Non-invasive ventilation

“And the Lord God formed a man from the dust of the ground, and breathed into his nostrils the breath of life, and the man became a living being.”

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Non-invasive ventilation (NIV)

NIV is increasingly used in intensive care units; yet, more patients are also dismissed from these units with a mask and a ventilator and even leave hospital with this treatment.

Course outline:

- General principles of NIV
- NIV for acute respiratory failure
- NIV for chronic respiratory failure
Non-invasive ventilation (NIV)

General Principles

What is NIV: definition

Any form of ventilatory support applied without use of a tracheal tube, laryngeal mask or tracheostomy:

- CPAP
- Volume < - > pressure cycled systems
NIV: aims (1)

= aims of “conventional” invasive ventilation: deliver positive airway pressure

1. to unload and support the respiratory muscles resulting in an increased alveolar ventilation and better gas exchange (resulting especially in PCO\(_2\) decrease and to a lesser amount also in PO\(_2\) increase)

2. to further improve pulmonary gas exchange (especially PO\(_2\) increase) by recruitment of underventilated alveoli

The rationale for using NIV is to reduce complications related to endotracheal tubing

Complications of invasive ventilation

<table>
<thead>
<tr>
<th>Related to tube insertion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspiration of gastric contents</td>
</tr>
<tr>
<td>Trauma of teeth, pharynx, oesophagus, larynx, trachea</td>
</tr>
<tr>
<td>Sinusitis (nasotracheal intubation)</td>
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<tr>
<td>Need for sedation</td>
</tr>
<tr>
<td>Related to mechanical ventilation</td>
</tr>
<tr>
<td>Arrhythmias and hypotension</td>
</tr>
<tr>
<td>Barotrauma</td>
</tr>
<tr>
<td>Related to tracheostomy</td>
</tr>
<tr>
<td>Haemorrhage</td>
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<td>Trauma of trachea and oesophagus</td>
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<tr>
<td>False lumen intubation</td>
</tr>
<tr>
<td>Stomal infections and mediastinitis</td>
</tr>
<tr>
<td>Tracheomalacia, tracheal stenosis and granulation tissue formation</td>
</tr>
<tr>
<td>Tracheo-oesophageal or tracheo-esophageal fistula</td>
</tr>
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</table>

VAP = Ventilator associated pneumonia (nosocomial pneumonia)

Caused by loss of airway defence mechanisms:

- Airway colonization with Gram-negative bacteria
- Pneumonia

Occurring after removal of the endotracheal tube:

- Hoarseness, sore throat, cough and sputum
- Haemoptysis
- Vocal cord dysfunction and laryngeal swelling

For every intubated day, the patient has a 1% risk of developing VAP.
Pathogenic mechanisms of VAP

- Endotracheal tubes
  - Associated mucosal injury / elimination of cough reflex
  - Pooling of contaminated secretions above the ET tube cuff
  - Biofilm formation
  - Route of intubation (nose → sinusitis)

- Nasogastric tubes / enteral nutrition
  - Gastro-esophageal reflux
  - Aspiration to lower airways

- Ventilator circuit and respiratory therapy equipment
  - Contamination (from patients’ secretions)

Pooling of contaminated secretions above the tube cuff

Leakage of dye along the folds of the cuff
Formation of Biofilm

Frequency of nosocomial infections in patients receiving NIV and invasive MV

- Patients with hypercapnic respiratory failure:
  - COPD exacerbation
  - Cardiogenic pulmonary edema
- Case-control study:
  - 50 with NIV
  - 50 with conventional MV

Girou. JAMA 2000
NIV: aims (2)

= aims of “conventional” invasive ventilation: by increasing airway pressure

1. to unload and support the respiratory muscles resulting in an increased alveolar ventilation and better gas exchange (resulting especially in PCO\textsubscript{2} decrease and to a lesser amount also in PO\textsubscript{2} increase)
2. to further improve pulmonary gas exchange (especially PO\textsubscript{2} increase) by recruitment of underventilated alveoli

The rationale for using NIV is to reduce complications related to endotracheal tubing and to enhance patient’s management outside the ICU and increase comfort

General principles: aims

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NIV enhances patient’s comfort

- Avoiding or reducing the need for sedation
- Allowing for communicating
- Allowing for eating, drinking
- Allowing for cough and more adequate chest physiotherapy
NIV: Limitations

- Inappropriately prolonged NIV may delay intubation, resulting in a worse outcome
  - monitor patients and work together with the department for invasive ventilation (see below)
  - DNR strategy should be clear before starting NIV

- The mask interface may be claustrophobic

- Cave: pressure sores, usually over the nasal bridge

General principles: limitations

NIV pressure sores

Preventive: micro-foam

Curative: mepilex or comfeel
NIV: Contraindications

- Cardiac or respiratory arrest
- Fixed upper airway obstruction
- Recent facial and skull surgery or trauma/burns
- Recent upper airway/upper gastrointestinal (esophageal) tract surgery
- Severe encephalopathy (GCS<8); uncooperative / agitated patient
- Severe inability to cough or clear secretions
- Vomiting/bowel obstruction
- (Undrained pneumothorax)

No absolute contraindications. Most of the contraindications are derived from exclusion criteria for the RCTs. Therefore, it is more correct to state that NIV benefit has not been proven in these circumstances.

NIV: ventilatory modes

- CPAP
- BPAP
- CMV
- PS
- ACV
- PC
- CV
- AVAPS
ICU ventilator not that suitable!

- The presence of gas leaks is a near-constant feature of NIV and may affect triggering of the ventilator.

- ICU ventilators are not able to cope with large leaks in order to avoid asynchrony and auto-triggering; while special NIV ventilators can adequately compensate large gas leaks.

Tassaux. Intensive Care Med 2002
Miyoshi. Chest 2005

General principles: ventilatory modes
NIV: ventilatory modes

- CPAP
- Pressure
  - BPAP (other pressure ventilators)
    - S
    - PS
    - S/T
    - (T)
    - (A)PCV
  - (Volume
    - ACV
    - (CV))

Modern (Hybrid) Ventilators

BiPAP V60 Trilogy

General principles: ventilatory modes
BiPAP®

**Inspiration**
- Machine leakage through valve
- Leakage via exhalation hole
- ‘blower’
- Sensor
- Masker

**Expiration**
- ‘blower’
- Masker
- Continuous flow!

Pressure ↔ Volume

**Volume preset**
- Volume
- Flow
- Patient effort

**Pressure preset**
- Airway pressure
- Tidal volume set by operator
- Inspiratory pressure set by operator
- No respiratory effort
- Small respiratory effort
- Larger respiratory effort
Pressure ↔ Volume

Pressure preset NIV ↔ Volume preset NIV

- fixed tidal volume
- variable inspiratory pressures

Cave: no leak compensation

Slow reaction on triggering

Pressure ↔ Volume

Pressure preset NIV ↔ Volume preset NIV

- fixed inspiratory pressure
- fixed tidal volume
- variable inspiratory pressures

Good leak compensation

Cave: no leak compensation
Pressure ↔ Volume

Pressure preset NIV
- fixed inspiratory pressure
- variable tidal volume depending
  - change in inspiratory time
  - change in resistance (f.i. airway obstruction)
  - change in compliance (f.i. supine vs upright position in obesity)

Volume preset NIV
- fixed tidal volume
- variable inspiratory pressures

Good leak compensation

Cave: no leak compensation

TARGET-VOLUME NIV
- range of inspiratory pressures
- preset target volume to ensure tidal volume
Modern “BPAP”: different modes

- **S**
  
  In and expiratory time variable, dependent on the patient.

- **S/T**

![Graph showing different modes of BPAP](image)

Modern “BiPAP”: different modes

- **APCV**

  In PS with remaining rapid breathing pattern the PS only lasts a very short time → low volume with almost no alveolar ventilation.

  ![Graph showing APCV mode](image)
Target Volume NIV

- AVAPS (Respironics®): Average Volume Assured Pressure Support
  ![AVAPS Diagram]
  If the volume preset is not reached, AVAPS technique will smoothly increase pressure.

- IVAPS (Resmed®): Intelligent Volume Assured Pressure Support
  ![IVAPS Logo]

General principles: masks
Interfaces for BPAP ventilators

a « hole »! = a

b = anti-asphyxia valve for safety - allows you to breathe room air if the flow device malfunctions (problem with power supply)

General principles: masks

Interfaces for BPAP ventilators

a « hole »!

in between the mask and the tubing

Non-vented masks

General principles: masks
the “hole”

Fixed resistance, which results in varying leak rates depending on the pressure, but expiratory flow rates can exceed the airflow rate of the leak port, especially at low expiratory pressure → cave rebreathing

Variable resistance, with greater leaks at lower pressure (“bigger” hole) in case of some CO2 into the tubing it is washed out at the start of inspiration before subjects had a chance to rebreathe

NO

YES

Interfaces for BPAP-ventilators

If no « hole » → a valve

Non-vented masks

General principles: masks
Interfaces

Nasal mask

Nasal pillows

Interfaces

Oronasal mask

Full-face mask
Interfaces

Mouthpiece

Helmet: no direct contact to the skin

- Able to improve gas exchange*; yet**:
  - Large dead space
    - Decibels: 100 Db!!!
    - Patient-ventilator asynchrony / futile inspiratory efforts
    - Worsened CO2 clearance (despite higher press supp)

- A recent report suggests the need for dedicated monitoring when using the helmet to reduce accidental failure of fresh gas supply

*Navalesi. Int Care Med 2007; Antonelli. Anesthesiology 2004
### Interfaces: advantages and disadvantages

<table>
<thead>
<tr>
<th>Interface</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal</td>
<td>Less risk for aspiration</td>
<td>Mouth leak</td>
</tr>
<tr>
<td></td>
<td>Easier secretion clearance</td>
<td>Higher resistance through nasal passages</td>
</tr>
<tr>
<td></td>
<td>Less claustrophobia</td>
<td>Less effective with nasal obstruction</td>
</tr>
<tr>
<td></td>
<td>Easier speech</td>
<td>Nasal irritation and rhinorrhea</td>
</tr>
<tr>
<td></td>
<td>Easy to fit and secure</td>
<td>Mouth dryness</td>
</tr>
<tr>
<td>Oral</td>
<td>Better mouth leak control</td>
<td>Increased aspiration risk</td>
</tr>
<tr>
<td></td>
<td>More effective in mouth breathers</td>
<td>Difficulty speaking, eating, clearing</td>
</tr>
<tr>
<td></td>
<td></td>
<td>secretions</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Asphyxiation with ventilator malfunction</td>
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<tr>
<td>Mouthpiece</td>
<td>Less interference with speech</td>
<td>Less effective for acute respiratory</td>
</tr>
<tr>
<td></td>
<td></td>
<td>failure</td>
</tr>
<tr>
<td></td>
<td>Little dead space</td>
<td>Requires nasal or oral interface when</td>
</tr>
<tr>
<td></td>
<td>May not require headgear</td>
<td>sleeping</td>
</tr>
<tr>
<td>Total face mask</td>
<td>More comfortable for some patients</td>
<td>Nasal leak</td>
</tr>
<tr>
<td></td>
<td>Easier to fit</td>
<td>Cannot deliver aerosolized medications</td>
</tr>
<tr>
<td></td>
<td>Less facial skin breakdown</td>
<td></td>
</tr>
</tbody>
</table>

Hess. Respir Care 2012

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### Non-invasive ventilation (NIV)

**Respiratory Acidosis**

I can’t catch my breath.

Retention of CO₂ by Lungs

NIV for acute respiratory failure
NIV in acute failure: indications

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<td>Low</td>
<td>No recommendation made</td>
</tr>
<tr>
<td>Immune-compromised</td>
<td>Low</td>
<td>No recommendation made</td>
</tr>
<tr>
<td>De novo respiratory failure</td>
<td>Low</td>
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* All in the setting of acute respiratory failure; ** certainty of effect estimates: ⭐⭐⭐⭐⭐ high, ⭐⭐⭐⭐ moderate, ⭐⭐⭐ low, ⭐⭐ very low.

Rochewerg. ERJ 2017; 1602426

NIV in acute failure ~ COPD exacerbation

Hypercapnic respiratory failure secondary to AECOPD:
- convincing RCT’s ~
  - less mortality
  - less need for intubation
  - less nosocomial pneumonia

Indications: AECOPD
NIV did not prove to be more effective than standard medical therapy in preventing the occurrence of ARF, and in improving mortality and length of hospitalisation. Furthermore, > 50% of the patients did not tolerate NIV.

Start early, but not too early

NIV in acute failure ~ COPD exacerbation

Where to start?

- Severity:
  - pH > 7.35
  - pH 7.35-7.25
  - pH < 7.25, alertness
  - pH < 7.20 and/or: Neurological status, Fatigue, ETI indication, MOF

- Location:
  - Ward
  - Ward
  - ICU
  - T-Triale

- Intervention:
  - Drugs+oxygen
  - NPPV
  - NPPV
  - ETI
  - Discharge
  - NPPV

The practice in AECOPD

Barbe. ERJ 1996
Keenan. Respir Care 2005

Ambrosino. Eur Respir J 2008
NIV in acute failure ~ COPD exacerbation

How to start?

Before start
- DNR status clear!!
- Chest radiography recommended
- Optimal medical therapy should be started, targeting O2 sat. 88-92%

Step 1: install the patient
- Put patient in a comfortable sitting position (> 30° angle) and talk to the patient
- If patient is very anxious, a short-acting sedative drug can help (lorazepam 0.5 mg IV)
- Oxygen (FiO2 25-60%)
- Humidifier probably increases tolerance
- Do not attach the mask immediately, let the patient get used to the ventilation

The practice in AECOPD

Mask choice

- Many clinicians will prefer to use an oronasal or full face mask for NIV initiation; these masks minimize mouth leak that is common in patients in distress and naive to NIV and have been shown to result in better quality of ventilation than nasal masks

Elliott. ERJ 2004

The practice in AECOPD
Step 2: increase P and monitor the patient

Application of mask on patient
Start 4 cmH2O EPAP and 12 cmH2O IPAP
- increase IPAP in steps of 2 cmH2O until 14-16 cmH2O (depending on tolerance/leakage)
- adjust FiO2 ~ SaO2 90-95%

after 30-60 min re-evaluate

- (> 10%) decrease in RR (resp rate)
- or (> 10%) decrease in PaCO2
  yes → NIV continuation at the present settings
  no

  increase IPAP if possible (cave: synchronisation ventilator/patient)
  and/or after discussion with doctor: use plateau valve and/or increase EPAP and/or change
  type of mask

after 30-60 min: re-evaluate
- (> 10%) decrease in RR
- or (> 10%) decrease in PaCO2
  yes → NIV continuation
  no → Consider STOP NIV

The practice in AECOPD

Intrinsic PEEP (PEEPi)

End expiratory increased Raw / expiration time = too short

→ auto-PEEP

COPD

(normal P = 0)
Intrinsic PEEP (PEEPi)

Inspiration

Air entering ~ △P=5

Labour:

+10 → 0
0 → -5
P mus = 15

Auto-PEEP correction with EPAP=PEEP (CPAP)

End expiratory

EPAP=PEEP (CPAP) = 8
instead of 0 (atmospheric)

COPD
Intrinsic PEEP (PEEPi)

Inspiration

air entering $\Delta P=5$

less labour: $+10 \rightarrow +3$

P mus = 7

EPAP decreases work of breathing

Appendini. AJRCCM 1994;1069
Step 2: increase P and monitor the patient

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(> 10%) decrease in RR
or (> 10%) decrease in PaCO2

no

Consider to STOP NIV

NIV continuation at the present settings

NIV continuation

Relative risk of failure at 4 hours compared to admission.

<table>
<thead>
<tr>
<th>Initial pH</th>
<th>pH at 4 Hrs</th>
<th>RR -8/min</th>
<th>RR -4/min</th>
<th>No change</th>
<th>RR +4/min</th>
<th>RR +8/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.35</td>
<td>0.27</td>
<td>0.38</td>
<td>0.53</td>
<td>0.74</td>
<td>1.03</td>
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<tr>
<td>7.30</td>
<td>0.51</td>
<td>0.72</td>
<td>1.00</td>
<td>1.40</td>
<td>1.95</td>
<td></td>
</tr>
<tr>
<td>7.25</td>
<td>1.05</td>
<td>1.46</td>
<td>2.04</td>
<td>2.85</td>
<td>3.97</td>
<td></td>
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<tr>
<td>7.35</td>
<td>0.13</td>
<td>0.19</td>
<td>0.26</td>
<td>0.36</td>
<td>0.51</td>
<td></td>
</tr>
<tr>
<td>7.25</td>
<td>0.25</td>
<td>0.35</td>
<td>0.49</td>
<td>0.68</td>
<td>0.96</td>
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Confalonieri. ERJ 2005: if persistance of pH 7.25
and respiratory rate > 35=NIV failure

Plant. Thorax 2001
How to monitor

**Essential**
- Regular clinical observation by experienced staff
- Continuous pulse oximetry
- Arterial blood gases after 1-4h NIV and after 1h of any change in ventilator settings or FiO2
- Respiratory rate and synchrony machine/patient
  - Inspiratory effort detected by ventilator and followed by inspiratory cycling
  - Expiratory effort followed by expiratory cycling

**Desirable**
- Electrocardiogram
- More detailed physiological information such as leak, expired $V_T$ etc...

The practice in AECOPD

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**Step 3: how to wean**

- First 24h maximal respiratory support
- Afterwards intermittently according to respiratory evolution (especially during the night)
- Switch to nasal mask

The practice in AECOPD
**NIV in cardiogenic edema**

**Convincing RCT’s ~**
- less mortality
- less need for intubation

**Insufficient evidence to recommend either bilevel NIV or CPAP**
(10 cm CPAP or EPAP 6-10 / IPAP 12-20)

**Justifiable reluctance in acute coronary syndrome or cardiogenic shock (no data)**

*Indications and practice: cardiogenic edema*

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**Impact on pre and afterload!**

*Monnet. Curr Opin Crit Care 2007*
NIV in acute failure: indications

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Rochewerg. ERJ 2017; 1602426

NIV in acute failure: indications

Decompensated OHS

- Central nervous system
  - Decreased central respiratory drive

- Respiratory
  - Restrictive chest physiology
  - Pulmonary hypertension
  - Hypoxemia/hypercapnia

- Airway
  - Potential difficult airway
  - Obstructive sleep apnoea

- Cardiovascular
  - Coronary artery disease
  - Congestive heart failure

- Others
  - Difficult vascular access
  - Difficult positioning

No RCT’s

OHS patients treated with NIV have better outcomes than patients with COPD

\[ p=0.002 \]

Carrillo. AJRCCM 2012

Indications: decompensated OHS
Decompensated OHS: practice ~ AECOPD

CAVE OSA:

if increase in IPAP increase does not result in expansion of the thorax or increase in estVE: increase EPAP up to 10 cm

PLUS

in sitting position!

no flexion of the neck!

NIV in acute failure: indications ?!

Acute on chronic respiratory failure in patients with chest wall deformity or neuromuscular diseases

NO RCT evidence, but because of its beneficial effects in the domiciliary setting, a trial of NIV should be considered.

Vianello. Intensive Care Med 2000 have shown that in NMDs the outcome of NIV is better than with invasive ventilation. If patients do require intubation, they should be extubated and weaned to NIV as soon as possible.

NIV should even be started for hypercapnic patients with CWD or NMD admitted acutely without waiting for acidosis to develop.

The combined approach, especially in NMDs, of optimal airway clearance techniques, including cough assistance device, add to the effectiveness avoiding intubation.

Indications: decompenated NM disorders
Cough assistance

Figuur 3

Airstacking

Cough assistance

Thoracic (-abdominal) thrust
Coughing

Air stacking +/- Manual compression (MIC-ass)

If PCF (MIC-ass) < 160 l/min + bronchial secretions

Mechanical cough assist (Cough Assist TM / mechanical in-exsufflator)
Generates strong expiratory flow (600 L/min) through an instant application of negative pressure (-40 → -60 cmH2O) after maximum insufflation of the lung with positive pressure (40 cmH2O)

Max. pos P: 60 cm H2O
Max. neg P: 60 cm H2O

4 to 5 cycles followed by a rest
Questions?