnia.
- CXR: not always required but consider if WOB increases or fio2 requirement increases to rule out post extubation atelactasis, collapse or air leak.

Volume-targeted ventilation may accelerate weaning from MV. There is a strong evidence base for using caffeine and distending airway pressure after extubation.

Ventilatory settings to consider extubation readiness:
Conventional Ventilation (AC, SIMV/PSV)
- SIMV: PIP $\leq$ 16 cm H_2O, PEEP $\leq$ 6 cm H_2O, Rate $\leq$ 30, FiO_2<0.40
- AC/PSV, birth weight (BW) <1000 g: MAP $\leq$ 7 cm H_2O and FiO_2< 0.30
- AC/PSV, BW >1000 g: MAP $\leq$ 8 cm H_2O and FiO_2<0.30

Volume ventilation
- Tidal volume ≤ 4.0 mL/kg (5 mL/kg if <700 g or >2 weeks of age) and FiO_2<0.30

How to wean:

Gradually lower the ventilatory settings, as the lung pathology resolves. This will be appreciated by improvement in tidal volume and SPO_2 and decrease in FiO_2 and decrease in respiratory distress.

- Decrease PIP first. Decrease PIP by 1-2 cm at a time, till PIP is 14 to 16. Decrease FiO2 not more than 5% at a time, while keeping SPO2 in the range of 88-95%. Once baby comes to modest PIP (14-16) and low FiO2 (<30-40%, decrease the rate gradually to 30-40 per minute before extubating.
- After extubation, if infant is comfortable, he may be weaned directly to room air or minimal supplemental oxygen, if required (based on saturations: target 88-92%.
- If infant has respiratory distress after extubation, he may be put on nasal ventilation (CPAP or nasal IMV).
- In nasal IMV, ventilatory mode is switched to IMV but delivery method is either through nasal prongs same as for CPAP or through an ET tube cut to a length of 5-6 cm and inserted through one of nares to length of 3-4 cm inside. PIP is set to 14-16, PEEP 5-6, rate 30-40 and FiO2 as required. Required flow rate at times may be little higher to generate required pressures, if there is significant leak through nose or mouth. At times strapping the chin might help.
- Once infant remain stable on nasal IMV, settings are lowered further and then gradually switch to CPAP and subsequently wean to room air.

Failure of conventional ventilation:
- If an infant is unable to maintain oxygenation and other blood gas parameters on conventional ventilation despite high settings, it may be considered as failure of conventional ventilation and indication of switching to high frequency ventilation. Arbitrarily this can be considered at PIP level >25 in PT infants and >30-35 in term infant.

Causes of extubation failure:
- Severe or multiple episodes of apnea
- Hypoxemia
- Hypercapnia
- Upper airway obstruction
    - Edema of the epiglottic area
    - Subglottic edema/stenosis

Risk factors for extubation failure:

- Lower gestational age (<26 weeks)
- Prolonged ventilation (>10–14 days)
- History of previous extubation failure
- Use of sedatives (eg, morphine, fentanyl)

- Multiple reintubations: upper airway problems
- Evidence of residual lung injury: BPD, pulmonary interstitial emphysema
- Extubation from high ventilatory settings
- Extubation from high FiO2
- Hemodynamically significant PDA

Criteria for reintubation

- Severe apnea requiring positive pressure ventilation
- Multiple episodes of apnea: >6 within 6 hours
- Hypoxemia: FiO2 >50% to maintain SpO2 >88%
- Hypercapnia: PCO2 (partial pressure of carbon dioxide) >60 with pH <7.25
- Excessive work of breathing with severe retractions

Ventilator Associated Pneumonia(VAP)

Ravi Sachan, Kumar Ankur

Ventilator-associated pneumonia (VAP) is a serious complication related to mechanical ventilation in the neonatal period. However, lack of a specific definition and difficulties obtaining noncontaminated samples of the lower respiratory airway render microbiological diagnosis and etiological treatment extremely difficult.

Definition

The Centers for Disease Control and Prevention (CDC) defines VAP as a nosocomial infection diagnosed in patients undergoing MV for at least 48 h. Diagnosis of a VAP episode requires a combination of radiological, clinical, and laboratory criteria (table 1) .However, CDC/ NNIS criteria refer to infants younger than 1 year and do not define specific criteria for the newborn period in term or preterm infants.

Table 1. Diagnostic criteria for VAP in infants younger than 1 year

Radiological signs	Patient with one or more (in patients with underlying diseases two or more) chest X-rays with one of the following findings: – new or progressive and persistent infiltrate – consolidation – cavitation – pneumatoceles
Clinical signs and symptoms	Worsening of gas exchange [e.g. oxygen desaturations (e.g. pulse oximetry <94%), increased oxygen requirements, or increased ventilation demand] and three of the following: – temperature instability with no other recognized cause – leukopenia (<4,000 WBC/mm³) or leukocytosis (>15,000 WBC/mm³) and left shift (>10% band forms) – new onset of purulent sputum, or change in the character of sputum, or increase in respiratory secretions, or increased suctioning requirements – apnea, tachypnea, nasal flaring with retraction of chest wall or grunting – wheezing, rales, or rhonchi – cough – bradycardia (<100 beats/min) or tachycardia (>170 beats/min)
Microbiolocical findings	At least one of the following: – positive growth in blood culture not related to another source of infection – positive growth pleural fluid culture – positive quantitative culture from a minimally contaminated LRT specimen [e.g. BAL (≥10⁴ CFU/ml) or protected specimen brushing (≥10³ CFU/ml)] – ≥5% BAL-obtained cells contain intracellular bacteria on direct microscopic examination (e.g. Gram stain) – histopathological exam shows at least one of the following criteria for pneumonia: abscess formation or foci of consolidation with intense PMN accumulation in bronchioles and alveoli, positive quantitative culture of lung parenchyma (≥10⁴ CFU/g tissue), or evidence of lung parenchyma invasion by fungal hyphae or pseudohyphae

WBC = White blood cells; LRT = lower respiratory tract; CFU = colony-forming units.

Pathogenesis and Pathogen

Pathogenesis of Ventilator Associated Pneumonia

Endogenous Sources of Micro-organism

1) Impaired natural protection/clearance system allow increase colonization of nasopharynx

2) Colonized oropharynx & gastric fluid pool along tube in neonates

3) Colonized tracheal secretions

Mechanism for pneumonia

1) Aspiration of colonized fluids from any of the above sources into lungs can result in pneumonia

2) A hematogenous source seeding the lungs may rarely cause pneumonia

Blood ◄► **_Pneumonia_**

Fig. 1. Endogenous sources of organisms responsible for VAP. (*Courtesy of* Walt Earhart, Wheaton Franciscan Healthcare; with permission. *Reproduced from* NeoreviewsPlus, copyright August 2010, Question 8, AAP; with permission.)

Pathogenesis of Ventilator Associated Pneumonia

Exogenous Sources of Micro-organism

1) Hands of healthcare worker

2) Ventilator circuit

3) Biofilm of endotracheal tube

Mechanism for pneumonia

Pneumonia occurs when colonized secretions are inhaled into lungs through the endotracheal tube
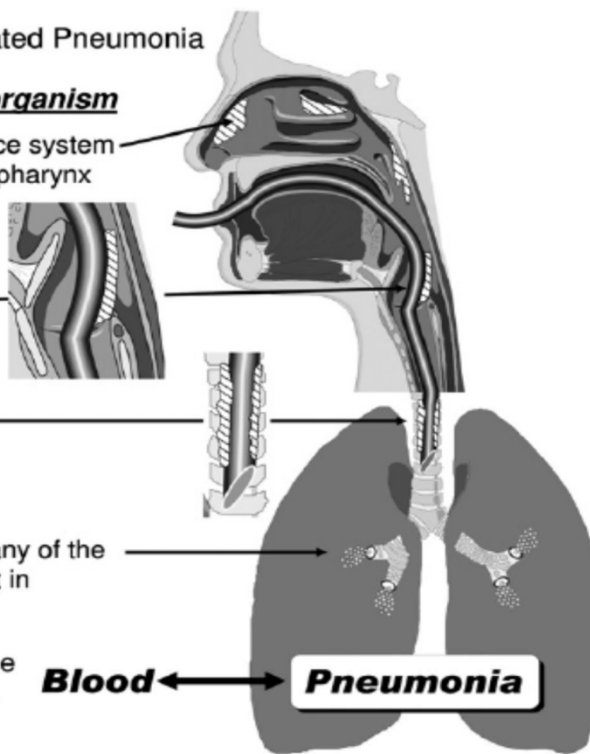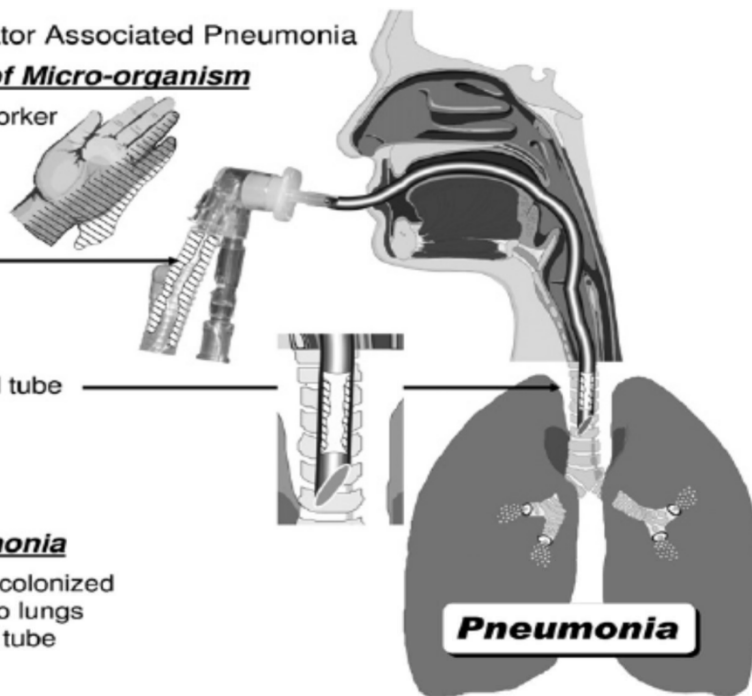
Pneumonia

Fig. 2. Exogenous sources of organisms responsible for VAP. (*Courtesy of* Walt Earhart, Wheaton Franciscan Healthcare; with permission. *Reproduced from* NeoreviewsPlus, copyright August 2010, Question 8, AAP; with permission.)

Etiological diagnosis is hindered by the difficulty in obtaining noncontaminated samples from the
infants' airways. The CDC permits the diagnosis of 'clinically defined pneumonia', based only on clinical and radiological findings, without any isolated pathogen.

Isolation of pathogen without clinical and radiological signs is not diagnostic of VAP and could just represent colonization of the airways.

The most common pathogens isolated in the neonatal population are Pseudomonas aeruginosa,Enterobacter and Staphylococcus aureus . However, isolation of other microorganisms such as Klebsiella pneumoniae and Escherichia coli and Acenatobacter spp has also been reported.

Sample Collection method

Both noninvasive and invasive (bronchoscopic) techniques are equally employed for sample collection. Bronchoalveolar lavage (BAL)/or those taken from protracted specimen brush (PBS) is highly specific and at present the standard for microbiological sampling. Contrarily, noninvasive techniques such as tracheal aspirates are more accessible and easy to use, but they tend to overdiagnose VAP and, as a result, increase the use of antibiotics.

Risk factors

Prematurity and/or low birth weight, reintubation,primary blood stream infection,prior antibiotic use, sedation non initiation of enteral feeds,parenteral nutrition,endotracheal suctioning,days on MV,transfusion of any blood product,genetic syndrome, steroids,histamine type 2 receptor blockers are the risk or predisposing factors for developing VAP out of which prematurity and days on MV are the most relevant ones

Treatment

Understanding the microbiology of VAP is critical for choosing empirical broad-spectrum antibiotic . However, there are no consensus guidelines for antibiotic treatment either in neonates or in children, and empirical treatment should be selected according to the nosocomial flora and resistance patterns of each individual unit. The duration of antibiotic administration for VAP in the newborn period is still unknown. No published data in this regard are available in the literature.

Prevention

The CDC and American Thoracic Society have published guidelines for the prevention of health care-associated pneumonia.

1. Bundle Care Practice.

- Elevating the head of a ventilated patient's bed to between 30 and 45 degrees to reduce the risk of aspiration of contaminated oropharyngeal and gastrointestinal contents.
- Clearing secretions from above the cuff of the endotracheal tube whenever the tube is repositioned.
- Use of closed multiuse suction catheters allowed endotracheal suctioning without

disconnecting patients from the ventilator. It reduces environmental contamination of the endotracheal tube but potentiate for bacterial contamination when pooled secretions in the lumen are reintroduced into the lower respiratory tract with repeated suctioning.

- Breathing circuit condensate collected in the tubing should be drained away to prevent aspiration.

2. Committee suggest the use of ETT with dorsal lumens to allow drainage of respiratory secretions, orotracheal instead of nasotracheal intubation, and a change of ventilators' respiratory circuits only if they are visibly contaminated. Some studies have shown that ETT with nano-modified coatings reduced the incidence of VAP by preventing biofilm formation and ETT colonization and providing free radical destruction of pathogens but of note published experiences in the neonatal period are lacking.

3. Hand Washing : Routine hand washing is one of the most important strategies to reduce nosocomial infections . In a 2-year-long surveillance intervention with NICU patients, increased hand hygiene compliance (from 43 to 80%) significantly reduced the incidence of respiratory infections from 3.35 to 1.06 infections per 1,000 patient days.

4. Rapid Extubation : Since the duration of MV appears to be a major risk factor for the development of VAP in neonates, promptly weaning patients off the ventilator appears to be a desirable strategy to prevent VAP.

5. Use of Histamine : 2 Receptor Antagonists or Antacids is believed to increase the risk of VAP as acid gastric content may make colonization with pathogenic organisms difficult.However there is no published experience in the neonatal period.

6. Selective Decontamination : Selective decontamination consists of the establishment of a regimen of topical or intravenous antimicrobials in an attempt to reduce the burden of pathogenic bacteria in aspirated secretions. Randomized studies in pediatric patients have shown conflicting results . In a prospective cohort nonrandomized study, NICU patients received oral polymixin E, tobramycin, and nystatin correctly (during the first 5 days) or incorrectly (after 5 days) or they did not receive any decolonization . Results revealed that correct selective decolonization had a protective effect toward nosocomial infections of an intestinal origin. However, a separate analysis of the impact on respiratory infections alone was not performed . Accordingly, no recommendation regarding selective decontamination in neonates is warranted.

7. Probiotics : Recent meta-analysisof 7 randomized controlled trials conducted in adult populations concluded that probiotics showed no beneficial effect in patients who are mechanically ventilated, did not significantly decrease the incidence of VAP, and should not be recommended for routine clinical application.

Conclusion

It is important that caregivers treating ventilated preterm infants keep diagnosis of VAP in mind when a sudden worsening of a patient with risk factors occurs and clinical signs suggest this entity.

As a general rule, patients should be extubated as soon as possible as a main strategy to prevent VAP. This update aims to draw attention to this subtle but severe complication affecting preterm infants in the NICU.

Suggested Readings

1. Centers for Disease Control and Prevention. Criteria for defining nosocomial pneumonia. Available at:http:// www.cdc.gov/ ncidod/ hip/ NNIS/ members / pneumonia / final / PneuCriteriaFinal.pdf.
2. Strategies to Prevent Ventilator-Associated Pneumonia in Neonates. Jeffery S. Garland.
3. Ventilator-Associated Pneumonia in Neonatal Patients: An Update
    María Cernada, María Brugada, Sergio Golombek, Máximo Vento

How to choose a neonatal ventilator

Kumar Ankur, Anil Batra

In the last two decades the role of mechanical ventilation in the neonatal intensive care unit (NICU) has been rapidly evolving. Prior to the early 1970s, neonates either died without access to appropriate ventilators, or they were supported in the first days of life with pediatric volume ventilators and a large pre-set tidal volume (VT) (approximately 18 mL/kg). The ongoing clinical management was based principally on subjective assessment of optimal chest rise, breath sounds, color, and maintaining normal blood gas values. Nearly 3 decades following the advent of the microprocessor, several advances in neonatal ventilator technology have ushered in a number of proposed improvements to the neonatal ventilator.

Published information on the value and effectiveness of individual devices and features is limited. Improved patient outcomes due to technology innovation are difficult to demonstrate. Broadly, there are two types of ventilators available in market:

- Cradle to grave ventilators
- Dedicated neonatal ventilators

Cradle to grave ventilators (newborn to adult ventilators) are promoted by manufacturers as ventilator suitable for patients with all age groups. Though many clinicians are fancied with this notion, these ventilators are not at par with dedicated neonatal ventilator in mechanics. Dedicated neonatal ventilators such as Drager Babylog 8000 plus has lower ventilator- imposed expiratory resistance, better trigger response time and more accurate tidal volume measurements than ventilators common for all ages. However, these are reasonable option in less busy centres, as these ventilators will be utilized more often across different age groups than dedicated neonatal ventilators and hence will be more cost effective.

Selection of a neonatal ventilator for your unit should be based upon:

- Technical features and skill and ability of clinician to utilize technology to its best
- Level of expertise of nursing and resident staff to understand mechanics and troubleshooting
- Type and age group of patients being treated
- Budget of purchase and maintenance cost
- After sale service

Technical specifications of a ventilator:

Conventionally ventilators are categorized in pressure control ventilators and volume control ventilation. In contrast to adult ventilation, pressure control ventilation has been more popular in neonates. In following paragraphs is a brief discussion of various modes of ventilation, one might be looking for in a ventilator (Details of individual modality is covered elsewhere).

Early neonatal pressure ventilators were relatively inexpensive and simple to operate. Mechanical breaths were machine-triggered, intermittent flow, time-cycled, and pressure controlled and is known as continuous mandatory ventilation (CMV). There was no flow of gases for spontaneous breaths in between the mechanical breaths. Subsequently, intermittent mandatory ventilation (IMV) provided continuous flow, intermittent pressure-controlled breaths. Continuous flow of gases in circuit allowed spontaneous breaths in between mechanical breaths. However, there is no synchrony between ventilator driven breath and

spontaneous breaths. Hence there is potential for mechanical breaths to be delivered out of phase with the neonate's spontaneous respiratory effort, resulting in the patient fighting the ventilator. To improve patient-ventilator synchrony and patient comfort, neonates require sedation and or neuromuscularly paralysis.

Patient triggered ventilation is further sophistication, where machine senses patient's efforts and delivers mechanical breath in synchrony with patient's breath. However, technical limitations of detecting and responding rapidly to small patient efforts in neonates challenged scientists till 90's. Respiratory monitoring during ventilation has grown more comprehensive and sophisticated. Today most neonatal ventilators incorporate small, lightweight, hot-wire or variable orifice flow sensors that can accurately and precisely measure flow and pressure changes at the proximal airway and provide patient-triggered ventilation in even the smallest of patients. Flow-triggering with a sensor placed at the proximal airway is currently preferred for neonatal ventilation over pressure-triggering or flow sensor placed more distally (close to machine). A proximal flow sensor is necessary not only for triggering, but for accurate VT measurement and airway graphic display.

The most widely used forms of patient-triggered ventilation in the NICU are what have been referred to as "assist/control" and "synchronized intermittent mandatory ventilation" (SIMV). These forms of patient-triggered ventilation are preferred because premature neonates often have unpredictable breathing patterns.

In PSV the patient controls the start of inspiration, the start of expiration, the inspiratory time, the breathing frequency, and the minute volume, so the patient has complete control of the breath, which enhances patient comfort and patient-ventilator synchrony. It is important to note that to use PSV the neonate must have sufficient respiratory drive, though some of the newer PSV modes have an apnea backup mode.

Recently with improved volume monitoring capabilities and lung mechanics measurements has generated interest in volume targeted ventilation. Most of the currently available neonatal-capable ventilators allow setting the VT as low as 2–3 mL and, remarkably, with great precision and accuracy.PSV has evolved to employ adaptive targeting: for example, "volume support" (on the Servo-i ventilator, Maquet, Solna, Sweden) or "pressure support volume guarantee" (on the VN500, Drager, Lubeck, Germany), which is a mode that automatically adjusts the inspiratory pressure to maintain a minimum pre-set VT target. Adaptive pressure control involves volume-targeted breaths that automatically adjust inspiratory pressure based on VT measurements to target a minimum inspiratory or expiratory VT.

Nasal neurally adjusted ventilatory assist (NAVA) is a novel form of ventilation, which uses the electrical activity of the diaphragm (EAdi) to determine the timing and magnitude of inspiratory pressure delivery during spontaneous breathing. The EAdi signal is obtained with a 5.5 French esophageal catheter placed at the level of the diaphragm. When positioned properly, the EAdi signal can accurately and reliably trigger and cycle a positive-pressure breath, independent of air leak. Additionally, the magnitude of the inspiratory pressure assist is a product of the EAdi signal and the pre-set NAVA level. However, NAVA requires frequent bedside attendance and requires intact respiratory drive of infant. Although many of the difficulties associated with flow sensors at the airway might be bypassed with this new

EAdi method, new issues such as proper sensor placement and higher cost might develop instead.

High frequency ventilation is a mode of ventilation where mechanical breaths are given at very high rates (300-900 per minutes) with a very small tidal volume (1-2 ml/kg). This mode of ventilation has been shown to be superior to conventional ventilation and lung protective. However this mode requires more careful monitoring and frequent adjustments in ventilator parameters.

While a tremendous amount of resources have gone into the design and testing of these devices and modalities, the question remaining is, have these advances actually improved outcomes for neonates? Current-generation ventilators have added a new level of complexity and expense to neonatal care. We are an equipment-centered profession. The more complex the device, the more we dive into its intricacies and master its details. There is a great desire to constantly modernize our technology and stay current. It is not uncommon for clinicians to be seduced by new advanced features found on neonatal ventilators. However, increased complexity of technology may increase risk to patients. These risks include misunderstanding and misapplication by clinicians. But in reality many of these features are never used in clinical practice. Further, improvements to neonatal ventilators have not come without a cost. Thus, the seasoned clinician is left wondering if the costs and risks of novel ventilators and ventilation modes outweigh the clinical benefit to patients.

In my opinion, SIMV, if used appropriately with judicious selection and frequent adjustment of ventilator settings may clinically be as effective as newer modes and may be less complicated and is easy to understand and practice by resident and nursing staff. Newer ventilators with complex modes of ventilation should be resorted to only if required expertise can be assured round the clock.

One should choose a ventilator based on what is his requirement and not by the fact that which machine has maximum number and/or most recent of modes. One should list his specification needs and then pick the machine which fulfils all the requirements within the stipulated budget rather than throwing unnecessary money on an unnecessary functions. Critical points to be looked into while purchasing a neonatal ventilator are:

- There should be patient trigger mode/modes
- Sensing mechanism should be close to ET tube; not at machine end
- Heated wire anemometer is preferred
- Modes of ventilation depend upon unit's expertise and budget. Minimum requirement is CPAP and SIMV.
- Spares and after sale service (try to have AMC)

Prototype specification for a basic neonatal ventilator

Modes of ventilations
    Pressure control modes
CPAP mode
IMV
SIMV
Pressure support ventilation (can be optional)
       Volume guarantee (can be optional)
       High frequency ventilation (optional)
       provision of nebulization during uninterrupted mechanical ventilation

Flow sensor:
-heated wire anemometer (preferred)
-Should be distal (close to endotrachela tube end)

Circuits & humidifiers:
--should provide a humidifier with temperature display of distal end at circuit
--humidifier should have both nasal and ET modes options
--should be compatible with most commercially available ventilator circuits and humidifier

Ventilatory Controls (should have following controls with described ranges)
Inspiratory time : 0.1 sec to 1 sec
Respiratory freq: 1-150breaths/min
Inspiratory flow: auto adjusting
Tidal volume: lower limit 2 ml; upper limit 150ml or more
Peak pressure limit upto 60-70 mbar
CPAP/PEEP limit 0-20 mbar
FiO2: 21-100%
Triggering: preferably flow triggering

Measure parameters (should display following parameters):
PIP
PEEP
MAP
Respiratory rates
Inspiratory time
Expiratory time
I/E ratio
Tidal volume
Minute ventilation
FiO2
Compliance
Resistance
Leak

Spontaneous breaths (%)
Alarms: Low gas supply/pressure (for o2 as well as air)   Low battery   Low FiO2   FiO2 sensor inoperable   High / low PIP   High /low PEEP   High / low tidal volume   High / low minute ventilation   Apnea   Leak in circuit   Hose/tubings kinking /obstruction   Inverse ratio ventilation
Waveforms & loops Display: -pressure, flow and volume waveform   -pressure-volume and flow-volume loops
BATTERY OPERATIONs: Operating time at least 1 hr   Alarm for power failure
Power requirements: 100-240 volt
Other specifications (optional):   -Ability to print data in tabulated format as well as ability to print waveforms   -Ability to export data to external device (USB device, hard disc, computer)   -Ability to retrieve and save breath to breath data of all measured parameters in excel format   -Connectivity to other devices (with Ethernet, dicom etc)
Spares and warranty

Abbreviations

A/C: Assist Control

ARDS: Acute, respiratory distress syndrome

CPAP: Continuous Postive Airway Pressure

CMV: Conventional Mechanical Ventilation

ET: Endotracheal Tube

IMV: Intermittent Mandatory Ventilation

INSURE: INtubation-SUrfactant administration-Rapid Extubation

MAS: Meconium aspiration syndrome

MAP: Mean airway pressure

MV: Minute Ventilation

NIV: NonInvasive Ventilation

NIPPV: Nasal intermittent positive pressure ventilation

NICU: Neonatal Intensive Care Unit

PIP: Peak Inspiratory Pressure

PAP or δp: Pressure above peep

PEEP: Positive end expiratory pressure

PTV: Patient Triggered Ventilation

PSV: Pressure Support Ventilation

PPHN: Persistent pulmonary hypertension of newborn

PRVC: Pressure Regulated Volume Control

RDS: Respiratory Distress Syndrome

SIMV: Synchronised Intermittent Mandatory Ventilation

SBT: Spontaneous breathing trials

Ti: Inspiratory Time

Te: Expiratory Time

VT: Tidal Volume

VG: Volume Guarantee

VAPS: Volume Assured Pressure Support

VAP: Ventilator acquired pneumonia

WOB: Work of breathing