

The Basics of Non-Invasive Mechanical Ventilation

VENTILATOR SETTINGS

TRIGGER

- **How sensitive the machine is to the patients' attempts to breathe.** Obviously, should be fairly sensitive. About -1 mmHg is a good setting. This way if the patient sucks a tiny amount of air in, the ventilator will deliver a full breath.

TIDAL VOLUME

- **How much air you push into your patient.** This is usually 6 to 8 ml of air per kg of patient. Any more than 10 ml/kg will probably cause barotrauma.

A typical 70kg patient will require 420- 560 ml tidal volume.

A SAFE NUMBER TO REMEMBER IS 500 ml. Start there and adjust according to response.

RESPIRATORY RATE

- **Self-explanatory. Not applicable to ventilation of a patient-initiated type (like BiPAP)**
- **12 to 16 is a good rate. Increase this to increase the amount of CO₂ being exhaled and the amount O₂ being inhaled.**

PEEP

- **Positive End-Expiratory Pressure**
 - **Typically, starts at 5mmHg.**
 - **Has circulatory repercussions: reduces preload (reduced venous return to the heart), which may be useful or harmful, depending on the situation (for example, in fluid overload and CCF, you may actually want to reduce the preload; reduced stretch may paradoxically improve the activity of a sick heart)**
- If venous return is decreased, its decreased from everywhere, INCLUDING THE BRAIN.** If you are being careful with intracranial pressure, you may want to keep the PEEP low.

Rule of thumb: PEEP reduced cardiac function when its normal, and improves it if increased preload is the problem.

FLOW RATE

- **60 L/min is usually enough**
- **You need a higher flow rate if you're trying to huff off CO₂.** The point is that if the flow rate is high, the breath takes less time; and therefore more time can be devoted to expiration (which is what you want to be doing if you're trying to *get rid of* a gas.)
- **Flow rate determines inspiration to expiration ratio (I:E); the typical I:E is between 1:2 and 1:3**
- The downside of short inspiratory time is decreased mean airway pressure and thus decreased oxygenation
- The downside of a high flow rate is a high peak airway pressure, and therefore more chance of barotrauma.

FLOW PATTERN

- **i.e a constant steady pressure, or a "ramp wave" where pressure increases gradually**
- **The ramp wave is better for almost every situation.**

INSPIRED OXYGEN FRACTION

- **Depends on the situation; generally, the aim is to set this as low as possible while still supplying enough oxygen to meet requirements**
- **The people with an acute MI will need more oxygen than those with chronic COPD**
- **Generally, aim for sats over 90 and a PaO₂ over 60.**

EFFECTS OF MECHANICAL VENTILATION

BROAD INDICATIONS

- insufficient oxygenation
- And / Or
- insufficient ventilation

Basically, anything you can describe as "respiratory failure", which includes failure to fill the lungs with enough air, or failure to get enough gas exchange surface recruited.

The following sorts of people will be seen on NIV:

- Severe COPD exacerbations with high CO₂
- Acute pulmonary oedema
- Acute severe asthma
- Guillain-Barre or myasthenia gravis
- Chest trauma

They will have these signs: respiratory rate over 30; low PaO₂ (say, under 55) despite high FiO₂; high A-a gradient; etc etc

IMPROVED GAS EXCHANGE

as a consequence of improved ventilation - perfusion matching.

This is because of decreased physiologic shunting. I.e. positive inspiratory pressure increases the number of alveoli being used to exchange gases.

Physiologic shunting:

Normally 2 to 5% of the blood going through the lungs is not oxygenated.

This is usually because some of the lung is not ventilated, eg. when there is atelectasis. It stands to reason if you ventilate the useless lung, the total amount of oxygenated blood will increase.

SHUNT EQUATION:

$$\frac{\text{Volume of blood not oxygenated}}{\text{Total cardiac output}}$$

$$= \frac{\text{Alveolar oxygen} - \text{Arterial oxygen}}{\text{Alveolar oxygen} - \text{venous oxygen}}$$

REST TIRED RESPIRATORY MUSCLES

Breathing is hard work for people with severe asthma or COPD. Apart from improving their comfort, NIV

- Improves the quality of each breath taken because they were taking such poor-quality shallow breaths
- Reduces metabolic demand by taking over some of the muscles' work
- Reduces acidosis – in this case, lactic acidosis from tired respiratory muscles- an effect separate to the reduction of a respiratory CO₂ related acidosis

IMPROVE CARDIAC OUTPUT IN CCF

Normally, PEEP will reduce cardiac output by reducing venous return. However- if the ventricle is diseased, it will decompensate.

The more you stretch the left ventricle, the harder it contracts. Up to a point. Too much preload- and the diseased ventricle will decompensate beyond this point, and so cardiac output will drop.

Frank-Starling curve:

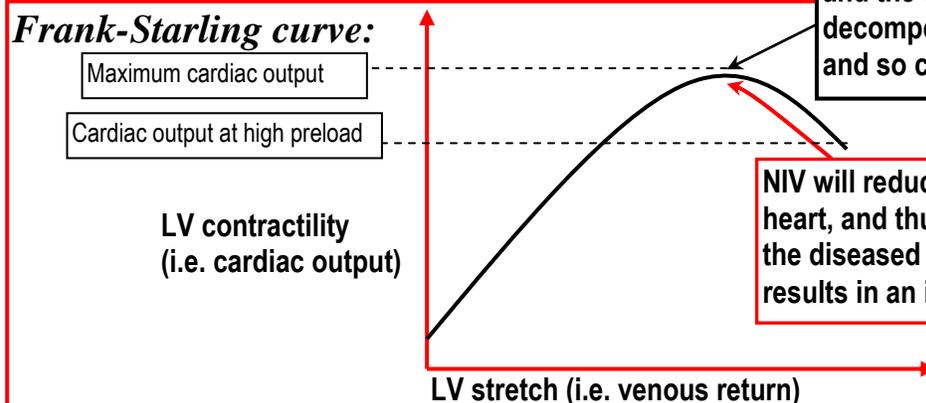
Maximum cardiac output

Cardiac output at high preload

LV contractility
(i.e. cardiac output)

LV stretch (i.e. venous return)

NIV will reduce the venous return to the heart, and thus decrease the load on the diseased left ventricle. This in turn results in an improved cardiac output.



EFFECTS ON THE LUNG:

Barotrauma:

Ruptured alveoli as a consequence of pressures being set too high!

If an alveolus is ruptured, air ends up in the interstitial space of the lungs.

This leads to pneumothorax, pneumomediastinum, pneumoperitoneum, and subcutaneous emphysema.

Needless to say, this is bad.

Most common in Asthma and interstitial lung disease eg. asbestosis.

Solution: turn down the tidal volume and/or flow rate

Ventilator-associated Acute Lung Injury:

The consequence of long-term barotrauma, i.e. alveoli constantly being overdistended. The result is injury to lots of alveoli, with acute pulmonary oedema.

Most often seen in restrictive lung disease, or when there already is some sort of acute lung injury

Auto-PEEP:

Positive airway pressure at the end of expiration; i.e. incomplete expiration.

Is the respiratory rate set too high? Is the inspiratory time too slow (flow rate too low)?

Auto-PEEP exacerbates the hemodynamic effects of PEEP.

Solution: increase the duration of expiration; either increase the flow rate or decrease the resp rate.

Physiologic dead space:

PEEP tends to increase physiologic dead space by shoving lots of air into a portion of the lung which is not receiving a corresponding increase in blood flow. So its wasted unexchanged air, i.e. dead space.

Physiologic shunt:

If there is increased shunt because somewhere the alveoli are not getting any air, PEEP will open them up

And increase their perfusion (thus, a greater proportion of the blood exiting the lung will be oxygenated, and therefore the shunt will decrease)

Diaphragm atrophy:

Use it or lose it. The diaphragm atrophies remarkably quickly; as few as 18 hours on a ventilator may result in a significant decrease in diaphragmatic exercise tolerance. The diaphragm atrophies remarkably quickly; as few as 18 hours on a ventilator may result in a significant decrease in diaphragmatic exercise tolerance.

RESPIRATORY MUSCLES IN GENERAL WILL ATROPHY.

EFFECTS ON THE CIRCULATION:

Decreased Venous Return:

If you increase pressure inside the thorax, less venous blood will flow there. That makes sense.

If the patient is volume-depleted, you will find that this makes venous return even worse.

Increased Pulmonary Vascular Resistance:

You are pushing air into the lungs under pressure. It makes sense that trying to push anything else (like blood) into that lung will now be harder. You will have to fight the pressure from PEEP. Therefore, right ventricular output will decrease in proportion to increasing PEEP.

Decreased cardiac output:

The venous return is impaired, so not much blood is entering the heart;

Pulmonary resistance is increased, so you aren't pumping much blood to the left ventricle anyway.

The right ventricle is struggling, and is distended, pushing the septum over into the left side – which means there is not much space to fill. And if the left ventricle doesn't fill much, it doesn't pump as hard.

However: if the left ventricle is diseased, it will have trouble moving blood when the preload is normal or increased. So by reducing preload, you can improve LV function. This is useful in BiPAP for pulmonary oedema, because those patients usually have poor LV function.

OTHER EFFECTS:

Stress Ulceration:

PEEP for longer than 48 hours will cause gastric ulceration. This is mainly due to decreased splanchnic perfusion- for some reason; and we don't exactly know why. Aminotransferase and LDH will be raised in these patients.

Renal-mediated Fluid Retention:

Because of decreased cardiac output, PEEPed kidneys will become confused, and act in a stereotypically "shocked" manner; e.e. they will mistake falling output for a drop in volume, and kick off the fluid-preserving rennin-angiotensin cascade. The result is fluid retention and oedema.

Increased Intracranial Pressure:

The decrease of venous return to the heart causes venous blood to pool in the brain; therefore ICP rises in proportion to decreased venous return.

The uses of non-invasive ventilation

- hypercapnic acidosis of COPD
- cardiogenic pulmonary oedema
- hypoxic respiratory failure
- post-extubation patients with weak respiratory muscles
- patients whose respiratory muscles are for whatever reason ineffective (Eg. Guillain-Barre)

BiPAP huffs the CO₂ .
CPAP helps the left ventricle.
Both improve oxygenation.

There should be an improvement in pH and PaCO₂ within the first 30 minutes to 2 hours.

If the patient is fighting the ventilator, or if the ABGs still look like crap, you may want to intubate them.

BiPAP in COPD:

- reduces mortality in severe hypercapnic acidotic COPD (11% vs 21 %)
- reduces intubation rates (16% vs 33%)
- the COPD has to be quite severe before the benefits become apparent.
- Acts by reducing the physiological shunt and therefore increasing oxygenation and the rate of CO₂ removal.

BiPAP and CPAP in CARDIOGENIC PULMONARY OEDEMA:

- reduces mortality (11% vs 20 %)
- seems to work best for pulmonary oedema which presents with high CO₂
- improves cardiac output by reducing left ventricular strain

IF the oedema is not causing hypercapnea, use CPAP.

If the patient has high CO₂, use BiPAP.

Generally, one should try to reduce preload with diuretics first; but if the blood pressure is too low (owing to a decreased, decompensated, feeble cardiac output) the NIV solution is better.

CPAP in PNEUMONIA:

- reduces mortality (18% vs 39 %) – but only among the ICU population.
- Seems to work best for people who are hypoxic secondary to their pneumonia
- The patients need to be able to manage their secretions

PRACTICAL NIV: “what does that number mean?”

CPAP: not considered “true” ventilatory support.

- start at **1cm per 10 kg** bodyweight. 7cm for a normal person.
- Starting pressure of **NO MORE THAN 10cm!**
- **Watch resp rate and ABGs. If rate fails to decrease and O2 sats fail to improve, titrate CPAP up by 1 or 2 cm increments.**

When to use CPAP and not BiPAP:

- **cardiogenic pulmonary oedema which remains hypoxic despite lasix, but which is NOT hypercapnic**
 - **severe pneumonia**
- for everything else, BiPAP is your choice.

BiPAP:

Two main numbers to remember: IPAP and EPAP

IPAP: inspiratory positive airway pressure

EPAP: expiratory positive airway pressure

- Little skinny people (< 60kg) start at **10 and 4cm (IPAP and EPAP)**
- Large fat people (> 60kg) start at **12 and 6cm**
- **Dial up the oxygen to keep sats above 88%**
- Repeat the ABG in 60 minutes
- If the CO2 is still over 50, increase IPAP by 2cm.
- If they also have pulmonary oedema, obesity, or Auto-PEEP, increase EPAP by 1cm increments. (Auto-PEEP can be identified by a failure of the patients’ breaths to initiate a machine inspiration, i.e. asynchrony)
- **IPAP can go up to 20.**
- **EPAP can go up to 10.**