RESPIRATORY FAILURE

Dr Graeme McCauley
KGH
Definitions

• Failure to oxygenate - PaO2 < 60
• Failure to clear CO2 - PaCO2 > 50
• Acute vs Chronic
• Hypoxemic failure - type I
• Hypercapneic failure - type II
Causes of Resp Failure

Hypoventilation and V/Q mismatch are the major causes

Hypoventilation
- CNS depression
- neuromuscular

V/Q mismatch
- blood/pus/fluid in the alveolar spaces
Pathophysiology

Type I
- V/Q mismatch +/- shunt
- leading to increased AaDO2

Type II
- PaCO2 related to VCO2/ Va
- decrease Va due to reduction in Min Vent
  or increase in Vd ( dead space vent )
Pathophysiology II

Respiration occurs at Alveolar/cap units
O2 diffuses into the blood/bind with Hgb
Quantity of O2 on Hgb depends on PaO2
Oxygen dissociation curve
Normal lungs have alveoli under/over Vent
and under and over perfused
Normally therefore there are areas of V/Q
mismatch like shunt and dead space
ARDS

Acute Respiratory Distress Syndrome
- Ashbaugh 1967 Denang Lung
- diffuse pulmonary infiltrates
- severe pancreatitis/blood transfusions
  sepsis/blast injuries
- progression of Acute Lung Injury
  a form of diffuse alveolar injury
ARDS II

Pathology
- diffuse alveolar damage
- capillary membrane injury
- increased permeability/ fluid alveolar
- injury to vascular endothelium (sepsis)
  and or alveolar epithelium (aspiration)
- protein rich fluid into the alveoli
ARDS III

PMN cells predominate

Cytokines: TNF, LTD, MIF. IL-6 and others

Inhomogeneous process with relatively normal alveoli more compliant

Overdistention may lead to Barotrauma or vulotrauma (shearing with cycle of collapse at end inspiration/reexpansion with positive pressure)
ARDS IV

Marked increase in AaDO2
Pulmonary Hypertension always present

Treatment

- maintain FiO2 < 0.60 ( O2 toxicity )
- lower Vt 6 ml/kg
- PEEP
- Plateau airway pressure < 30 cm H2O
- nitric oxide to treat Pulm Hypertension
Hypercapnic Disorders: Definitions

Hypercapnia: \( \text{PaCO}_2 \geq 45 \text{ mm Hg} \)

Hypercapnic respiratory failure: hypercapnia plus acidosis

- Acute: no or minimal metabolic compensation
- Chronic: appropriate metabolic compensation
Causes of Hypercapnic Resp. Failure

Neural & Neuromuscular

- Brain
  - Drugs
- Motor neurons
- Neuromuscular junction
- Respiratory muscles

Chest Wall

- Kyphoscoliosis
- Ankylosing spondylitis
- Flail chest

“Medical” Diseases

- COPD
- Severe asthma
- Late stage interstitial lung disease
- Pulmonary edema
- Sleep apnea / obesity-hypoventilation
- Hypothyroidism

Environmental – Industrial, Natural
Pulmonary Function Analysis

Diagnosis: **EMPHYSEMA**

| Age: 70 | Height(cm): 169 | Weight(kg): 50.0 |

**Spirometry**

<table>
<thead>
<tr>
<th>Ref</th>
<th>Pre Mess</th>
<th>% Ref</th>
<th>Post Mess</th>
<th>% Ref</th>
<th>Post Mess</th>
<th>% Chg</th>
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<tr>
<td>PVC</td>
<td>2.68</td>
<td>2.35</td>
<td>88</td>
<td>2.48</td>
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<td>FEV1</td>
<td>2.13</td>
<td>0.77</td>
<td>36</td>
<td>0.80</td>
<td>37</td>
<td>3</td>
</tr>
<tr>
<td>FEV1/FVC</td>
<td>62</td>
<td>33</td>
<td>12</td>
<td>32</td>
<td>14</td>
<td>14</td>
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<tr>
<td>PEF25-75% L/sec</td>
<td>2.30</td>
<td>0.27</td>
<td>12</td>
<td>0.31</td>
<td>14</td>
<td>14</td>
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<tr>
<td>PEF</td>
<td>0.35</td>
<td>2.69</td>
<td>50</td>
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<td>25</td>
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<tr>
<td>PEF70% L/sec</td>
<td>2.99</td>
<td>0.30</td>
<td>10</td>
<td>0.34</td>
<td>11</td>
<td>13</td>
</tr>
</tbody>
</table>

**Lung Volumes**

| VC | 2.68 | 2.35 | 88 |
| IC | 1.21 | 1.21 |
| ERV | 0.97 |
| TLC | 4.84 | 0.74 | 119 |
| FRC PL | 2.76 | 0.83 | 164 |
| RV | 2.12 | 3.39 | 160 |
| RVTLC | 43 | 39 |

**Diffusion**

| VA | 3.05 |
| DLco | 6.2 | 41 |
| DLcoVA | 2.33 | 62 |

**Airway Resistance**

| Raw | 1.29 | 0.09 | 457 |
| Gw | 0.662 | 0.164 | 25 |
| Vg | |

Comments:

pH=7.42 / PaCO2=33 / PaO2=64 / HCO3=21 / O2 Sat=83% / 100% .21

**FAXED**
AECOPD + Respiratory Failure

- COPD global mortality 6th 1990 → 3rd 2020
- Without NIMV mortality 1st Quartile ≈ 20%
  ≈ 48% without yr.
- Mortality & age (≥ 70)
  
  FEV₁ < 700 ml
  
  FEV₁/FVC (< 30%)
  
  Comorbidity (Cardiac)

- NIV recent & NIMV 75% alive @ 1 yr.
  48% " 3 yr.

- Short term prognosis reasonable
- Long term prognosis guarded

Pathophysiology

- Infection
  
  - Infection (AEC)
  
  - AHRHYTHMIA: (Ischemic cardiac event)
AVF in COPD due to ↑ leading to Resp. System → Resp. Muscle Dysfunction

AECA: ↑ Secretion, Plugging → Bronchioles, ↑ Inflammation, ↑ RAW.

↑ Exp. flow ± present return to FRC to Result in DHI (Dynamic Hyperinflation)

↑ use expiratory muscles → ↑ airway collapse (due to loss elasticity).

↑ Vt (resp flow) → ↑ Ti, ↑ TE butt flux + WOB.

DHI leads to AUTO-PEEP (auto-positive end-expiratory pressure)

i) due to premature airway collapse & pressure remains in distal alveoli

ii) this ↑ pressure must be overcome in order to initiate the next breath

iii) this further ↑ WOB → muscle fatigues & compromised muscle position (flattened diaphragm)
- COPD & exacerbation leads to ↑ dead space ventilation (Ventilated non-perfused lung) in bills.

- ↑ CO2 ↑ VD/Vt need ↑ Ve (mini ventilator). This further ↑ WOB and ↑ production CO2.

- Muscles fatigue due to WOB and often home ↓ O2 delivery - associated LV dysfunction R & CO. due to DHI + i seps. (↑ ↑ intra-thoracic & pleural with reduced venous return)
SEVERE AECOPD

↑ Airway Obstruction

↑ Airway Resistance

↑ Dynamic Hyperinflation

↑ Vd (Wasted Ventilation)

↑ WOB

Uneven Distribution Ventilation

↑ V/Q mismatch

Muscle Fatigue

↑ Pale & Pulse

Acidosis
  - Respiratory
  - Metabolic
[Diagram showing pulmonary and systemic circulations with annotations such as "Paw", "LAW", "PEEP", "VT", "RVSP", "RAP", "V.R.", "LV", "AVJS", "Shunt", and "DHI"].
DHI

↓ ExtTun → DHI

↓ VA (ventr. vent)

↓ Pco

↓ Vd/VT

⇒ ↓ Alveolar Pr (P↑E↑)

⇒ ↓ Hypotension

⇒ ↓ Def. synchrony

↓ ↓ VR → PArx

↓ ↓ VR → Pr
Dynamic Hyperinflation (DHI)

i) MEP cycle (spontaneous) before preceding Exp flow completed

ii) PEEPs (auto-PEEP) as consequence of DHI

[Graphs showing flow patterns]
TREATMENT

- Steroids, Bronchodilators
- ABx
- NIPPV (NIV), BiPAP (BiLEVEL)

NIPPV
- ↓ Weber
- ↓ V/G clearance
- COPD
  (↑ Po2, CHF)

Flattened Diaphragm

↑ Antimapping

↑ Raw

↑ PEEP

Muscle weakness

↑ CO2

↑ CBF

Resp. muscle failure

↓ VT

↓ Po2

Breath sounds

Pleural effusion

Surgical

Anesthesia
MODALITIES OF VENTILATORY SUPPORT

- NPPV → non-invasive positive pressure ventilation
  → Bi-level or BiPAP support.
  → particularly useful in Acute Exacerbation
    of COPD.
  → also useful in Acute Pulmonary Edema (Cardiogenic
    Non-Cardiogenic).

- CMV → controlled mandatory ventilation
  → Fully controlled & Spontaneous Breaths.

- ACV → Assist/Control Ventilation
  → Machine delivers full VT with each breath.

- SIMV/IMV → spontaneous intermittent mechanical
  ventilation (Synchronized)
  → may add pressure support &
  → improve VT & Spontaneous Reflexes.

- CPAP → continuous positive airway pressure

- PCV/VCV → pressure controlled ventilation
  → Volume controlled ventilation
Ventilation Principles (M.V.)
- consider NIPPV (18°)

→ correct ABG + AB & O₂.
→ provide support while other reasons
→ rest muscle respiration.

No need to optimize patient - ventilator synchrony
to allow for:
1) muscles to rest
2) reduce AEI.

Modes:
1) Volume cycled
2) Pressure cycled

AC:
→ assist control both machine delivered & pt
→ trigger (assisted breaths)

VCV (AC) → present VT (V̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̇̆
- Neumuscle relax, nore hypo to AC vs. SIMV (6Won) (ENG studies)
- VCV vs. VCV → less Barotrauma
- PGV may also ↑ D.H.I. wi COPD as pt controls Vt / hgr

Parameters
- Return PaO2 to pt baseline (HCO3-)
  - VT: 8-10 mL/kg
  - Rate: 15/min to allow adequate TE
  - Treat E & T & add D.H.I. (equation 9)
  - Add PEEP to overcome PEEPi
  - 1: drain need to increase breath
  - 2: 20% sug. on endotracheal tube
  - 3: Keep PEEPi < 0.75

Consequence
- Hemodynamic collapse due to HTP (4 PEEPi)
- 4 Volume return
- 4 RV overload (: CO + preload)
- Barotrauma
Pulmonary Edema

- LVSWR, VO₂ consumption, impaired muscle content
- In a lower volume tone
- In brief:
  - NIPPV (140/60mmHg) supports CPP above
  - CPP
  - Add less slowly (doesn’t reverse as quickly)

- Bridging to prevent re-intubation leads to often due to upper airway obstruction, muscle fatigue, and Pulmonary Edema
- These often are helped by NIPPV
- Most NIPPV rather than CPP to improve mechanics + reduction

Techniques
- Pressure MAP target > Volume (L/min)
- Pressure reduce barotrauma, match patient flow requirements.
- Pressure cycle IAP (CPP + ΔP max) to CPP(CPP)
- Like pressure support added to CPAP, ventilated FIO₂
NONINVASIVE PRESSURE VENTILATION (NIPPV)
(in the Acute Care Setting)

- ET intubation + M.V. - Standard of Care in Life Threatening Respiratory Failure.
- NIPPV vs compare to ET intubation +/- M.V.
- NIPPV avoids complications ET intubation at the cost of losing control of airway

(2pts+)

<table>
<thead>
<tr>
<th>recomm</th>
<th>mask</th>
<th>N/DETT/PN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumothorax</td>
<td>0%</td>
<td>8%</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>15%</td>
<td>14%</td>
</tr>
<tr>
<td>Hypostasis</td>
<td>0%</td>
<td>9.5%</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>1%</td>
<td>12%</td>
</tr>
<tr>
<td>Sepsis</td>
<td>0%</td>
<td>12%</td>
</tr>
</tbody>
</table>

* V.A.P. much less common but shorter duration NIPPV confirm this start

* several studies using NIPPV as "bridge" - no mortality in NIPPV failure -> ET intubation compared to ET intubation + NIPPV (no delay)
CRITERIA

- pt. Selection
  i) Sx + Signs Acute Resp Distress
  ii) Pacv 745 Pac2/Fio2 < 800

- early intervention
  - experienced + dedicated RT/nurse
  - Physician involvement
  - Sedo/mnt ps.
  - patient comfort/ cooperation

CONTRAINDICATIONS

- uncooperative, intubation, agitated pt.
- inability to protect airway
  - hemodynamic instability
  - acute adrenal process
  - facial issues - burns, surgery etc.
**Criteria - Inclusion:**

- Mild-severe Resp distress
- Tachypnea > 24 BPM
- Accessory/paradoxic muscle use
- PaCO₂ > 45, PaO₂/FIO₂ < 200mm Hg.

**Exclusion:**

- Arrest Resp
- Unable to protect Airway
- Secretions ++
- Agitated / Uncooperative (maybe 20 + Pulse)
- Hemodynamically unstable
- Facial #

**Masks**

<table>
<thead>
<tr>
<th></th>
<th>Nasal</th>
<th>Full Face</th>
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<tbody>
<tr>
<td>VA</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Cough</td>
<td>+++</td>
<td>+++</td>
</tr>
<tr>
<td>Speak</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Audble</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>VD</td>
<td>+</td>
<td>+++</td>
</tr>
</tbody>
</table>
- E.I.T. → 4 sedation needs
  → Often delayed which leads to fatigue
  and longer period resp. muscle recovery.
- NIPPV → may be removed frequently to assess pro.

**Physiology**

- When waveform/purpose appropriate, NIPPV
  assume much of the work (VE, CO)
- Lift cardiac loading,↑ adrenergic discharge
- Improved 02 delivery → improves acid-base
  (NO) → pH
- IAP/EAP components → ↑ mean AB
- Recruitment collapsed collapsed lung units
- Improved distribution lung water
  → May increase return ↑ cardiac output +
  ↓CRT & dead space + shunt
- Some hemodynamic disturbances less common
  with patient triggered ventilation than
  pressure inflation
  → V/Q matching better for spont. vs. mechanical
  breathing.
EXAMPLES

- COPD (Exacerbation)
  - Most common indication
  - all studies showed rates of ETT vs NPPV (80-90%)
  - significant improvements in ABGs, 2 L/min
  - Hyperdynamic hyperinflation → iPEEP (blood)
  - V/Q mismatches
  - New Breathing pattern - spontaneous breath support (NPPV) were able to accommodate these patients.
  - iPEEP (EPAP) overcomes iPEEP
  - Common Pneumonia (iatrogenic injury)
  - Failure to improve rapidly (Code)
  - Wound leaking
  - Decline status.

- Severe Asthma
  - pt. who previously had experienced OETT found NPPV more comfortable
  - Length support, keep stay, shorten ETT
  - alert & treated early.
Neuromuscular Causes of Hypercapnic Respiratory Failure

• Skeletal Muscle Diseases
  – Some (but not all) of the Muscular Dystrophies
    • Duchenne muscular dystrophy
    • Merosin-negative congenital muscular dystrophy
    • Myotubular myopathy 1
    • Autosomal dominant distal myopathy
    • One of the autosomal recessive limb-girdle muscular dystrophies
Neuromuscular Causes of Hypercapnic Respiratory Failure

- **Neuromuscular Junction Disorders**
  - Myasthenia gravis
  - Lambert Eaton myasthenic syndrome
  - Botulism
  - Organophosphate poisoning

- **Motor Neuron Disorders**
  - Amyotrophic lateral sclerosis
  - Guillain-Barre syndrome
  - Poliomyelitis
Iatrogenic Hypercapnia and Mechanical Ventilation

- Study of low vs conventional tidal volume / pressure mechanical ventilation for ARDS
- Much higher incidence of hypercapnia (pCO2 > 50 mm Hg) in low tidal volume / low pressure group

**Table 5. Hypercapnia in the Treatment Groups.**

<table>
<thead>
<tr>
<th>VARIABLE*</th>
<th>LIMITED-VENTILATION GROUP (N = 60)</th>
<th>CONTROL GROUP (N = 60)</th>
<th>P VALUE</th>
</tr>
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<tbody>
<tr>
<td>Maximal PaCO₂ — mm Hg</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Mean ±SD</td>
<td>54.4±18.8</td>
<td>45.7±9.8</td>
<td>0.002</td>
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<tr>
<td>Range</td>
<td>28–116</td>
<td>29–72</td>
<td></td>
</tr>
<tr>
<td>Arterial pH</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>7.29</td>
<td>7.34</td>
<td>0.036</td>
</tr>
<tr>
<td>Range</td>
<td>6.99–7.49</td>
<td>7.08–7.51</td>
<td></td>
</tr>
<tr>
<td>Hypercapnia — no. of patients (%)</td>
<td>31 (52)</td>
<td>17 (28)</td>
<td>0.009</td>
</tr>
<tr>
<td>Duration of hypercapnia — hr</td>
<td>146±265</td>
<td>25±22</td>
<td>0.017</td>
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Pickwickian Syndrome

Did Mr. Pickwick have:
1. Sleep apnea with hypersomnolence
2. Obesity-hypoventilation syndrome
3. Both
4. Neither
Pickwickian Syndrome

Little boy who would always fall asleep
Obesity Hypoventilation Syndrome (OHS)

• Definition
  – BMI > 30 kg/m²
  – Awake arterial pCO₂ > 45 mm Hg
  – No other causes for hypercapnia
OHS in Hospitalized Patients

- Studied 4332 admissions to medical services
- 277 (6%) were severely obese (BMI > 35 kg/m²)
- OHS present in 31% with severe obesity
  - Mean pCO₂ of 52 ± 7 vs 37 ± 6 mm Hg in subjects with simple obesity
- When BMI > 50 kg/m², prevalence OHS was 48%

Nowbar et al., Am J Med 2004
Outcome Following Discharge

- Survival curves for patients with obesity-associated hypoventilation or simple obesity after discharge from hospital.
- Adjusted for age, sex, body mass index, electrolyte abnormalities, renal insufficiency, history of thromboembolism, and history of hypothyroidism.

Nowbar et al., Am J Med 2004
Prevalance OHS Among OSA

• Obesity hypoventilation syndrome (OHS) among subjects with obstructive sleep apnea (OSA)
  – Prevalence of 20-30%

• Predictors of OHS:
  – Serum bicarbonate level (P < 0.001)
  – Apnea hypopnea index (P = 0.006)
  – Lowest oxygen saturation during sleep (P < 0.001)

• Threshold bicarbonate level of 27 mEq/l:
  – Sensitivity 92%
  – Specificity 50%

Mokhlesi et al., Sleep Breath, 2007
Management of Hypercapnia

• Is it acute or chronic or acute on chronic?
• What is the underlying etiology?

• Treatment options
  – Specific therapy for underlying cause
  – No mechanical ventilation
  – Non-invasive mechanical ventilation
  – Invasive mechanical ventilation
Hypercapnic Respiratory Failure

Early Treatment Modalities

Figure 5. Photograph of a young girl with polio, showing Ibsen’s life-saving system: a tube supplying oxygen, a cuffed tracheotomy tube, and a medical student squeezing the rebreathing bag.

Polio Epidemic
Denmark, 1952

Polio -- Iron Lung Ward – 1950s

Rancho Los Amigos Hospital
Mechanical Ventilation for Acute Hypercapnic Respiratory Failure

- Intubation with conventional mechanical ventilation
- Non-invasive positive pressure ventilation (NPPV)
Criteria for Non-Invasive Ventilation in COPD

Selection criteria (at least two should be present)

• Moderate to severe dyspnea with use of accessory muscles and paradoxical abdominal motion
• Moderate to severe acidosis (pH 7.30-7.35) and hypercapnia (PaCO$_2$ 45-60 mm Hg)
• Respiratory frequency > 25 breaths/min

Criteria for Non-Invasive Ventilation in COPD

Exclusion criteria (any may be present)

- Respiratory arrest
- Cardiovascular instability (hypotension, arrhythmias, MI)
- Somnolence, impaired mental status, uncooperative patient
- High aspiration risk
- Viscous or copious secretions
- Recent facial or gastroesophageal surgery
- Craniofacial trauma
- Fixed nasopharyngeal abnormalities
- Extreme obesity

Masks for Non-Invasive Ventilation

• Types of mask
  – Nasal
    • More comfortable
    • Patient can eat
    • Minimal aspiration risk
    • Communication easier
  – Whole face
    • No entrainment of room air
    • May allow better ventilation

• Choice of mask
  – Hypercapnic respiratory failure
    • Nasal mask often sufficient
    • Sometimes need whole face mask
  – Hypoxic respiratory failure
    • Always need whole face mask
Ventilator Devices for Non-Invasive Ventilation

• Types of Ventilator Devices
  – BiPAP
    • Simple BiPAP – oxygen set by liter flow
    • Advanced BiPAP – can set FiO2
  – Conventional Ventilator

• Choice of Ventilator Device
  – Hypercapnic respiratory failure
    • Simple BiPAP is sufficient
    • Any of above may be used
  – Hypoxic respiratory failure
    • Need advanced BiPAP or conventional ventilator
Inspiratory and Expiratory Pressures

• Hypercapnic respiratory failure
  – Inspiratory pressure typically in 12 to 20 cm H₂O range
    • Lower values better tolerated
    • Higher values give better ventilation
  – Expiratory pressure not really needed
    • Except: many BiPAP machines require several cm H₂O to function properly

• Hypoxic respiratory failure
  – Inspiratory pressure typically in 12 to 20 cm H₂O range
  – Expiratory pressure gradually increased to
COPD – Non-Invasive Ventilation

• Total of 85 patients with COPD exacerbation from five hospitals in France, Italy and Spain

• Non-invasive ventilation
  – Face mask with foam inside to reduce dead space
  – Pressure support ventilator system with back-up rate
  – Inspiratory pressure 20 cm H$_2$O, no expiratory pressure
  – Oxygen to achieve saturation $> 90$

Brochard et al., NEJM 333:817, 1995
COPD – Non-Invasive Ventilation

Non-invasive ventilation significantly improved PaCO$_2$ and PaO$_2$.

Brochard et al., NEJM 333:817, 1995
COPD – Non-Invasive Ventilation

Table 1. Characteristics of Patients with Acute Exacerbations of Chronic Obstructive Pulmonary Disease Assigned to Standard Treatment or Noninvasive Ventilation, at Admission and after One Hour of Therapy.*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Standard Treatment</th>
<th>Noninvasive Ventilation</th>
<th>P Value†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Admission (n=42)</td>
<td>1 hour (n=39)</td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td>69±10</td>
<td>71±9</td>
<td>0.28</td>
</tr>
<tr>
<td>SAPS</td>
<td>13±5</td>
<td>12±4</td>
<td>0.54</td>
</tr>
<tr>
<td>Systolic pressure (mm Hg)</td>
<td>145±25</td>
<td>143±25</td>
<td>0.82</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>107±15</td>
<td>105±23</td>
<td>0.61</td>
</tr>
<tr>
<td>Encephalopathy score</td>
<td>1.6±1.2</td>
<td>1.9±1.3</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.8±1.3</td>
<td>1.5±1.1</td>
</tr>
<tr>
<td>Respiratory rate (breaths/min)</td>
<td>33±7</td>
<td>33±7</td>
<td>0.83</td>
</tr>
<tr>
<td></td>
<td></td>
<td>35±7</td>
<td>25±8</td>
</tr>
<tr>
<td>PaO₂ (mm Hg)</td>
<td>39±12</td>
<td>58±24</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td>41±10</td>
<td>66±17</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>67±16</td>
<td>72±18</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td></td>
<td>70±12</td>
<td>68±13</td>
</tr>
<tr>
<td>pH</td>
<td>7.28±0.11</td>
<td>7.26±0.11</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td></td>
<td>7.27±0.10</td>
<td>7.31±0.09</td>
</tr>
<tr>
<td>Bicarbonate (mmol/liter)</td>
<td>32±7</td>
<td>33±7</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>33±7</td>
<td>33±7</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>13.8±2.2</td>
<td>14.5±2.0</td>
<td></td>
</tr>
</tbody>
</table>

*Three patients in the standard-treatment group and one in the noninvasive ventilation group were intubated at one hour, before new measurements were performed. SAPS denotes simplified acute physiologic score. PaO₂ partial pressure of arterial oxygen, and PaCO₂ partial pressure of arterial carbon dioxide. pH, bicarbonate, and hemoglobin were measured in samples of arterial blood.

†P values refer to the comparison between the two groups at the time of admission.

‡P values refer to the comparison between data on admission and data at one hour in each group.
COPD – Non-Invasive Ventilation

Outcomes

• Reduced need for intubation
  – Non-invasive group 26% intubated (11/43)
  – Conventional group 74% intubated (31/42) (P < 0.001)

• Reduced complication rate
  – Non-invasive group 16% (7/43)
  – Conventional group 48% (20/42) (P = 0.001)

• Improved survival to hospital discharge
  – Non-invasive group 91% (39/43)
  – Conventional group 71% (30/42) (P = 0.02)

Brochard et al., NEJM 333:817, 1995
COPD – Non-Invasive Ventilation

Outcomes (cont’d)

• Reduced length of stay in hospital
  – Non-invasive group 23 ± 17 days
  – Conventional group 35 ± 33 days (P = 0.02)

• Lower proportion with length of stay > 4 weeks
  – Non-invasive group 18% (7/43)
  – Conventional group 47% (14/42) (P = 0.004)

Brochard et al., NEJM 333:817, 1995
Meta-Analysis: COPD and Non-Invasive Ventilation

*British Medical Journal*

Non-invasive positive pressure ventilation to treat respiratory failure resulting from exacerbations of chronic obstructive pulmonary disease: Cochrane systematic review and meta-analysis

Josephine V Lightowler, Jadwiga A Wedzicha, Mark W Elliott, Felix S F Ram

2003

Lightowler et al., BMJ 326:185, 2003
## Meta-Analysis: COPD and Non-Invasive Ventilation

Risk of treatment failure (mortality, need for intubation, intolerance)

Relative risk 0.51

### Table of Studies and Results

<table>
<thead>
<tr>
<th>Study</th>
<th>NPPV</th>
<th>Usual medical care</th>
<th>Risk ratio (fixed 95% CI)</th>
<th>Weight (%)</th>
<th>Risk ratio (fixed 95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avdeev et al 1998&lt;sup&gt;19&lt;/sup&gt;</td>
<td>7/29</td>
<td>12/29</td>
<td></td>
<td>11.2</td>
<td>0.58 (0.27 to 1.27)</td>
</tr>
<tr>
<td>Barbe et al 1996&lt;sup&gt;16&lt;/sup&gt;</td>
<td>4/14</td>
<td>0/10</td>
<td></td>
<td>0.5</td>
<td>6.60 (0.39 to 110.32)</td>
</tr>
<tr>
<td>Bott et al 1993&lt;sup&gt;2&lt;/sup&gt;</td>
<td>5/30</td>
<td>13/30</td>
<td></td>
<td>12.1</td>
<td>0.38 (0.16 to 0.94)</td>
</tr>
<tr>
<td>Brochard et al 1995&lt;sup&gt;3&lt;/sup&gt;</td>
<td>12/43</td>
<td>33/42</td>
<td></td>
<td>31.1</td>
<td>0.36 (0.21 to 0.59)</td>
</tr>
<tr>
<td>Celikel et al 1998&lt;sup&gt;14&lt;/sup&gt;</td>
<td>1/15</td>
<td>6/15</td>
<td></td>
<td>5.6</td>
<td>0.17 (0.02 to 1.22)</td>
</tr>
<tr>
<td>Dikensoy et al 2002&lt;sup&gt;20&lt;/sup&gt;</td>
<td>4/19</td>
<td>7/17</td>
<td></td>
<td>6.9</td>
<td>0.51 (0.18 to 1.45)</td>
</tr>
<tr>
<td>Plant et al 2000&lt;sup&gt;15&lt;/sup&gt;</td>
<td>22/118</td>
<td>35/118</td>
<td></td>
<td>32.6</td>
<td>0.63 (0.39 to 1.00)</td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>55/268</td>
<td>106/261</td>
<td></td>
<td>100</td>
<td>0.51 (0.38 to 0.67)</td>
</tr>
</tbody>
</table>

Test for heterogeneity: $\chi^2=7.59$, df=6, P=0.27
Test for overall effect: $Z=-4.82$, P<0.0001

Lightowler et al., BMJ 326:185, 2003
### Meta-Analysis: COPD and Non-Invasive Ventilation

**Risk of mortality**  
**Relative risk 0.41**

<table>
<thead>
<tr>
<th>Study</th>
<th>NPPV</th>
<th>Usual medical care</th>
<th>Risk ratio (fixed 95% CI)</th>
<th>Weight (%)</th>
<th>Risk ratio (fixed 95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avdeev et al 1998</td>
<td>3/29</td>
<td>9/29</td>
<td></td>
<td>15.6</td>
<td>0.33 (0.10 to 1.11)</td>
</tr>
<tr>
<td>Barbe et al 1996</td>
<td>0/10</td>
<td>0/10</td>
<td></td>
<td>0.0</td>
<td>Not estimable</td>
</tr>
<tr>
<td>Bott et al 1993</td>
<td>3/30</td>
<td>9/30</td>
<td></td>
<td>15.6</td>
<td>0.33 (0.10 to 1.11)</td>
</tr>
<tr>
<td>Brochard et al 1995</td>
<td>4/43</td>
<td>12/42</td>
<td></td>
<td>21.1</td>
<td>0.33 (0.11 to 0.93)</td>
</tr>
<tr>
<td>Celikel et al 1998</td>
<td>0/15</td>
<td>1/15</td>
<td></td>
<td>2.6</td>
<td>0.33 (0.01 to 7.58)</td>
</tr>
<tr>
<td>Dikensoy et al 2002</td>
<td>1/17</td>
<td>2/17</td>
<td></td>
<td>3.5</td>
<td>0.50 (0.05 to 5.01)</td>
</tr>
<tr>
<td>Plant et al 2000</td>
<td>12/118</td>
<td>24/118</td>
<td></td>
<td>41.6</td>
<td>0.50 (0.26 to 0.95)</td>
</tr>
<tr>
<td><strong>Total (95% CI)</strong></td>
<td>23/262</td>
<td>57/261</td>
<td></td>
<td>100</td>
<td>0.41 (0.26 to 0.64)</td>
</tr>
</tbody>
</table>

Test for heterogeneity: $\chi^2=0.82$, df=5, P=0.98  
Test for overall effect: $Z=-3.96$, P=0.00008

Lightowler et al., BMJ 326:185, 2003
Meta-Analysis: COPD and Non-Invasive Ventilation

Risk of intubation

Relative risk 0.42

<table>
<thead>
<tr>
<th>Study</th>
<th>NPPV</th>
<th>Usual medical care</th>
<th>Risk ratio (fixed 95% CI)</th>
<th>Weight (%)</th>
<th>Risk ratio (fixed 95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Avdeev et al 1998</td>
<td>5/29</td>
<td>8/29</td>
<td></td>
<td>8.8</td>
<td>0.62 (0.23 to 1.68)</td>
</tr>
<tr>
<td>Barbe et al 1996</td>
<td>0/10</td>
<td>0/10</td>
<td></td>
<td>0.0</td>
<td>Not estimable</td>
</tr>
<tr>
<td>Bott et al 1993</td>
<td>0/30</td>
<td>2/30</td>
<td></td>
<td>2.8</td>
<td>0.20 (0.01 to 4.00)</td>
</tr>
<tr>
<td>Brochard et al 1995</td>
<td>11/43</td>
<td>31/42</td>
<td></td>
<td>34.7</td>
<td>0.35 (0.20 to 0.60)</td>
</tr>
<tr>
<td>Celikel et al 1998</td>
<td>1/15</td>
<td>2/15</td>
<td></td>
<td>2.2</td>
<td>0.50 (0.05 to 4.94)</td>
</tr>
<tr>
<td>Dikenson et al 2002</td>
<td>2/17</td>
<td>7/17</td>
<td></td>
<td>7.7</td>
<td>0.29 (0.07 to 1.18)</td>
</tr>
<tr>
<td>Kramer et al 1995</td>
<td>1/11</td>
<td>8/12</td>
<td></td>
<td>8.5</td>
<td>0.14 (0.02 to 0.92)</td>
</tr>
<tr>
<td>Plant et al 2000</td>
<td>18/118</td>
<td>32/118</td>
<td></td>
<td>35.4</td>
<td>0.56 (0.34 to 0.94)</td>
</tr>
<tr>
<td>Total (95% CI)</td>
<td>38/273</td>
<td>90/273</td>
<td></td>
<td>100</td>
<td>0.42 (0.31 to 0.59)</td>
</tr>
</tbody>
</table>

Test for heterogeneity: $\chi^2 = 4.18, df = 6, P = 0.65$
Test for overall effect: $Z = -5.13, P < 0.0001$

Lightowler et al., BMJ 326:185, 2003
Other significant outcome improvements with non-invasive ventilation in COPD

- Reduced rate of complications
- Reduced hospital length of stay
- Improved pH, pCO2 and respiratory rate within one hour of initiation
Ventilation for Chronic Hypercapnia

- Clear role for chest wall and neuromuscular disease, and congenital central hypoventilation syndrome
- Often used for obesity-hypoventilation with sleep apnea (ie use BiPAP rather than CPAP)
- Controversial for obstructive lung diseases

- For neuromuscular diseases, often able to start with nocturnal only, and then move to 24 hours/day with disease progress
2. Indications for usage
   i. Symptoms (such as fatigue, dyspnea, morning headache, etc) and one of the following
   ii. Physiologic criteria (one of the following)
      a. \(\text{Paco}_2 \geq 45 \text{ mm Hg}\)
      b. Nocturnal oximetry demonstrating oxygen saturation \(\leq 88\%\) for 5 consecutive minutes
      c. For progressive neuromuscular disease, maximal inspiratory pressures \(< 60 \text{ cm/H}_2\text{O}\) or FVC \(< 50\%\) predicted
**NPPV and Restrictive Disorders**

**Table 4.** Contraindications to noninvasive ventilation for neuromuscular disease.

<table>
<thead>
<tr>
<th>Absolute contraindications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper airway obstruction</td>
<td></td>
</tr>
<tr>
<td>Uncontrollable secretion retention</td>
<td></td>
</tr>
<tr>
<td>Inability to cooperate</td>
<td></td>
</tr>
<tr>
<td>Inability to achieve adequate peak cough flow, even with assistance</td>
<td></td>
</tr>
<tr>
<td>Inability to fit interface or other noninvasive device</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Relative contraindications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Swallowing impairment</td>
<td></td>
</tr>
<tr>
<td>Inadequate financial resources</td>
<td></td>
</tr>
<tr>
<td>Inadequate family/caregiver support</td>
<td></td>
</tr>
<tr>
<td>Need for full-time ventilatory assistance</td>
<td></td>
</tr>
</tbody>
</table>

Perrin et al., *Muscle Nerve* 2004
Blood Gas Changes

![Graph showing PaCO2 (mm Hg) levels for different conditions: COPD, OHS, TB, NM, POLIO, KYPH with associated p-values: P=.008, P<.001, P=.002, P=.0002, P=.0009, P=.02.](image)
Survival: 7 Year Follow Up
Examples of People Undergoing Long Term Mechanical Ventilation

Christopher Reeve 1995 to 2004

Stephen Hawking ~1985 to present

This life-sustaining technology enabled Hawking to continue his research, write books, and maintain his family relationships.
Long-Term Ventilation Not for Everyone

Tuesdays With Morrie Versus Stephen Hawking: Living or Dying With ALS

There were other people who suffered from ALS, Morrie knew, some of them famous, such as Stephen Hawking, the brilliant physicist. . . . He lived with a hole in his throat . . . This was admirable, but it was not the way Morrie wanted to live.” (p. 161)