"Αναζωογόνηση σε μη τραυματία"

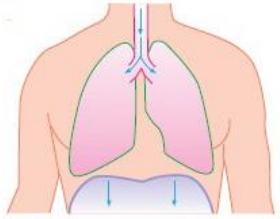
Βασικές αρχές αερισμού στην αναζωογόνηση

Ευφροσύνη Δήμα

Επ.Α/ Α ΚΕΘ

ΓΝΑ Ευαγγελισμός

Εισαγωγικά



Oxygenation

- to maximize O2 delivery to blood (PaO2)
- Alveolar-arterial O2 gradient (PAO2 – PaO2), PAO2 ≈ PIO2 - PACO2/RER
- Equilibrium between oxygen in blood and alveoli
- A-a gradient measures efficiency of oxygenation
- PaO2 partially depends on ventilation but more on V/Q matching

Ventilation

- to facilitate CO2 release and maintain normal
- Total amount of gas exhaled/min
- VE= VA+VD
- $\tilde{V}A=\tilde{V}co2 / Pco2 \times K$
- VA=(VT- VD)x f
- PaCO2= k x V'CO2 / V'A

  ή

  PaCO2= k x V'CO2 / VT x f (1- VD/VT)
- VE regulated by brain stem, responding to pH and PaCO2

DO2 = CaO2 x Q

VO2 = Q x (CaO2 – CvO2)

Εισαγωγικά

- Other key manoeuvres to ensure oxygen delivery to the alveolar capillary bed include:
  - Maintaining a satisfactory airway.
  - Ensuring adequate alveolar ventilation.
  - Reversing any respiratory depressants such as narcotics.
  - Invasive ventilation or NIV where necessary.
  - Treating airflow obstruction by bronchodilation or sputum clearance techniques.
  - Optimising transfer factor (diffusion capacity).

Supplemental oxygen therapy

- for all acutely hypoxaemic patients and those at risk of hypoxaemia, including patients with major trauma and shock.
- acute hyperventilation or diabetic ketoacidosis where an apparently breathless patient will not benefit from oxygen therapy.
- carbon monoxide or cyanide poisoning where a patient may benefit from oxygen therapy despite an apparent lack of hypoxaemia or breathlessness
- is given to improve oxygenation but it does not treat the underlying causes

Summary of risks of hyperoxaemia and supplemental oxygen therapy

hyperoxaemia can have deleterious physiological and clinical effects

- **Physiological risks**

1. Worsened V/Q mismatch.
2. Absorption atelectasis.
3. Coronary and cerebral vasoconstriction.
4. Reduced cardiac output.
5. Damage from oxygen free radicals.
6. Increased systemic vascular resistance.

Summary of risks of hyperoxaemia

- Clinical risks

1. Worsening of hypercapnic respiratory failure.
2. Delay in recognition of clinical deterioration.
3. Potentially worse outcomes in mild-to-moderate stroke.
4. Specific risk in patients with previous bleomycin lung damage or with paraquat poisoning or acid aspiration.
5. Unknown risk–benefit balance in acute coronary artery disease with normal oxygen saturation.
6. Association with increased risk of death in survivors of cardiac arrest and among patients on ICUs.
7. Uncontrolled supplemental oxygen therapy can be harmful to patients who are at risk of hypercapnic respiratory failure, especially if the PaO2 is raised above 10 kPa.
8. High-concentration oxygen therapy to produce hyperoxaemia (above normal oxygen saturation) and has been associated with increased risk of death in some patient groups (eg, patients with mild and moderate strokes, survivors of cardiac arrests and ICU patients).

Hyperoxemia

- Hyperoxaemia has been shown to be beneficial in the following clinical situations
  - Carbon monoxide and cyanide poisoning
  - Spontaneous pneumothorax
  - Some postoperative complications
  - Cluster headache

Low-flow oxygen delivery system

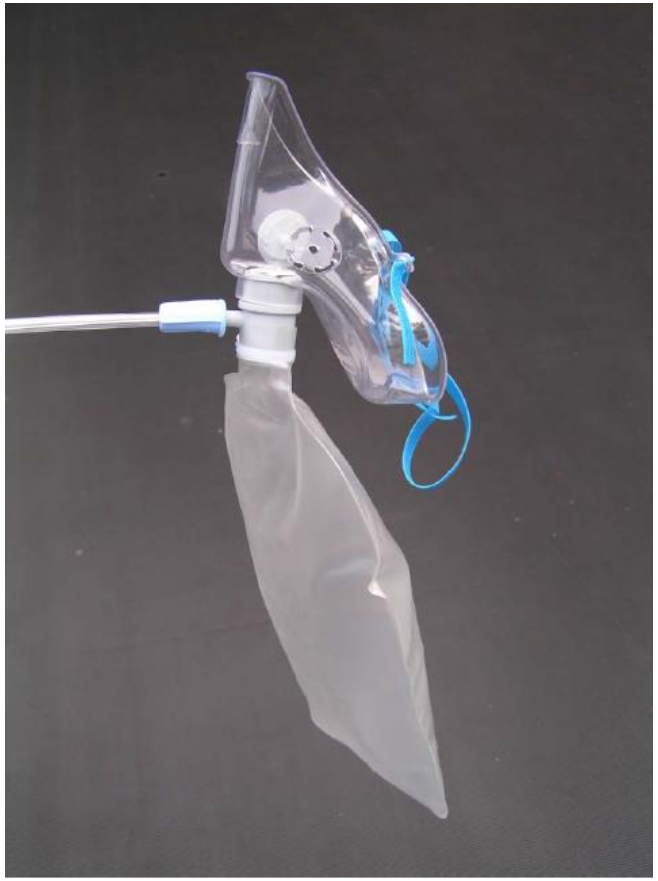
- Nasal cannula (1-6lt/min)
- Simple face mask (5-10lt/min)
- Reservoir mask (15lt/min)

High flow oxygen delivery systems

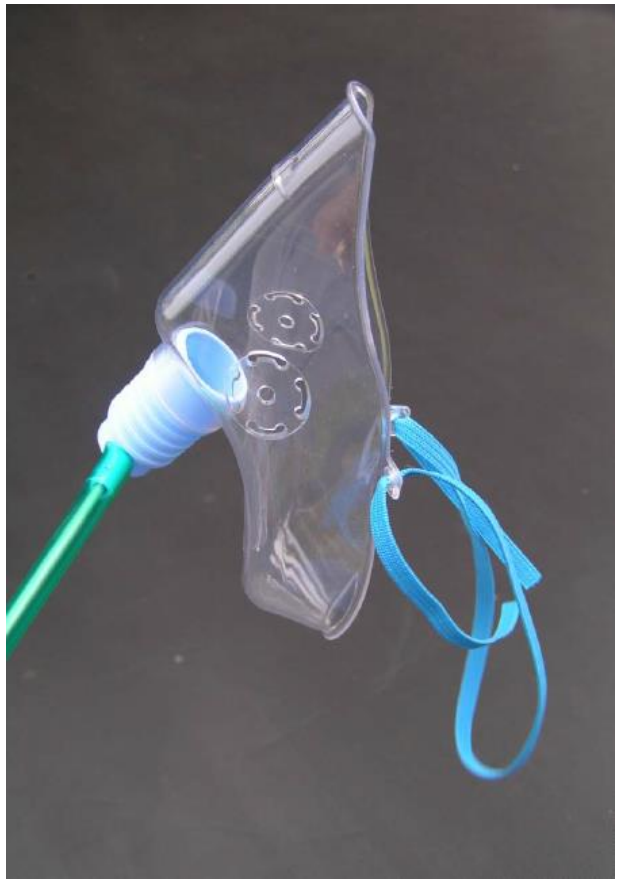
- Venturi mask (24%-60%)
- High flow nasal cannula (εως 100%)

High patient inspiratory flow: Inadequately heated and humidified O2.

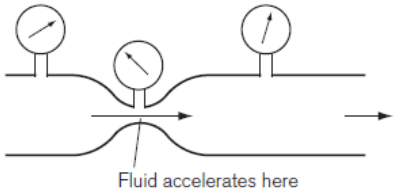
LOW FLOW OXYGEN DEVICES	HIGH FLOW OXYGEN DEVICES
Cannot deliver constant FiO2	Maintain constant FiO2
Flow 6 - 8 L/min	Delivering O2 at very high flow
Mixture of oxygen + room air	Flow usually 4 times the actual Minute volume
FiO2 varies with tidal volume -Shallow breathing = less entrainment of room air (high FiO2)	Used in – treatment of hypoxic patients requiring controlled increments in FiO2
- deep, hyperpneic breathing = more entrainment of room air (less FiO2)	- Young and vigorous patients with hypoxemia, with ventilatory requirement exceeding the capability of low flow systems

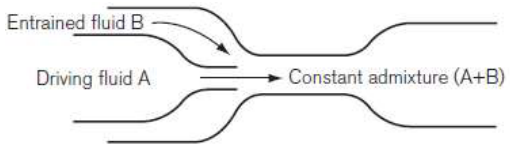


60-90%
15lt/min
Delivered O2
concentration variable



May deliver >50%
5-10lt/min
Respiratory failure I

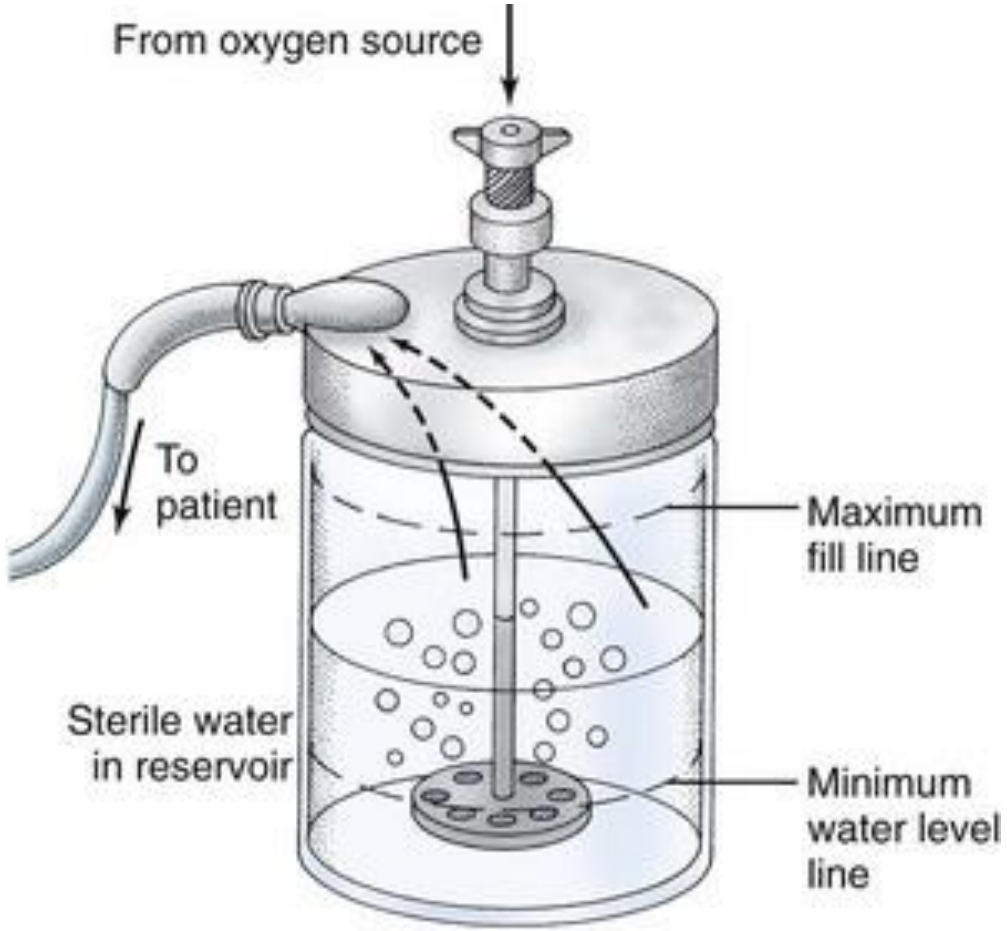




24%-60%
Accurate
concentration of O2
regardless flow rate
Respiratory failure 2

2-6lt/min
FiO2 = 20% + (4 ×
oxygen litre flow)
Mild hypoxia

Κλασσική Οξυγονοθεραπεία



Θερμοκρασία μίγματος αέρα: 10^{o} C

Σχετική υγρασία: 100%

Απόλυτη υγρασία: 9.4 mg / L

↓

Σε ΑΑ οι εισπνευστικές ροές = 30-120 L/min

↓

<10% του εισπνεόμενου αέρα εφυγραίνεται

Ricard et al, Intensive Care Med (2009) 35:963–965

O_2 delivery systems

- Choice of **delivery system** is based upon:
    1. Degree of hypoxemia
    2. Requirement for precision of delivery
    3. Patient comfort
    4. Cost

Table 2 Serious illnesses requiring moderate levels of supplemental oxygen if the patient is hypoxaemic

Section 8.11

The initial oxygen therapy is nasal cannulae at 2–6 L/min (preferably) or simple face mask at 5–10 L/min unless stated otherwise.
For patients not at risk of hypercapnic respiratory failure who have saturation below 85%, treatment should be started with a reservoir mask at 15 L/min and the recommended initial oxygen saturation target range is 94–98%. If oximetry is not available, give oxygen as above until oximetry or blood gas results are available. Change to reservoir mask if the desired saturation range cannot be maintained with nasal cannulae or simple face mask (and ensure that the patient is assessed by senior medical staff). If these patients have coexisting COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88–92% pending blood gas results but adjust to 94–98% if the PCO_2 is normal (unless there is a history of previous hypercapnic respiratory failure requiring NIV or IMV) and recheck blood gases after 30–60 min, see table 4.

	Additional comments	Recommendations
Acute hypoxaemia (cause not yet diagnosed)	Reservoir mask at 15 L/min if initial SpO_2 below 85%, otherwise nasal cannulae or simple face mask Patients requiring reservoir mask therapy need urgent clinical assessment by senior staff.	Recommendations D1–D3
Acute asthma pneumonia lung cancer		Recommendations F1–F3
Deterioration of lung fibrosis or other interstitial lung disease	Reservoir mask at 15 L/min if initial SpO_2 below 85%, otherwise nasal cannulae or simple face mask	Recommendation F4
Pneumothorax	Needs aspiration or drainage if the patient is hypoxaemic. Most patients with pneumothorax are not hypoxaemic and do not require oxygen therapy. Use a reservoir mask at 15 L/min if admitted for observation. Aim at 100% saturation. (Oxygen accelerates clearance of pneumothorax if drainage is not required.)	Recommendations F5–F6
Pleural effusions	Most patients with pleural effusions are not hypoxaemic. If hypoxaemic, treat by draining the effusion as well as giving oxygen therapy.	Recommendation F7
Pulmonary embolism	Most patients with minor pulmonary embolism are not hypoxaemic and do not require oxygen therapy.	Recommendation F8
Acute heart failure	Consider CPAP or NIV in cases of pulmonary oedema.	Recommendations F9–F10
Severe anaemia	The main issue is to correct the anaemia. Most anaemic patients do not require oxygen therapy.	Recommendations F11–12
Postoperative breathlessness	Management depends on underlying cause.	Recommendation J1

Table 3 Conditions for which patients should be monitored closely but oxygen therapy is not required unless the patient is hypoxaemic

Section 8.13

If hypoxaemic, the initial oxygen therapy is nasal cannulae at 2–6 L/min or simple face mask at 5–10 L/min unless saturation is below 85% (use reservoir mask) or if at risk from hypercapnia (see below).
The recommended initial target saturation range, unless stated otherwise, is 94–98%.
If oximetry is not available, give oxygen as above until oximetry or blood gas results are available.
If patients have COPD or other risk factors for hypercapnic respiratory failure, aim at a saturation of 88–92% pending blood gas results but adjust to 94–98% if the PCO_2 is normal (unless there is a history of respiratory failure requiring NIV or IMV) and recheck blood gases after 30–60 min, see table 4.

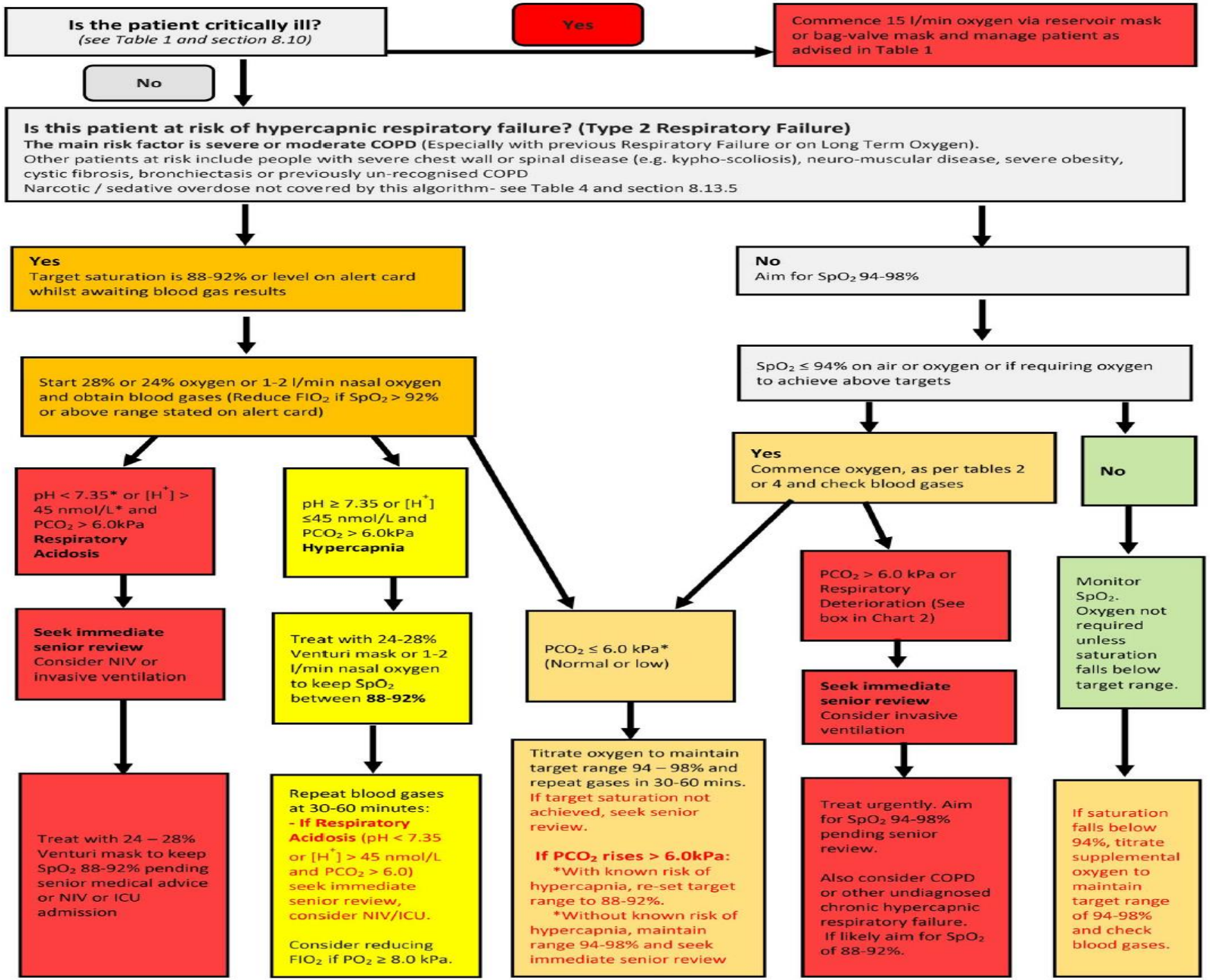
	Additional comments	Recommendations
Myocardial infarction and acute coronary syndromes	Most patients with acute coronary artery syndromes are not hypoxaemic and the benefits/harms of oxygen therapy are unknown in such cases. Unnecessary use of high concentration oxygen may increase infarct size.	Recommendation F13
Stroke	Most patients with stroke are not hypoxaemic. Oxygen therapy may be harmful for non-hypoxaemic patients with mild–moderate strokes.	Recommendation F14
Hyperventilation or dysfunctional breathing	Exclude organic illness. Patients with pure hyperventilation due to anxiety or panic attacks are unlikely to require oxygen therapy. Rebreathing from a paper bag may cause hypoxaemia and is not recommended.	See section 8.13.3
Most poisonings and drug overdoses (see table 1 for carbon monoxide poisoning)	Hypoxaemia is more likely with respiratory depressant drugs, give antidote if available, for example, naloxone for opiate poisoning. Check blood gases to exclude hypercapnia if a respiratory depressant drug has been taken. Avoid high blood oxygen levels in cases of acid aspiration as there is theoretical evidence that oxygen may be harmful in this condition. Monitor all potentially serious cases of poisoning in a level 2 or 3 environment (high dependency unit or intensive care unit).	Recommendation F15
Poisoning with paraquat or bleomycin	Patients with paraquat poisoning or bleomycin lung injury may be harmed by supplemental oxygen. Avoid oxygen unless the patient is hypoxaemic. Target saturation is 85–88%.	Recommendation F16
Metabolic and renal disorders	Most do not need oxygen (tachypnoea may be due to acidosis in these patients)	Recommendation F17
Acute and subacute neurological and muscular conditions producing muscle weakness	These patients may require ventilatory support and they need careful monitoring which includes spirometry. If the patient's oxygen level falls below the target saturation, they need urgent blood gas measurements and are likely to need ventilatory support.	Recommendation G4
Pregnancy and obstetric emergencies	Oxygen therapy may be harmful to the fetus if the mother is not hypoxaemic.	Recommendations H1–H4

Table 4 COPD and other conditions requiring controlled or low-dose oxygen therapy

Section 8.12

Prior to availability of blood gases, use a 24% Venturi mask at 2–3 L/min or 28% Venturi mask at 4 L/min or nasal cannulae at 1–2 L/min and aim for an oxygen saturation of 88–92% for patients with risk factors for hypercapnia but no prior history of respiratory acidosis. Adjust target range to 94–98% if the PCO_2 is normal (unless there is a history of previous NIV or IMV) and recheck blood gases after 30–60 min.

	Additional comments	Recommendations
COPD and other conditions causing fixed airflow obstruction (eg, bronchiectasis)	May need lower range if acidotic or if known to be very sensitive to oxygen therapy. Ideally use 'alert cards' to guide therapy based on previous blood gas results. Increase Venturi mask flow by up to 50% if respiratory rate is above 30 breaths/min.	Recommendations G1–G2 and section 8.12.1
Exacerbation of CF	Admit to regional CF centre if possible, if not discuss with regional centre or manage according to protocol agreed with regional CF centre. Ideally use 'alert cards' to guide therapy. Increase Venturi mask flow by up to 50% if respiratory rate is above 30 breaths/min.	Recommendations G1, G3, G6
Neuromuscular disease, neurological condition and chest wall deformity	May require ventilatory support. Risk of hypercapnic respiratory failure	Recommendations G1, G4, G6
Morbid obesity		Recommendations G1,G5, G6



Cardiac arrest
Major trauma
shock
Major sepsis
Drowning
Anaphylaxis
Major pulmonary haemorrhage
Epileptic fits
Major trauma injury
CO poisoning

Thorax 2017;72:i1–i90

Monitoring

Blood gases should be checked in the following situations

• All critically ill patients (grade D).

• Unexpected or inappropriate fall in SpO2 below 94% in patients breathing air or oxygen or any patient requiring oxygen to achieve the above target range (grade D).

• Deteriorating oxygen saturation (fall of ≥3%) or increasing breathlessness in a patient with previously stable chronic hypoxaemia (eg, severe COPD) (grade D).

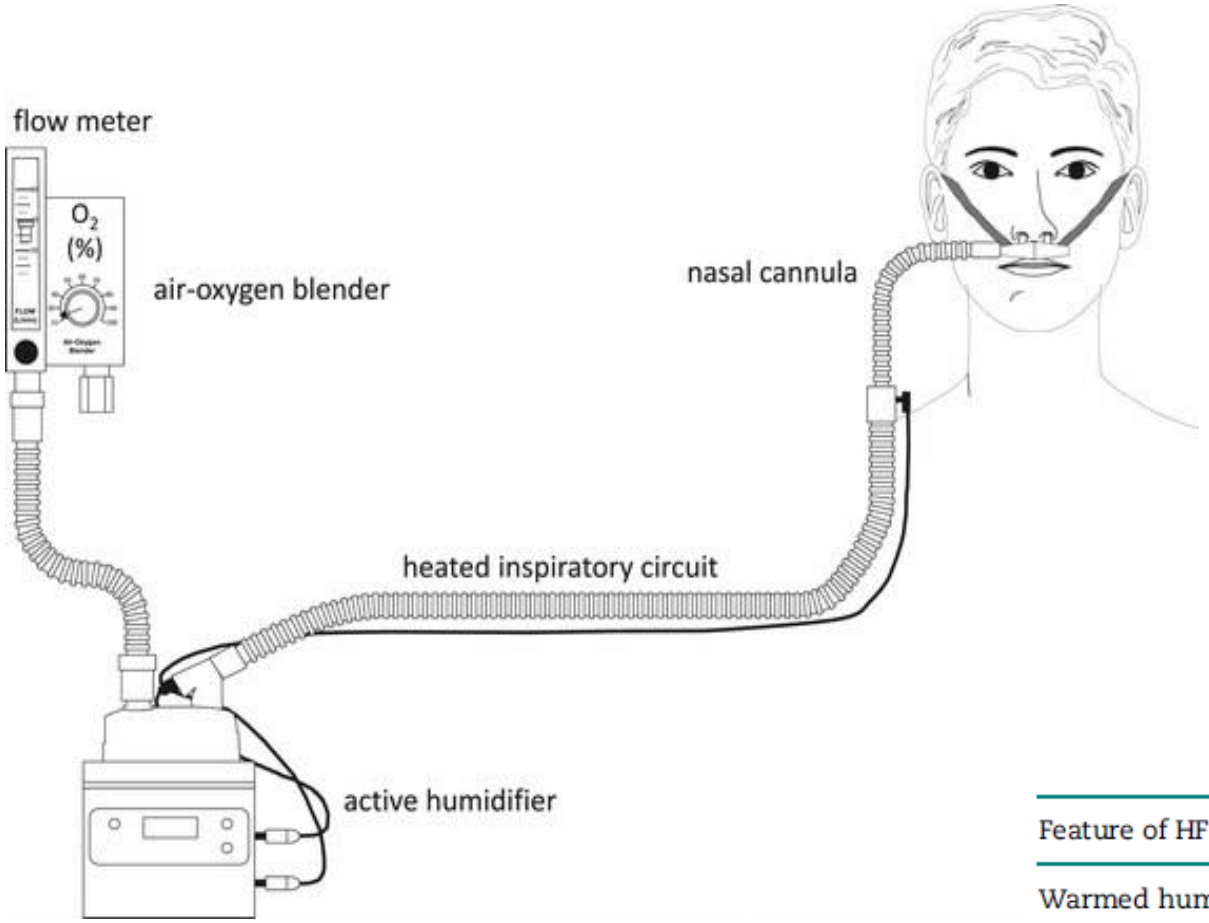
• Most previously stable patients who deteriorate clinically and require increased FiO2 to maintain a constant oxygen saturation (grade D).

• Any patient with risk factors for hypercapnic respiratory failure who develops acute breathlessness, deteriorating oxygen saturation, drowsiness or other features of carbon dioxide retention (grade D).

• Patients with breathlessness who are thought to be at risk of metabolic conditions such as diabetic ketoacidosis or metabolic acidosis due to renal failure (grade D).

Thorax 2017;72:i1–i90

HFNC



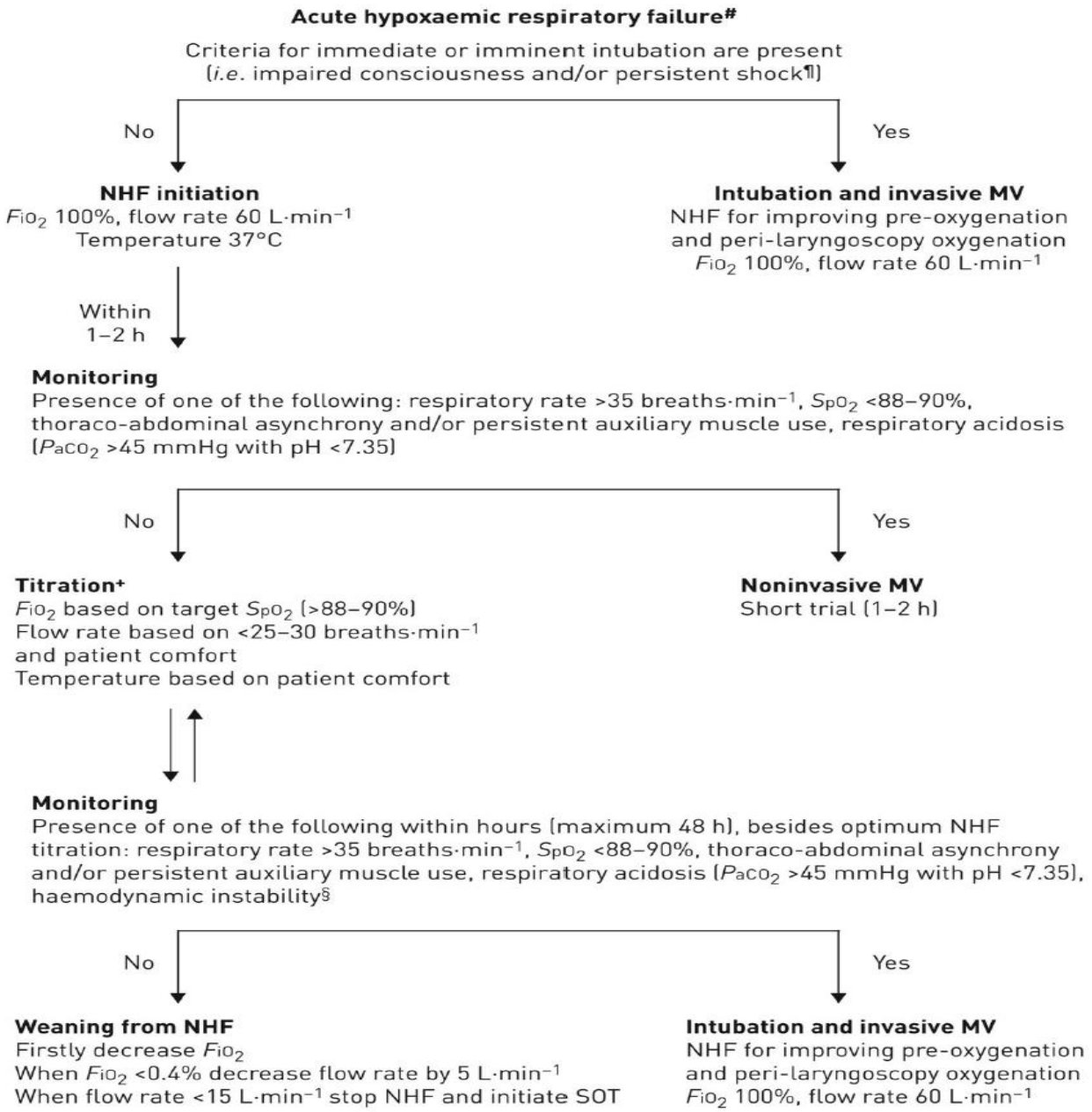
Feature of HFNOT	Physiological effect
Warmed humidified gas	Reduced airway surface dehydration Improved secretion clearance Decreased atelectasis CO_2 washout, reduction in anatomical dead space
Gas flow of up to 60 litre min^{-1}	Provides an oxygen reservoir Allows $F_{I_{O_2}}$ close to 1.0 to be delivered
PEEP	Increased end-expiratory lung volume Alveolar recruitment

HFNC

	Clinical effects
Acute hypoxaemic respiratory failure	• Reduces dyspnoea • Improves oxygenation • Decreases escalation to invasive support
Hypoxaemic failure in immunocompromised patients	• Reduces dyspnoea • Improves oxygenation • Reduces intubation rate • Reduces mortality (?)
Cardiogenic pulmonary oedema	• Improves oxygenation • Reduces cardiac afterload
Preoxygenation	• higher peripheral oxygen saturations • reduced lowest saturation
Postexubation	• Improves gas exchange • Decreases reintubation

✓ High-flow nasal oxygen should be considered as an alternative to reservoir mask treatment in patients with acute respiratory failure without hypercapnia.

Thorax 2017;72:i1–i90



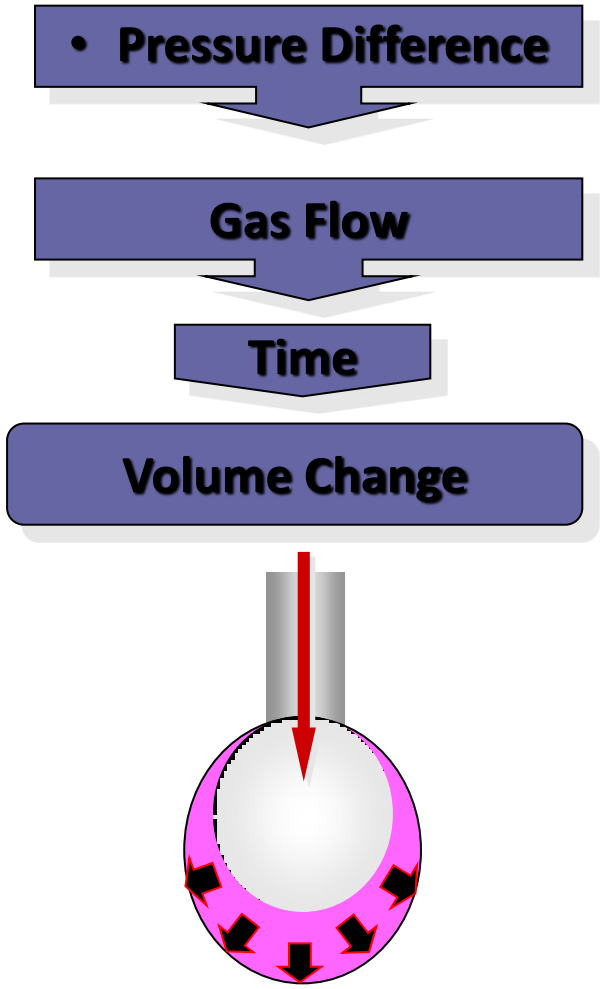
The **ROX Index** may be useful in identifying those patients with acute pneumonia and respiratory failure receiving HFN that are risk of failure and intubation.

ROX Index = SpO2 / FiO2 /RR
ROX Index ≥ to 4.88 measured at 2, 6, or 12 hours suggests the success high flow nasal therapy.
ROX Index < 2.85 at 2 hours, < 3.47 at 6 hours, or 3.85 at 12 hours is predictive of the need for intubation

Am J Respir Crit Care Med 2019;199: 1368-1376.

Eur Respir Rev 2017; 26: 170028.

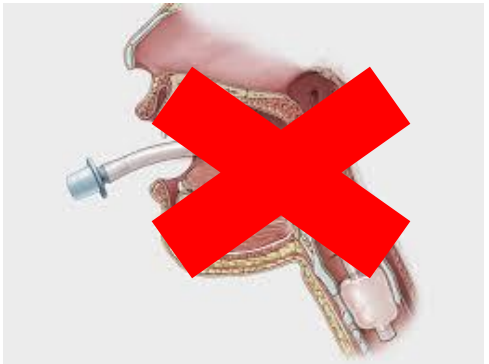
Mechanical ventilation



- Ventilators deliver gas to the lungs using positive pressure at a certain ***rate***.
- The amount of gas delivered can be ***limited*** by time, pressure or volume.
- The duration can be ***cycled*** by time, pressure or flow

NIV

NIV is the delivery of mechanical ventilation to the lung using techniques that do not require an endotracheal airway



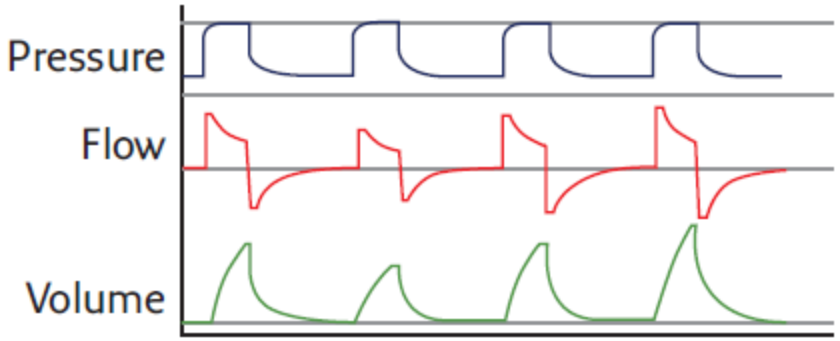
Ventilatory modes

- Pressure preset ventilators:

  pressure is constant;

  flow and volume differ

  with each breath.

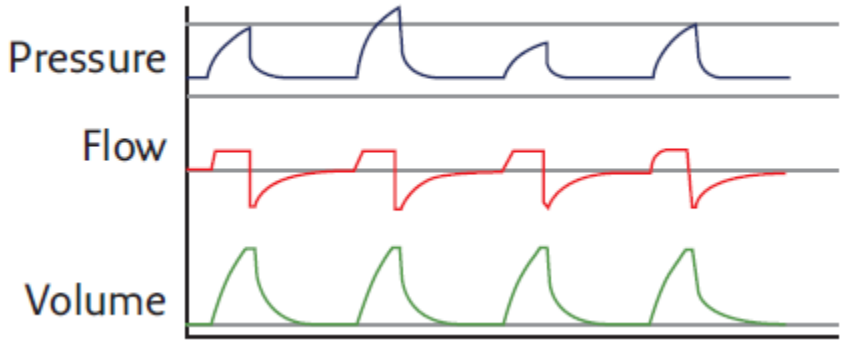


- Volume-preset ventilators*:*

  volume and flow are constant

  pressure is variable

  with each breath.



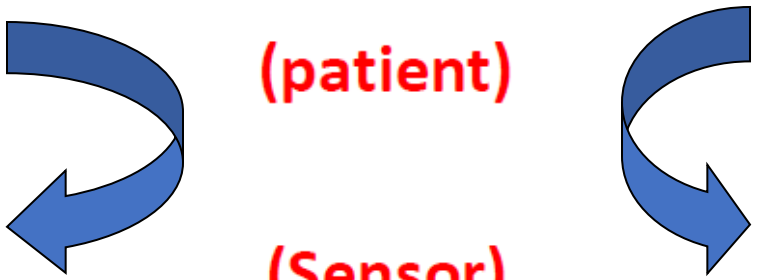
Pressure preset modes

- **Spontaneous mode (S) [Pressure Support]**
  - ➢ Triggered by patient, flow cycled
- **Timed (T) [Pressure Control]**
  - ➢ Triggered by ventilator (Time), Time cycled
- **Spontaneous/ timed (S/T)**
  - ▪ Pressure Support
  - ▪ back-up pressure control (PC)
  - ➢ Triggered by patient - or ventilator

S/T

Interchangeable mode

Not breathing



(patient)

(Sensor)

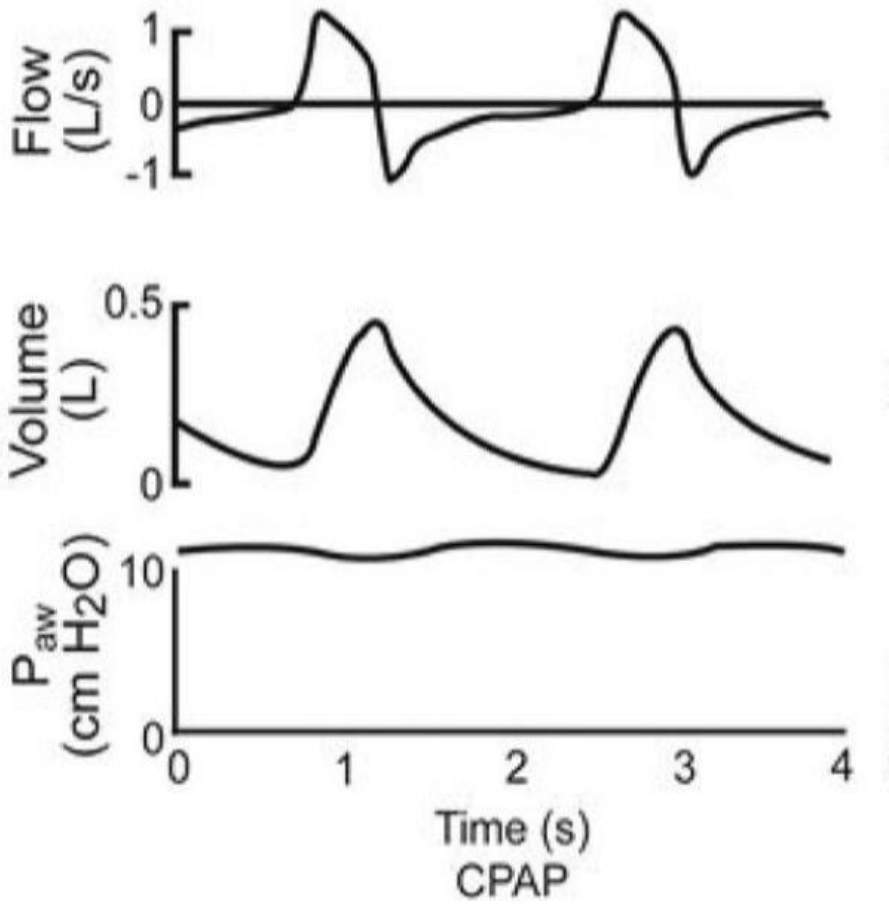
Actively breathing

- **Back-up pressure-controlled mode**
- **Time cycled**

(ventilator)

- **Pressure Support (IPAP)**
- **Flow cycled**

CPAP

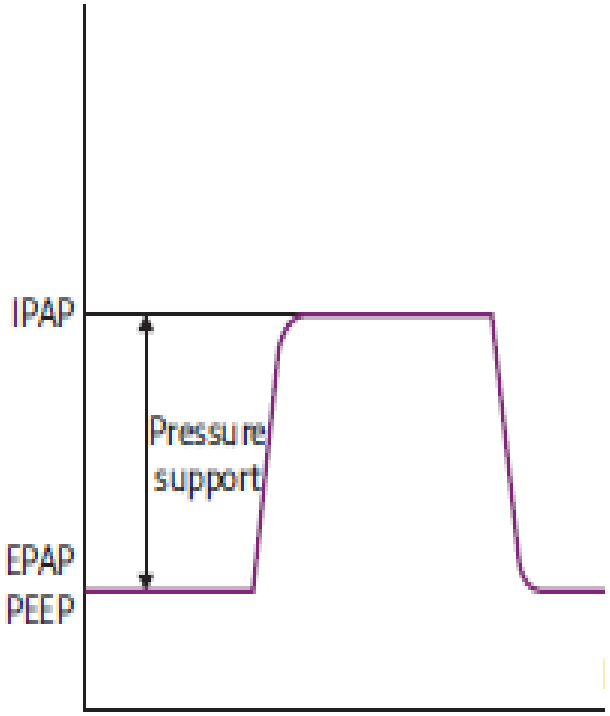


- CPAP provides a single continuous pressure throughout the respiratory cycle.
- If the patient fails to make a spontaneous respiratory effort, the ventilator will continue to deliver a constant pressure

Inspiratory positive airway pressure IPAP : the pressure delivered by the ventilator while the patient is inhaling

Expiratory positive airway pressure EPAP: the pressure delivered by the ventilator while the patient is exhaling

Pressure support : IPAP minus EPAP



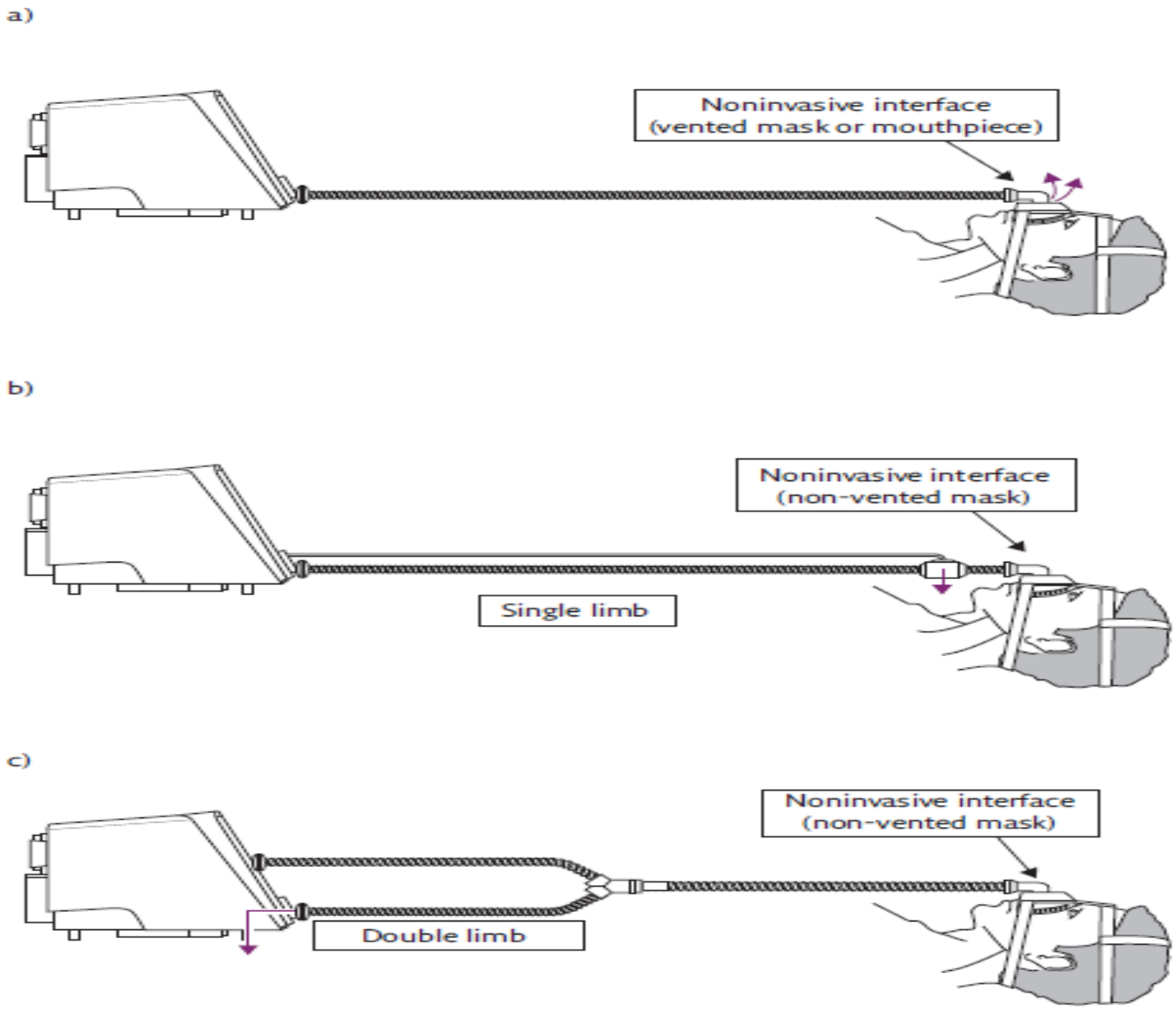
IPAP

- provides assistance to inspiration
- decreases the work of breathing by unloading the respiratory muscles
- increased *VT and minute ventilation,* and improved gas exchange

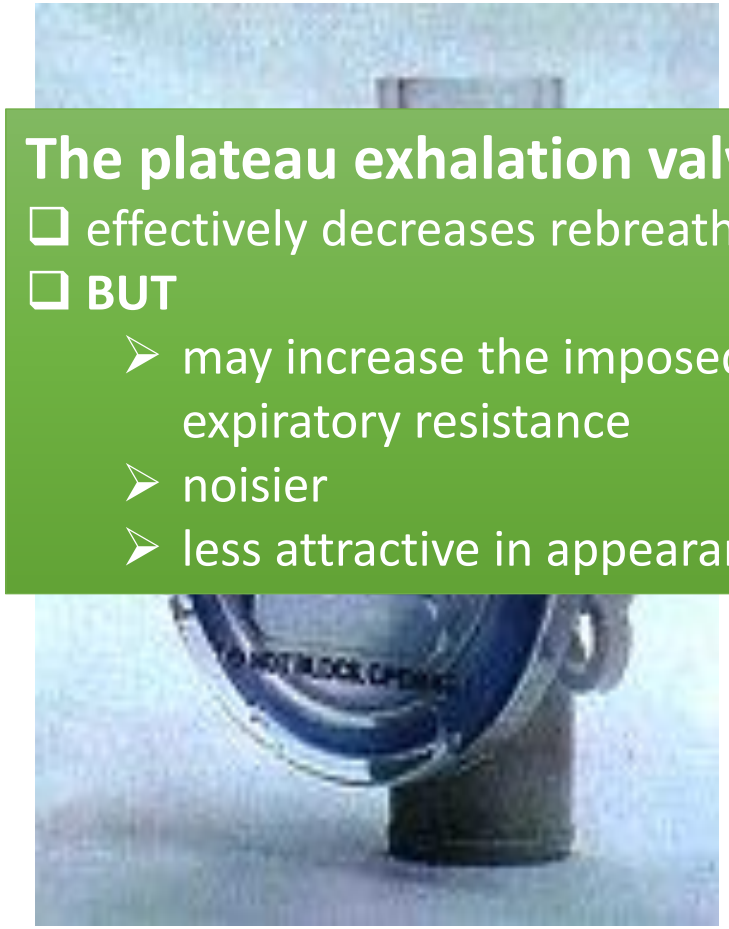
EPAP

- assists with the maintenance of upper airway patency in sleep
- helps to recruit/maintain lung volume, improving oxygenation
- helps to overcome the intrinsic PEEP, reducing the work of breathing and maximising effective triggering
- is necessary to ensure sufficient expiratory flow to flush carbon dioxide from the ventilatory dead space

The circuit


a)
Noninvasive interface
(vented mask or mouthpiece)
b)
Noninvasive interface
(non-vented mask)
Single limb
c)
Noninvasive interface
(non-vented mask)
Double limb

Single Limb –
Non vented circuit



The plateau exhalation valve

- ❑ effectively decreases rebreathing
- ❑ **BUT**
  - ➢ may increase the imposed expiratory resistance
  - ➢ noisier
  - ➢ less attractive in appearance

Plateau exhalation valve

Single Limb –
Vented circuit

Whisper Swivel

Safety considerations include:

- CO_2 rebreathing
- the risk of asphyxiation in case of vomiting
- the risk of asphyxiation in case of ventilator malfunction
- the patient's level of consciousness and ability to remove the mask autonomously
- potential disease transmission

Patients with highly transmittable airborne disease

- the use of a **non-vented mask**
- **covering the nose and mouth**
- within a **non-vented tubing circuit**
- and **filtering** of the **exhaled gases**

can reduce environmental spread

When should NIV be started?

The indication for mechanical ventilation in general must be determined

Important **clinical signs** are:

- respiratory distress;
- rapid shallow beathing;
- tachypnoea;
- accessory muscle use.

The use of NIV should be evidence

The patient should have no contraindications for NIV

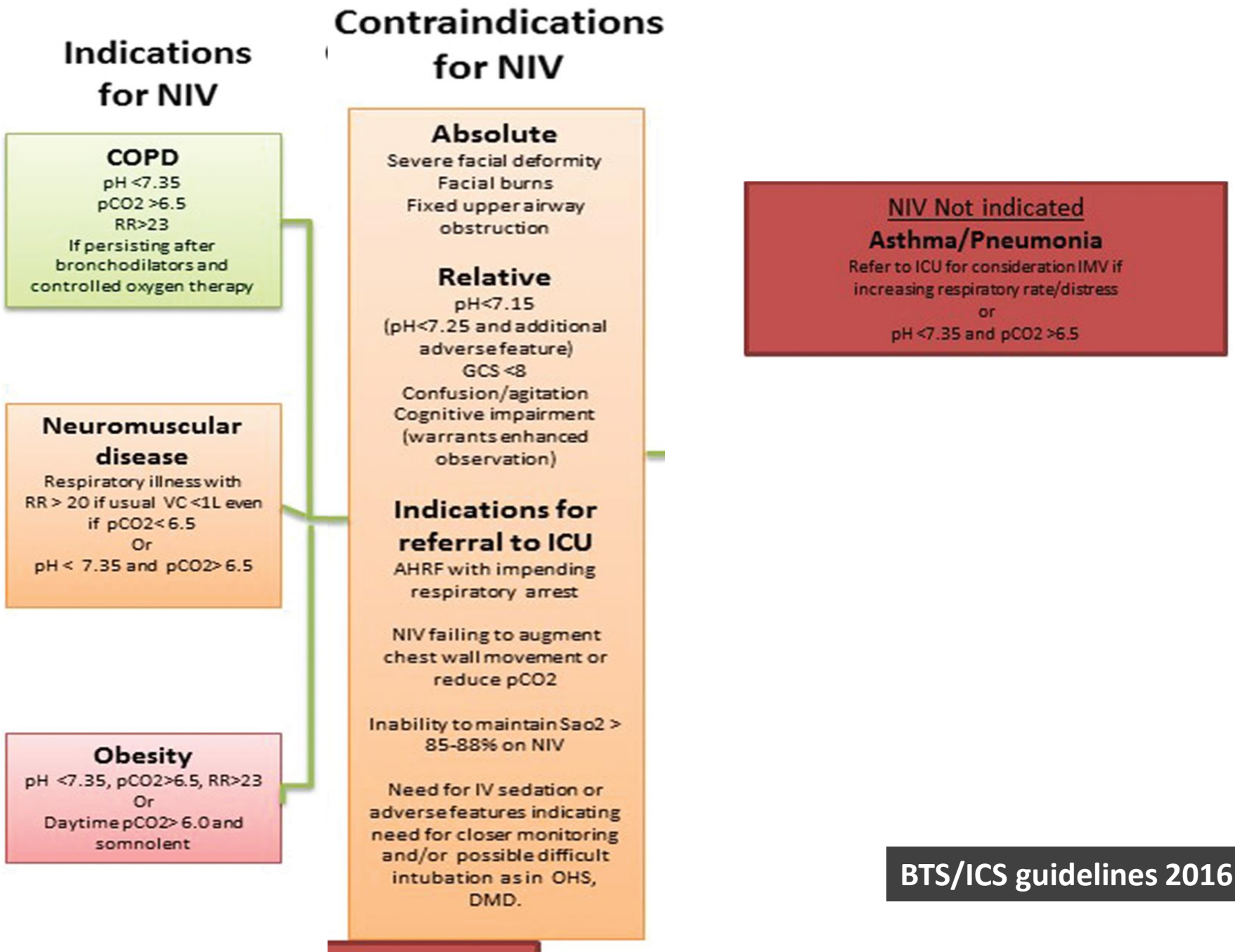
Patients should not suffer from diagnoses in which it has been shown that NIV is not effective

(*e.g. severe, rapidly progressive ARDS).*

The patient's wishes and advance directives should also be considered; some patients decide not to receive NIV

In all cases decide in advance what you will do if NIV fails:

- is the patient suitable for intubation and invasive ventilation
- is NIV the ceiling of care?



BTS/ICS guidelines 2016

Initiation of NIV

- **Alarms off**
- **Low pressures**
- **Adjust IPAP and EPAP according**
  - **to comfort**
  - **muscle rest**
  - **quality of ventilation**
- **Seek and correct leaks**
- **Assure patient-ventilator synchrony**
- **Take time**

NIV SETUP

Mask

Full face mask (or own if home user of NIV)

Initial Pressure settings

EPAP: 3 (or higher if OSA known/expected)

IPAP in COPD/OHS/KS 15 (20 if pH <7.25)

Up titrate IPAP over 10-30 mins to IPAP 20—30 to achieve adequate augmentation of chest/abdo movement and slow RR

IPAP should not exceed 30 or EPAP 8* without expert review

IPAP in NM 10 (or 5 above usual setting)

Backup rate

Backup Rate of 16-20. Set appropriate inspiratory time

I:E ratio

COPD 1:2 to 1.3
OHS, NM & CWD 1:1

Inspiratory time

0.8-1.2s COPD
1.2-1.5s OHS, NM & CWD

Use NIV for as much time as possible in 1st 24hours.
Taper depending on tolerance & ABGs over next 48-72 hours

SEEK AND TREAT REVERSIBLE CAUSES OF AHRF

* Possible need for EPAP > 8

Severe OHS (BMI >35), lung recruitment eg hypoxia in severe kyphoscolios, oppose intrinsic PEEP in severe airflow obstruction or to maintain adequate PS when high EPAP required

BTS/ICS guidelines 2016

Monitoring during NIV

BTS/ICS guidelines 2016

- Monitoring- Good practice points

❑ **Oxygen saturation** should be **continuously** monitored.

❑ **Intermittent measurement of pCO2 and pH** is required.

❑ **ECG monitoring** is advised if
  - the patient has a pulse rate >120 bpm
  - if there is dysrhythmia
  - possible cardiomyopathy.

Invasive Mechanical Ventilation

- Apnea, respiratory failure.
- Airway obstruction: variable-level obstruction in the upper and lower airways.
- Inadequate oxygenation (hypoxia), inadequate ventilation (hypercarbia).
- Disruption of the airway reflex.
- In case the patient is hemodynamically unstable.
- The consciousness changes as far as being unable to protect airway (GCS <8).
- Cardiopulmonary resuscitation.
- Flail chest/pulmonary contusion, in case the breathing effort puts the patient's life in danger. In case the treatment of patient is not successful without intubation.

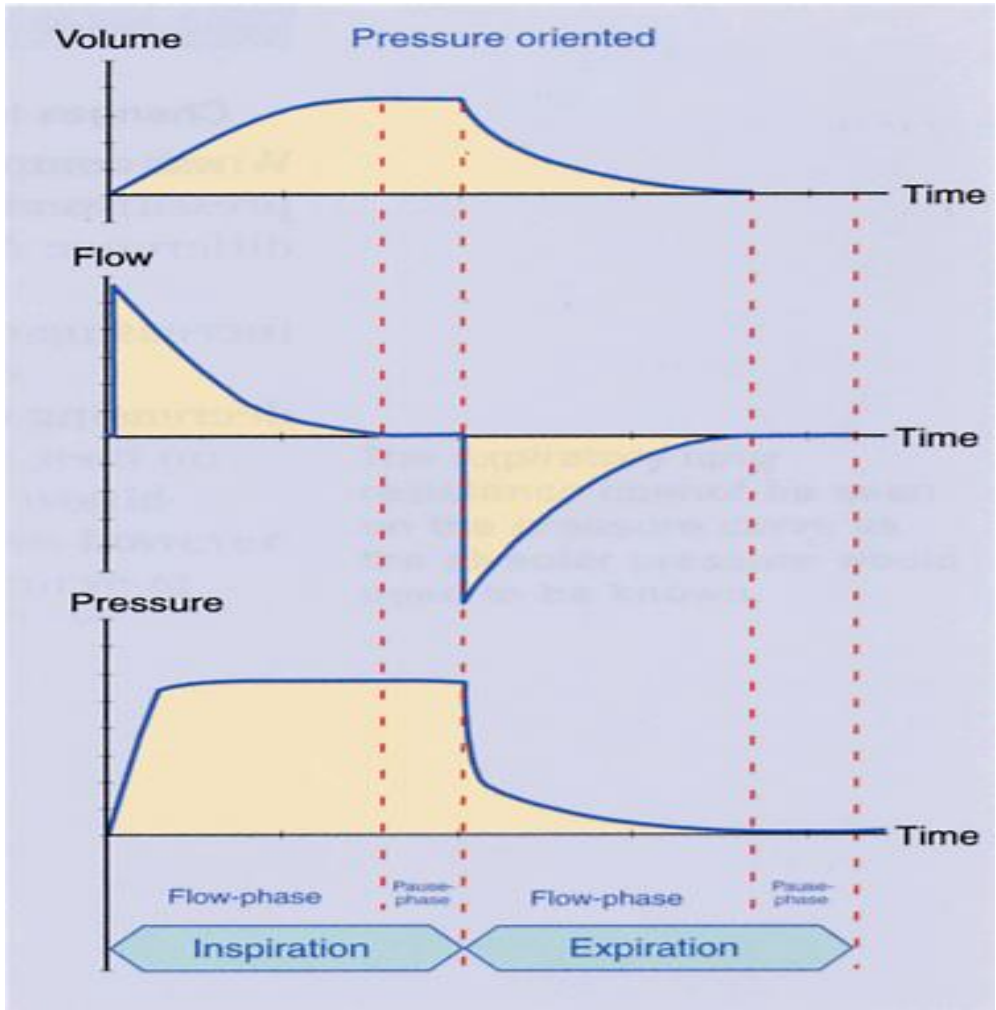
Indication for Intubation

- **Criteria**
  - Clinical deterioration
  - Tachypnea: RR >35
  - Hypoxia: pO_2<60mm Hg
  - Hypercarbia: pCO_2 > 55mm Hg
  - Minute ventilation>10 L/min
  - Tidal volume <5-10 ml/kg
  - Negative inspiratory force < 25cm H_2O (how strong the pt can suck in)

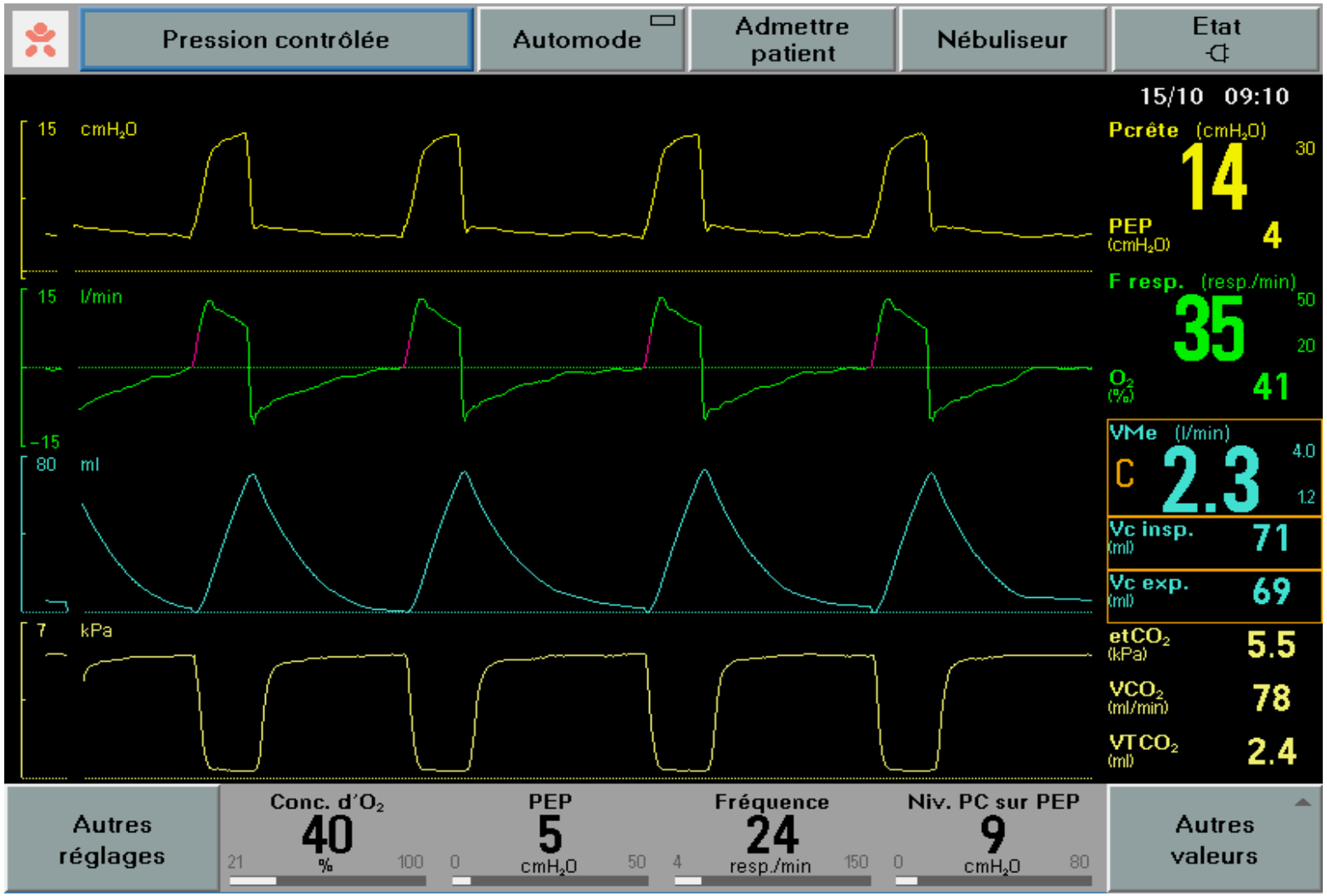
Pressure ventilation vs. volume ventilation

- **Pressure-cycled** modes deliver a fixed pressure at variable volume
- **Volume-cycled** modes deliver a fixed volume at variable pressure
- **Pressure-cycled modes**
  - Pressure Support Ventilation (PSV)
  - Pressure Control Ventilation (PCV)
  - CPAP
  - BiPAP
- **Volume-cycled modes**
  - Control
  - Assist
  - Assist/Control
  - Intermittent Mandatory Ventilation(IMV)
  - Synchronous Intermittent Mandatory Ventilation (SIMV)

Pressure control



Pressure control



Pressure Control Ventilation (PCV)

- Ventilator determines inspiratory time – no patient participation

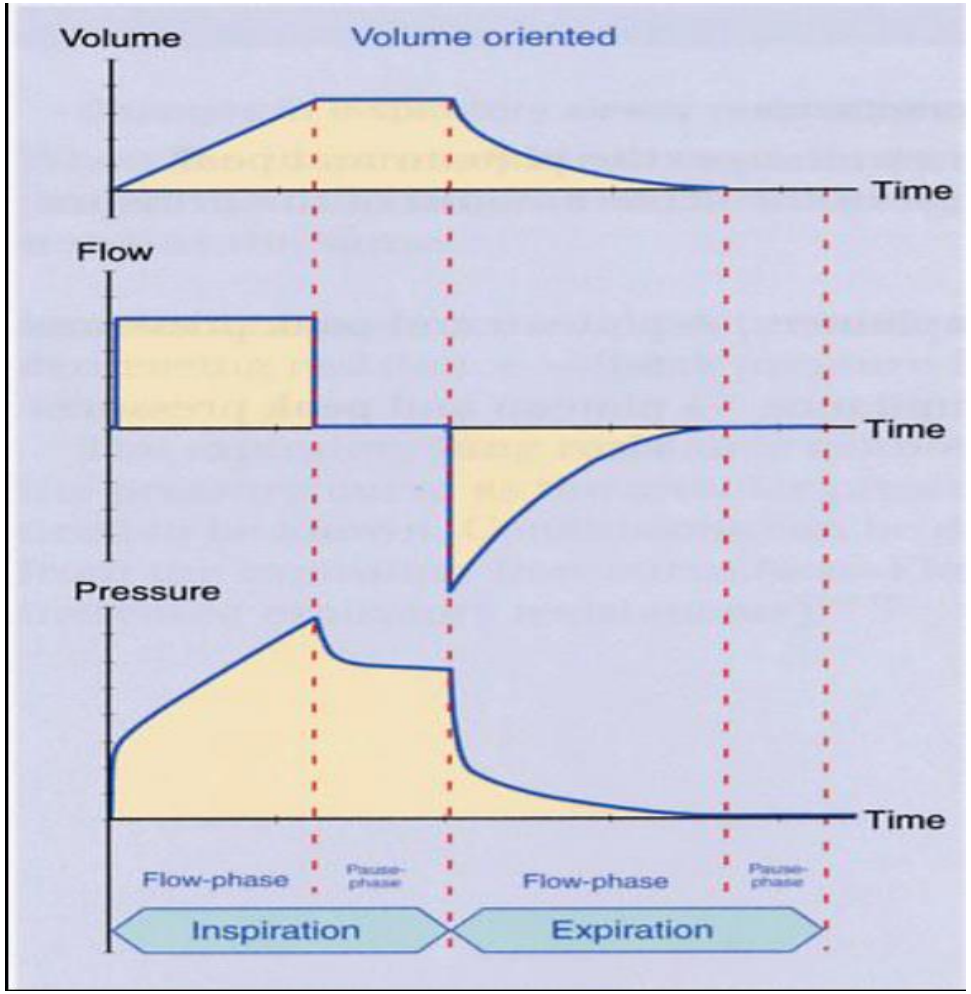
Parameters

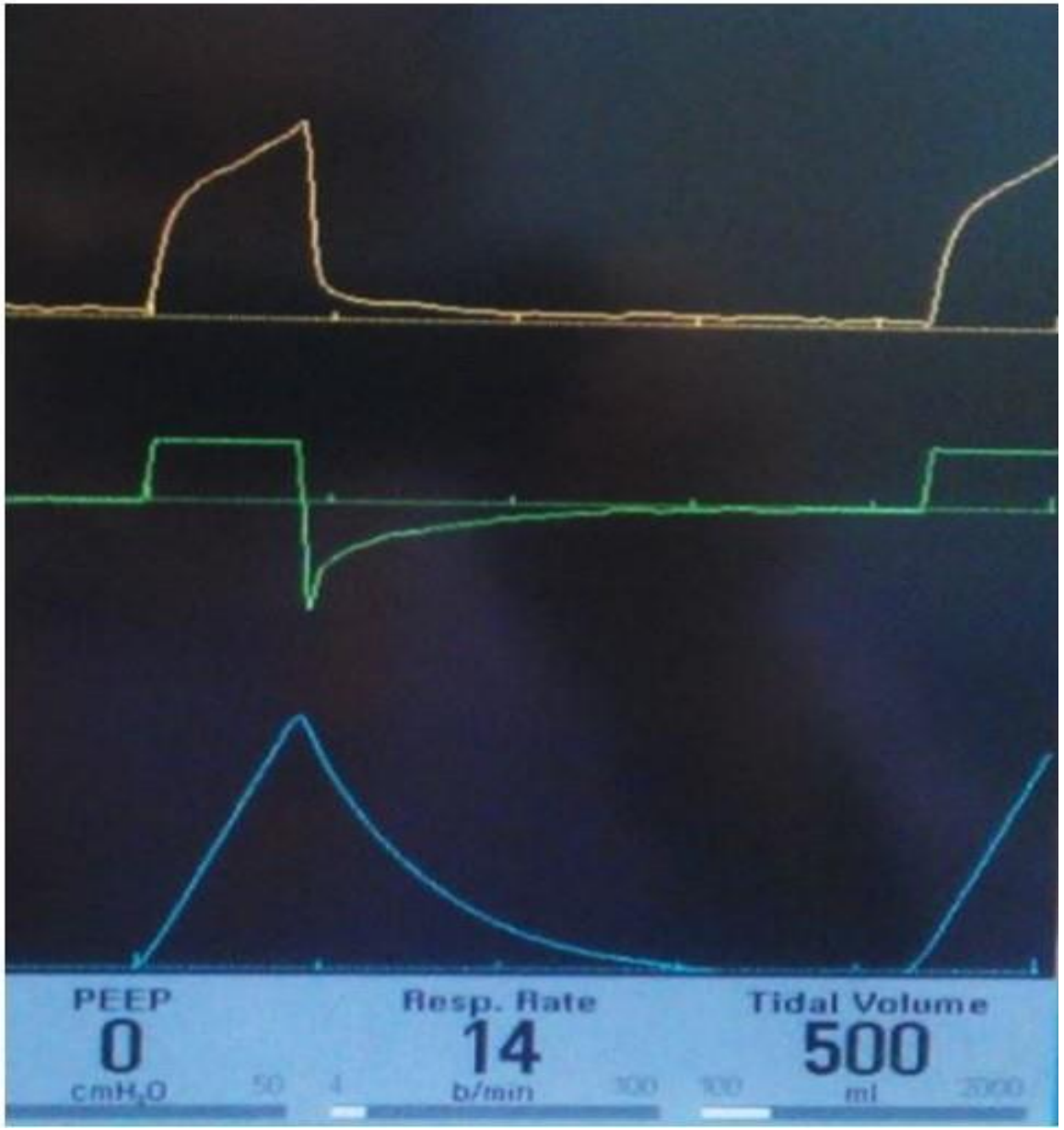
- Triggered by time
- Limited by pressure
- Affects inspiration only

Disadvantages

- Requires frequent adjustments to maintain adequate VE
- Pt with noncompliant lungs may require alterations in inspiratory times to achieve adequate TV

Volume control





Volume control

- sets the flow pattern, flow rate, trigger sensitivity, tidal volume, respiratory rate, positive end-expiratory pressure (PEEP), and fraction of inspired oxygen (FiO_2)
- guarantee flow and tidal volume, while airway pressures are variable.
- VT=4-6ml/kg, Pplateau=30cmH_20

Vent settings to improve <oxygenation>

PEEP and FiO2 are adjusted in tandem

- **FIO_2**
  - Simplest maneuver to quickly increase PaO2
  - Long-term toxicity at >60%
    - Free radical damage

- **Inadequate oxygenation despite 100% FiO2 usually due to pulmonary shunting**
  - Collapse – Atelectasis
  - Pus-filled alveoli – Pneumonia
  - Water/Protein – ARDS
  - Water – CHF
  - Blood - Hemorrhage

Vent settings to improve <oxygenation>

- **PEEP**
- **Increases FRC**
  - Prevents progressive atelectasis and intrapulmonary shunting
  - Prevents repetitive opening/closing (injury)
- **Recruits collapsed alveoli and improves V/Q matching**
  - Resolves intrapulmonary shunting
  - Improves compliance
- **Enables maintenance of adequate PaO2 at a safe FiO2 level**

- **Disadvantages**
  - Increases intrathoracic pressure (may require pulmonary a. catheter)
  - Rupture: PTX, pulmonary edema

Vent settings to improve <ventilation>

RR and TV are adjusted to maintain VE and PaCO2

- Respiratory rate
  - Max RR at 35 breaths/min
  - Efficiency of ventilation decreases With increasing RR
  - Decreased time for alveolar emptying
- TV
  - Goal of 8 ml/kg
  - Risk of volutrauma
- Other means to decrease PaCO2
  - Reduce muscular activity/seizures
  - Minimizing exogenous carb load
  - Controlling hypermetabolic states
- Permissive hypercapnea
  - Preferable to dangerously high RR and TV, as long as pH > 7.15

- I:E ratio (IRV)
  - Increasing inspiration time will increase TV, but may lead to autoPEEP
- PIP
  - Elevated PIP suggests need for switch from volume-cycled to pressure-cycled mode
  - Maintained at <45cm H2O to minimize barotrauma
- Plateau pressures
  - Pressure measured at the end of inspiratory phase
  - Maintained at <30-35cm H2O to minimize barotrauma

During CPR

- Maximal feasible inspired O_2 concentration.
- Expired air ventilation is effective, but
  - 16-17% O_2 concentration.
  - must be replaced as soon as possible.
- Self inflating mask
  - avoid high airway pressure predispose to gastric inflation.
  - avoid too low inspiratory flow prolonged inspiratory time reduced time available to chest compression.
  - deliver each breath over 1 sec,
  - 30:2
  - sniffing position, lift the jaw, tight seal, blow gently, observe the chest, use O2 if available at 15lt/min.
  - not try to compensate for leak by excessive compression of the bag.
  - adjust contact pressure, altering position of finger, thumbs, increase jaw thrust.
  - 2-person technique is preferable.

During CPR

- Tracheal intubation provides the most reliable airway.
- Properly trained, regular, ongoing experience with the technique.
- Laryngoscopy and intubation without stopping chest compression.
- Brief pause when tube is passed through vocal cord, <5 sec.
- Altrenatively, intubation after ROSC.
- No studies have shown that tracheal intubation increases survival after cardiac arrest.
- Confirm correct tube, ideally with capnography and secured it.
- Ventilate at 10 breaths/min, VT 500-600 ml, do not hyperventilate.
- Chest compression without pausing during ventilation.
- In the absence of personnel skilled in tracheal intubation: supraglottic airway.

After ROSC

- Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function.
- SpO2 94-98%.
- Normocapnia.
- Rational to apply protective ventilation: VT 6-8ml/kg, PEEP 4-8cmH_2O.
- Lowering the body temperature.

Συμπερασματικά

- Στόχος SpO2: 94-98%, Υπερκαπνία: 88-90%
- Αποφυγή υπεροξαιμίας
- HFNC: AAI
- NIV: AAII

Έγκαιρα IMV

- Προστατευτικός αερισμός
- CPR: μέγιστο O2, δεν υπεραερίζουμε
- ROSC: SpO2: 94-98%, νορμοκαπνία

ΕΥΧΑΡΙΣΤΩ