Management of Sleep Related Hypoventilation Syndromes

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Overview

• Types/ groups of hypoventilation syndromes
  – RAD criterion

• Polysomnography in hypoventilation
  – Redesigning the lab space
  – Diagnostic studies
  – Titration studies

• Device options
  – Standard NIV Devices
    • Mode options
    • Manufacturer differences
  – Auto Titration Devices
    • The wrong devices
    • VAP devices
  – Mechanical ventilation with mask
  – Biphasic Cuirass Ventilation
RAD Criterion

- RAD = Respiratory Assist Device = Noninvasive ventilation (NIV)
- Standard protocols for OSA do not apply to the disorders that contained in the RAD guidelines
- The hypoventilation syndromes covered under the guidelines include the following:
  - Restrictive thoracic disorders
  - Severe COPD
  - Central Sleep Apnea
  - Hypoventilation Disorders(generic)

https://www.noridianmedicare.com/dme/train/presentations/respiratory_assist_device_q_a.html
RAD criterion: Central Sleep Apnea

- Symptoms of either excessive sleepiness disrupted sleep.
- AND
- In lab PSG must be performed documenting all of the following criterion:
  - an AHI >5 and
  - central AHI >50% of the Total AHI and
  - central AHI >5

https://www.noridianmedicare.com/dme/training/presentations/respiratory_assist_device_q_a.html
Central Sleep Apnea

- Most of this topic will be discussed by Dr. Naughton. However, please note the following diagnosis that are rare but still associated with central apnea.
  - They are different in that they present with hypoventilation rather than hyperventilation, and as such are a small part of any discussion of disorders of hypoventilation in sleep.

- Brain Stem Disorders
  - Radiation to the brain stem for lesions such as astrocytomas
  - MS/ Devics

- CNS lesions
  - Bilateral Strokes
  - Chiari Malformations
  - Cerebral Palsy

- Medications
  - Intrathecal Narcotics/ Baclofen
Central Sleep Apnea

**CSR- CSA**
- Apnea is prominent in NREM
- The disease is associated with hyperpnea
- The disease can be treated with servo ventilation

**Non CSR-CSA**
- Apnea is most prominent in REM
- The disease is associated with hypoventilation
- The disease should NOT be treated with servo ventilation

If you have identified CSA on a PSG, it is important to think about the cause:
  1) CSR- CSA
  2) Non CSR – CSA
  3) Alternative hypoventilation disorder: most commonly RTD
Severe COPD

RAD Criterion

• An arterial blood gas carbon dioxide reading (PaCO2), done while awake and breathing the patient's usual FIO2, is ≥52 mm Hg,

AND

• Sleep oximetry demonstrates oxygen saturation ≤88% for at least five continuous minutes, done while breathing the patient's usual FIO2

AND

• Prior to initiating therapy, obstructive sleep apnea (and treatment with continuous positive airway pressure) has been considered and ruled out.

What is COPD anyway?

• In this case PFT’s are not needed.

• Hypercapnea and Hypoxemia are all that is needed--- This may apply to many end stage parnechymal lung diseases

• The term “OBSTRUCTIVE” should be dropped and replaced with “CPD” chronic pulmonary disease

https://www.noridianmedicare.com/dme/train/presentations/respiratory_assist_device_q_a.html
COPD and Re-Admission Rates

- 3rd most common cause of hospital readmission among Medicare beneficiaries
- 22.6% of all Medicare beneficiaries
- 1 in 12 adults (40–64 yr old) hospitalized for COPD are readmitted to the hospital within 30 days of discharge.

↑risk of reexacerbations within the first few weeks of the index exacerbation

Roozbeh Sharif AnnalsATS Volume 11 Number 5| June 2014
In 2012, the Affordable Care Act established strong financial incentives for hospitals and physicians to reduce readmissions. The law required CMS to establish the Hospital Readmissions Reduction Program:

- HF, pneumonia, and AMI

In 2015, the program expanded to include:

- COPD elective hip/knee replacement

Aggregate payments for excess readmissions =

\[
\text{[sum of base operating DRG payments for AMI x (excess readmission ratio for AMI-1)] + [sum of base operating DRG payments for HF x (excess readmission ratio for HF-1)] + [sum of base operating DRG payments for PN x (excess readmission ratio for PN-1)] + [sum of base operating payments for COPD x (excess readmission ratio for COPD-1)] + [sum of base operating payments for THA/TKA x (excess readmission ratio for THA/TKA -1)]}
\]

### Clinical Spectrum of COPD patients seen in the hospital

<table>
<thead>
<tr>
<th>COPD</th>
<th>COPD/OSA Overlap</th>
<th>Severe COPD</th>
<th>COPD Exacerbation</th>
<th>COPD “Plus”</th>
</tr>
</thead>
</table>
| Clinical Presentation | • Baseline PaO₂ and PaCO₂ are normal  
• PSG demonstrates sleep apnea  
• Spirometry: **mild to moderate obstruction** | • Chronic need for supplemental O₂  
• Chronic PaCO₂ > 52 mmHg.  
• Would benefit from the use of NIV in both the ICU and Home | • At baseline may not have hypercapnia or hypoxemia  
• Is in the midst of an acute episode with high WOB/CO₂ and hypoxemia, with need for inpatient NIV. | • COPD "+" means that NIV may be more helpful for hospitalized patients with:  
✓ COPD + MI  
✓ COPD + HF  
✓ COPD + CAP  
✓ …. |
| Clinical Risk(s) | • Pulmonary Hypertension | • Readmission  
• Mortality | • Intubation risk | • Worsening of the underlying disease (e.g. HF) |
| Device | • CPAP therapy | • Bi-Level (ST/S) or VAPS  
• O₂ bleed in | • Bi-Level (ST/S) or VAPS  
• O₂ bleed in | • Bi-Level (ST/S) |
COPD - OSA Overlap
The story of hypoxemia

Stats

- Large variation in reports
  - In General population
    • 0.5% - 4%
  - In OSA
    • 9%-56%
  - In COPD
    • 5% - 85%
- Cor Pulmonale
  - OSA – 20%
  - Overlap – up to 80%
  - 5 Year Survival Rate – 30%

CPAP Therapy

- CPAP + STD Care: 71%
- STD Care alone: 26%

Khati, SB. Cleveland Clinic Journal of Medicine. 2016 February;83(2):127-140

Severe COPD

What if you have a patient with classic severe COPD?

- There are currently three schools of thought:
  - NIV is *helpful* and the more the better “High-Intensity” PS is needed
  - NIV can be a *detrimental*. The so called “Deventilation Syndrome” may occur in which NIV causes AM dyspnea.
  - NIV has *no role*. Given the mixed data in chronic COPD, NIV should not be used.

- Why could it work? Is it all about CO2?
  - Resting muscles
  - Restoring central drive with improved chemosensativity
  - Reduced daytime by adjusting load
Severe COPD
The story of CO2

- When considering patients with severe COPD (FEV1 <33% and PaO2 <50)
  - At all points studied, increasing age amplifies the risk of mortality for those with COPD
  - Hypercapnea is an independent risk factor for mortality risk
  - Hypoxemia and correction of that hypoxemia does not seem to have a significant independent impact on mortality

Life expectancy NL vs COPD Pt on LTOT

2 factors drive the difference: Age and PaCO2 >43

Pascal Foucher CHEST 1998; 113:1580-87
Severe COPD: No Role

Why is there no benefit?

- No physiologic goal.
  - How do you determine best titration?
- PS was to low.
  - On average IPAP was 10-18
- The lesson of long term studies
  - Fleeting results
  - With high mortality on both sides – do long term survivors represent a different group

What about the US?

- Long Term Oxygen Therapy?
  - Easy but not cheap
- How much pressure support is enough?
  - Hyperinflation vs CO2 reduction
Severe COPD: No Role – Early studies

Initiation process for non-invasive ventilation (NIV) within recognised randomised controlled trials.

<table>
<thead>
<tr>
<th>Study</th>
<th>Characteristics of trial</th>
<th>Starting settings</th>
<th>Recommended alterations</th>
<th>Oxygen utilisation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kramer et al.</td>
<td>NIV used in ITU or ‘step-down unit’</td>
<td>IPAP 8 cm H₂O, EPAP 2 cm H₂O</td>
<td>Increase IPAP by 1 cm H₂O every 15 to 30 minutes ‘as tolerated’</td>
<td>Entrain oxygen for SaO₂ of 90% or greater</td>
</tr>
<tr>
<td>Wood et al.</td>
<td>NIV on admission</td>
<td>IPAP 8 cm H₂O, EPAP 2 cm H₂O to 4 cm H₂O</td>
<td>Adjust IPAP for tidal volume of 8 to 10 ml/kg. Adjust EPAP with oxygen for SaO₂ &gt; 92%</td>
<td>Entrain oxygen for SaO₂ of 92% or greater</td>
</tr>
<tr>
<td>Plant et al.</td>
<td>NIV applied on respiratory wards</td>
<td>IPAP 10 cm H₂O, EPAP 4 cm H₂O</td>
<td>Increase IPAP by 5 cm H₂O until discomfort or 20 cm H₂O reached</td>
<td>Entrain oxygen for SaO₂ of 85 to 90%</td>
</tr>
<tr>
<td>2000 [17]</td>
<td>COPD only</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thys et al.</td>
<td>NIV on admission</td>
<td>IPAP 10 cm H₂O, EPAP 4 cm H₂O</td>
<td>Increase IPAP by 2 cm H₂O until discomfort or mask leak or 20 cm H₂O reached</td>
<td>Add via nasal cannulae for SaO₂ of 90%</td>
</tr>
<tr>
<td></td>
<td>Placebo NIV used by control group</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

IPAP, inspiratory positive airway pressure; EPAP, expiratory positive airway pressure; ITU, intensive therapy unit; COPD, chronic obstructive pulmonary disease; SaO₂, oxygen saturation.

K. Ward, H. Horobin / Physiotherapy 98 (2012) 151–159

**Standard NIV use in COPD**

- Most appropriate in the setting of hypercapnea and hypoxemia
- Those with highest baseline levels of dyspnea are best to consider
- There are many studies but the data is flawed due to
  - Inconsistent inclusion criterion
  - Varying lengths of observation
  - Differing primary end points

**Protocols**

- Goals of care vary:
  - Tolerance
  - VT
  - O₂ sat
- Amount of PS used is very low
With these 5 well done RCT’s
1) CO2 did not change
2) Survival did not change
3) All required long hospital stays to start therapy
4) Re-admission rates were not impacted.
Severe COPD: No Role – Late Studies

Cochrane on studies 1991 - 2009

- 7 total studies
  - No changes in CO2
  - No changes in 6MWD
  - No changes in HRQoL
  - No changes in Spiro/MIP
  - No changes in Sleep
  - No changes in Dyspnea

- IPAPS of the 7 studies
  - 12, 14, 10, 13, 18, 16, 15

- PaCO2 of the 7 studies
  - 51, 56, 52, 54, 56, 43, 46

Severe COPD - NIV

Largest Trial of NIV

- Baseline CO2 >46
  - Average CO2 = 53
- Average PAP = 13/5
- Morning CO2
  - LTOT – raised ~19 points
  - NIV +LTOT – raised ~13 points

N=72 in each arm

R D McEvoy; Thorax 2009;64:561–566
Severe COPD: No Role - Summary

Noninvasive Positive Pressure Ventilation for Chronic Respiratory Failure Patients With Stable Chronic Obstructive Pulmonary Disease (COPD): An Evidence-Based Analysis

COPD Working Group
Severe COPD: High-Intensity

Why would traditional NIV fail?

• More aggressive Inspiratory Pressures is needed (~28).
• Better address elevated CO2.
• Poor compliance.
• Detrimental hyperinflation.

If some did not work why not try a lot?
Severe COPD: High Intensity vs Low Intensity

Changes after 1 hour
• Both increase VT – LI 560 vs HI 670
• WOB reduced to Zero with HI vs LI
• PaCO₂ – LI 55.2 vs HI 49.4
• BORG scores were equal
• SBP dropped from baseline 126 to 112 with HI
• CO dropped from 5.5 to 4.0 with HI
• O₂ delivery dropped from 577 to 409 with HI

Changes after 6 weeks
• Increased Vt in HI (mean 96ml)
• Mean reduction in PaCO₂ in HI (-9.2)
• Improvements in dyspnea, FEV1, VC, QoL
• More drop outs with LI

References:
3) Michael Dreher Thorax 2010;65:303-308

Polysomnography of long term NIV users with COPD
• More drop outs with LI
• No change in SWS
• PaCO₂ was lower (-6.4) in HI
Severe COPD: High Intensity vs no NIV

Mortality

- PRCT – with 12 mo. f/u
  - 6 years to recruit 201 patients from 36 centres
- PCO2 >52
- GOLD stage IV
  - FEV1/FVC <70% and FEV1 <30%
- NIV-
  - set to reduce PaCO2
    - ~20%
    - <48
  - admitted patients electively for a mean of 5.6 days
- QoL
  - Improved SF-36, St. George

Severe COPD: NIV and mortality

**Summary of key data from long-term studies of domiciliary NIV in COPD**

<table>
<thead>
<tr>
<th>Study</th>
<th>Number of patients</th>
<th>Mean age (years)</th>
<th>FEV₁ litre (% predicted)</th>
<th>BMI (kg/m²)</th>
<th>PaCO₂ (kPa)</th>
<th>Settings and compliance, for NIV</th>
<th>Deaths (12 months)</th>
<th>NIV</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Köhleim et al, 2014</td>
<td>102</td>
<td>62-2</td>
<td>64-4</td>
<td>25%</td>
<td>24-8</td>
<td>7-8</td>
<td>11-8%</td>
<td>33%</td>
<td></td>
</tr>
<tr>
<td>Struik et al, 2014</td>
<td>101</td>
<td>63-9</td>
<td>63-5</td>
<td>0-67 (26%)</td>
<td>7-9</td>
<td>7-7</td>
<td>30%</td>
<td>29%</td>
<td></td>
</tr>
<tr>
<td>McEvoy et al, 2009</td>
<td>72</td>
<td>68-8</td>
<td>67-2</td>
<td>0-55 (23%)</td>
<td>25-4</td>
<td>7-25</td>
<td>17%*</td>
<td>22%*</td>
<td></td>
</tr>
<tr>
<td>Windisch et al, 2005</td>
<td>34</td>
<td>63-4</td>
<td>..</td>
<td>1-03</td>
<td>28-3</td>
<td>7-1</td>
<td>14% †</td>
<td>3% †</td>
<td></td>
</tr>
<tr>
<td>Clini et al, 2002</td>
<td>43</td>
<td>64</td>
<td>66</td>
<td>27%</td>
<td>26</td>
<td>6-7</td>
<td>18%</td>
<td>17%</td>
<td></td>
</tr>
</tbody>
</table>

**NOTE:** In the trials that have looked at mortality – either LI-PS was used and there was no mortality benefit OR HI was used – EXCEPT Struik
Severe COPD: High Intensity vs no NIV

Mortality

- PRCT – 12 mo observation
- Mortality – no difference
- PCO2 – (base ~58)↓ by 3.75 mm Hg
- TcCO2 – ↓ by 4.5 mm Hg
- Spiro - no difference (FEV1=27%)
- HRQoL – trend (not significant) to NIV
- Mood - no difference
- Dyspnea - no difference

Severe COPD: High Intensity vs no NIV

Why is there a difference?

• The patients were enrolled after AECOPD
• Their hypercapnia may not have represented a true baseline
• They did not have significant hypoxemia (Baseline PaO2 ~60)

What about Re-Admission?

Severe COPD: High Intensity PS-HOT-HMV – New data from the UK

• RCT of NIV in hypercapnia due to COPD post acute exacerbation

Introduction: The study is designed to investigate the effect of home mechanical ventilation (HMV) in patients with severe chronic obstructive pulmonary disease (COPD). The goal is to test the hypothesis that HMV and home oxygen therapy (HOT) reduce hospital re-admission compared to HOT alone in COPD patients post acute hypercapnic exacerbation. The primary outcome is 1-year admission free survival.

Method: multicenter randomized-controlled UK trial (2010) with intervention

<table>
<thead>
<tr>
<th>Patient Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>• BMI = 22</td>
</tr>
<tr>
<td>• LTOT before enrollment = 70%</td>
</tr>
<tr>
<td>• &gt; 3 COPD admit /last year = 53%</td>
</tr>
<tr>
<td>• FEV1 = 23%</td>
</tr>
</tbody>
</table>

n = 116 COPD patients | HOT-HMV group = 57 patients | HOT group = 59 patients

Participants are randomly allocated to receive home oxygen therapy with or without home mechanical ventilation. They are followed up for 12 months to see if patients who received home mechanical ventilation have fewer hospital re-admissions.

https://clinicaltrials.gov/ct2/show/NCT00990132
Severe COPD: High Intensity PS-HOT-HMV – New data from the UK

Participant inclusion criteria
1. In patient admit for Acute hypercapnic COPD at least 2 weeks
2. Smoking >20 P-Yr-Hx
3. FEV1 < 50%
4. FEV1/FVC <60%
5. PaCO2 >52.5
6. PaO2 <55 or < 60 with polycythemia or PHTN
7. Nocturnal hypoxia (SpO2 <90% for >30% sleep time)

Summary of Primary Settings:
• IPAP = ~24 cmH2O (22 to 26)
• EPAP = ~4 cmH2O (4 to 5)
• BUR = ~14 (14 to 16)

Devices/Modes Utilized:
• Of those receiving NIV, half received the ResMed VPAP III ST-A
• The other half received the Respironics Harmony 2
• Both devices were used in ST mode.
**Severe COPD: HI PS- HOT-HMV – New data from the UK**

**Results**: The addition of home mechanical ventilation (HMV) to home oxygen therapy (HOT) improved hospital readmission. The median time before readmission was 3 times higher in the HOT-HMV group than in the HOT group: 4.3 months versus 1.4 months.

1. Murphy p et al. Improving admission free survival with home mechanical ventilation (HMV) and home oxygen therapy (HOT) following life threatening COPD exacerbations. Abstract presented at the European Respiratory Society annual congress 2016.

![Graph showing admission free survival by treatment arm]

HOT-HMV reduces readmission or death by 50%
Europe vs US

Key Factors

• Who manages home based NIV
• Weight of the patients
• Availability of sleep labs
• Availability of in patient beds
• Access to out patient respiratory care support
  – RT’s vs Physiotherapist
A US experience- Study #1

Retrospective Evaluation

- Hypercapnic (PaCO2 = 55) COPD patients continue NIV after D/C for AECOPD.
- Average settings 22/6
- Continued from ICU settings

RESULTS
- Reduction in hospital readmissions
  - 40% vs 75%, p < 0.0001
- Improved mortality

A US experience - Study #2

- Population
  - GOLD 2-4
  - BODE Index Score ≥5
  - Either
    - PaO2 ≤ 60
    - PaCO2 ≥ 52
- Standard therapy
- Then a test of adding NIV

“Real World Test”

Our objective was to test the hypothesis that
  - advanced PAP modality – a form of NIPPV mode – called averaged volume assured pressure support with auto-expiratory positive airway pressure (AVAPS-AE), which is capable of treating hypoventilation due to COPD, could reduce rehospitalizations in an “at-risk” COPD population.

A US experience- Study #2

Study Plan

• Retrospective study (N=397)
  – Mostly Male and Caucasian
  – Complex medically (1/3 with CAD/HF/DM/Anxiety)

• Quality improvement programs
  – Barnes Healthcare Services (Valdosta, GA)
    – 2010-2014

• Program components
  – Pharmacist for med teaching
  – RT for
    • AVAPS- AE /NIV support
    • Home O2
    • Care Co-Ordination
      – Visits q30 d X 3 visit then q90 d
    • Stop smoking plans

Study Results

Table 2: Hospital Re-admissions following initiation of quality improvement program:

<table>
<thead>
<tr>
<th>Number of COPD-related Admissions</th>
<th>Patients with admission in the year prior to program initiation (n [%])</th>
<th>Patients with admission in the year post program initiation (n[%])</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0 (0%)</td>
<td>348 (87.7%)</td>
</tr>
<tr>
<td>1</td>
<td>0 (0%)</td>
<td>40 (10.1%)</td>
</tr>
<tr>
<td>(\geq 2)</td>
<td>397 (100%)</td>
<td>9 (2.2%)</td>
</tr>
</tbody>
</table>

Severe COPD - NIV therapy initiation in the US

The role of the sleep lab

• The AASM currently has no protocol suggestions on the support of patients with Severe COPD.
• Can you do this in your lab?
  – No agreed upon titration algorithms in the US
  – When and how to add o2 vs increase PS is challenging for both tech’s and doc’s
  – The role of CO2 monitoring is of uncertain value
    • Normalizing is not realistic given the time needed to normalize bicarb
    • Preventing elevation may be the best short term goal
  – The reduction in WOB is the best out come but it is unclear how this can be measured in the sleep lab
Severe COPD- NIV therapy initiation in the US

The role of the sleep lab

OSA/COPD Overlap Syndrome

- These patients are treated under the scheme for Obstructive Sleep Apnea
  - This requires polysomnography
  - These patients are most often treated with CPAP therapy
  - The goal of PAP therapy is to treat concurrent OSA – NOT therapy for COPD.

Severe COPD with Respiratory Failure

- This is a broad category which does not actually require a diagnosis of COPD
  - These patients have BOTH hypercapnea (CO2 > 52) AND hypoxemia (O2 saturation <88% for ≥5mins.)
  - OSA is not thought to be the cause of respiratory disturbance. (PSG no longer required to prove this)
COPD with daytime fatigue +/- persistent dyspnea

Is the PaCO2 ≥ 52?

Yes

Obtain Overnight Oximetry

Sat ≤ 88% on 2L- NC

Yes

Diagnosis: **Severe COPD**

Start either HI-PS vs LI – PS Bi-level (S)

No diagnostic or titration PSG needed

No

Obtain Polysomnogram

AHI > 5/hr

No

Yes

Diagnosis: **OSA/COPD overlap**

Start with CPAP therapy

A titration PSG is recommended***

No

Diagnosis: **COPD**

No PAP

***Insurance may require an auto pap

Lab Protocols
Severe COPD- NIV
The role of the sleep lab

A limited monitoring approach

- Protocol
  - TCM / O2 monitor
  - Trained Nurse
- A potential template for a lab titration protocol

Murphy, PB Thorax. 2018 Mar 30. [Epub ahead of print]
Severe COPD- NIV therapy initiation in the US
The role of the sleep lab

Goals for titration

• What PS is needed?
• What Mode of ventilation can be used?
  – S vs ST vs VAPS
• What is the Role of the technologist in the study?

Timing of the study

• The importance of starting therapy during an AECOPD
  VS
• The importance at studying during a period of stability
Severe COPD- NIV therapy initiation in the US

The role of the sleep lab

Meta- Analyses of 7 studies

- CO2 improvements
  - For IPAP of >18
  - For >5hr/nt of use

AVAPS vs PS in Chronic COPD

- Patients randomized to AVAPS vs PS in a crossover model
  - Target was 8 cc/kg
  - Mode was ST
  - CO2 improved in both arms
    - 63 to 54 in PS (IPAP avg = 16.8)
    - 60 to 55 in AVAPS (IPAP avg = 18.7)
  - Compliance was equal with both technologies

AVAPS vs PS  
Acute Hypercapnic COPD Exacerbation

- N=22 with COPD exacerbations and hypercapnic encephalopathy with:
  - A GCS <10
  - A pH of 7.25-7.35
  - Therapy assignments:
    - N= 11 BiPAP S/T with AVAPS
    - N= 11 BiPAP S/T

<table>
<thead>
<tr>
<th></th>
<th>Initial PaCO₂</th>
<th>Hr 1 PaCO₂</th>
<th>Hr 3 PaCO₂</th>
<th>Hr 12 PaCO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bipap ST</td>
<td>64.8</td>
<td>58.3</td>
<td>53.2</td>
<td>50.1</td>
</tr>
<tr>
<td>AVAPS</td>
<td>63.0</td>
<td>50.7</td>
<td>45.4</td>
<td>43.6</td>
</tr>
</tbody>
</table>

Briones Claudett et al. BMC Pulmonary Medicine 2013, 13:12
Studies of VAPS in COPD

HI – NPPV vs VAPS

- N=27 stable COPD pt’s on HI – NIPPV
- MV was unchanged between devices
- IPAP avg = 24.1

Emelie Ekkernkamp COPD, 11:52–58, 2014
Severe COPD - NIV therapy initiation in the US
The role of the sleep lab

Step 1:
While awake before starting the study
- Pre heat water in the humidifier
- Minimize O2 bleed in

Patient falls asleep

Step 2:
Pick a mode
- Bi-Level S for a RAD device
- VAPS ST for a Mechanical Vent with Mask

Step 3:
EPAP
- Increase for snoring
- Increase for obstructive

IPAP
- Increase to try and reach >18
- Increase for high RR
- Increase to reach Vtex 8cc/kg IDBWT

Ti time
- Short to allow for longer exhale time as per patient comfort
  - Fast rise
  - Limited Ti (Ti max)

Timing
- Adjust trigger and cycle to comfort and reduce dys-synchrony
  - Trigger – medium to low
  - Cycle – medium to high
A US experience- Study #2

Skip the sleep lab

- AVAPS- AE mode
- Barnes study Targeted 5-7 cc/kg

Barnes Settings
- Vt Target: 5-7 cc/kg
- RR: Auto
  - set lower than awake spontaneous RR
  - Allow for adequate deflation
  - Avoid air trapping
- EPAP: 5-15
  - set in auto mode
  - To prevent upper airway collapse
- PS: 2-26
  - HI vs LI – not the point here – in this case PS is a slave to the Target VT
Severe COPD: Deventilation Syndrome

- Morning deventilation dyspnea
- Hypothesis
- Progressive hyperinflation
- Patient-ventilator asynchrony (PVA)
  - Negative impact on sleep quality
  - Reported PSG’s with NIV in COPD have not looked for markers of asynchrony.
- Addition of diaphragm EMG to score unrewarded breaths
- Scoring of double and auto (Device triggered) triggered breaths
  - Addressing PVA improved sleep quality and AM dyspnea


To Reduce PVA:

A) unrewarded inspiratory efforts:
   1) reduced pressure support,
   2) if needed increase (EPAP)
   3) set expiratory trigger at a higher percentage of peak inspiratory flow to avoid delayed cycling
   4) start with rise to 100 msec, and increased it by 50–100 msec.
   5) Increase back-up respiratory rate was increased
   6) Maximal inspiratory time (T_{I,MAX}) was set so that the I/E ratio was 1:2.5m - 1:3.

B) Auto-triggering was identified
   1) Mask fit was re-fit to prevent air leaks
   2) Inspiratory trigger sensitivity was reduced.

C) Double triggering
   1) Inspiratory time (T_{I,MIN}) increased
# What is the cost of therapy?

<table>
<thead>
<tr>
<th>CMS Name</th>
<th>Common Name</th>
<th>Monthly Costs USD</th>
<th>Diagnosis</th>
<th>Qualifying Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventilator</td>
<td>Invasive Mechanical Ventilator, Home Ventilator</td>
<td>~1500</td>
<td>Respiratory Failure</td>
<td>Unclear??????</td>
</tr>
<tr>
<td>E0464</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory Assist Device</td>
<td>BilevelPAP(S), BiLevelPAP(ST)</td>
<td>~400</td>
<td>Severe COPD</td>
<td>PaCO2 ≥ 52 AND 02 sat &lt;88% for &gt;5min on 2 L NC</td>
</tr>
<tr>
<td>E0470 E0471</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPAP Device</td>
<td>CPAP, autoCPAP</td>
<td>~100</td>
<td>OSA</td>
<td>AHI &gt; 5 with sx</td>
</tr>
<tr>
<td>E0601</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# CMS Nation Coverage Determination

<table>
<thead>
<tr>
<th>Severe COPD with Respiratory Failure Treated with a Ventilator with Mask E0464</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Documentation needs to support requirements for ventilator therapy.</td>
</tr>
<tr>
<td>- Documentation needs to contain a valid date stamp /MD signature.</td>
</tr>
<tr>
<td>- Proof of Delivery is on file, with a patient signature.</td>
</tr>
<tr>
<td>- Medical documentation must be on file.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Imminent Death</th>
</tr>
</thead>
<tbody>
<tr>
<td>- No agreed upon definition</td>
</tr>
<tr>
<td>- Use would need to be anywhere from 20-24 hours a day but this is not documented by CMS</td>
</tr>
</tbody>
</table>

Why do we care?

Figure 1: Claims for E0464 Ventilators Grew Faster Than Those for Other Ventilators

Is it happening to you?

- Three DME’s appear to be driving most of the costs


Diagnoses on E0464 ventilator claims have shifted from neuromuscular to respiratory conditions
Why are physicians spending all this money?

- A fundamental misunderstanding of the healthcare set up for our patients with advanced pulmonary disease
  - Driving to a store and buying tanks is unrealistic
  - In home RT services are only provided when covered by this fee
- These concerns lead to a competitive bidding program
Why are physicians spending all this money?

- In using a mechanical ventilator with mask – The physician will be able to
  - Pick from a wider range of vendors
  - Assure RT care for the patient at home
  - Avoid having to comply with the RAD Criterion
  - Facilitate NIV set up after AECOPD
Hypoventilation

What can that mean?

• Generic basket term

What disorders could this include?

  – Obesity Hypoventilation
  – Central Congenital Hypoventilation Syndrome
  – Diaphragm Disease
    • Bilateral damage due to distant radiation (remote Hodgkins)
    • Parsonage Turner Syndrome (acute brachial neuropathy and acute brachial amyotrophy)

Definition of OHV:
1) BMI ≥ 30
2) PaCO2 > 45
3) PH > 7.35
   1) base excess ≥ 2
4) No COPD, NMD, Scoliosis
5) Most often – OSA AHI > 30

RAD criterion

• An initial PaCO2 done while awake and breathing the patient’s prescribed FIO2 is ≥ 45 mm Hg.

AND

• Spirometry shows an FEV1/FVC ≥ 70% and an FEV1 ≥ 50% of predicted.

AND

1. An arterial blood gas PaCO2 done during sleep worsens ≥ 7 mm Hg compared to the original result in Criterion A.

OR

2. A facility-based PSG demonstrates oxygen saturation ≤ 88% for 5 minutes of nocturnal recording time (minimum recording time of 2 hours) that is not caused by obstructive upper airway events.
Hypoventilation

What to look for on a sleep study?

• Obesity Hypoventilation -
  – ETCO2/ TCM elevations in SWS and REM
  – Severe OSA/ Tachypnea

• Central Congenital Hypoventilation Syndrome
  – ETCO2/ TCM – elevations in NREM
  – NO discrete apnea/hypopnea events
  – Hypoxemia may not be present

• Diaphragm Disease
  – ETCO2/ TCM – may be elevated during wake
  – May not be able to lay flat for study
  – Drops out of proportion in REM

PSG

• Score as hypoventilation during sleep:
  – when >25% of the total sleep time as measured by either the arterial PCO2 or surrogate is spent with a PCO2 >50 mm Hg

OR

• Score hypoventilation during sleep if EITHER of the below occur:
  – There is an increase in the arterial PCO2 (or surrogate) to a value >55 mmHg for ≥10 minutes.

OR

  – There is ≥10 mmHg increase in arterial PCO2 (or surrogate) during sleep (in comparison to an awake supine value) to a value exceeding 50 mmHg for ≥10 minutes.
Obesity Hypoventilation: CPAP vs NIV vs Life style changes

- Subjects with OHV with OSA. Randomized to CPAP v NIV v Life style changes – 2mo follow up
- PCO2
  - Lowest in NIV Group
  - No diff CPAP vs NIV
- HRQoL
  - NIV better for
    - 6MW
    - FEV1 and FVC
  - CPAP = NIV
    - ESS
    - Unrefreshing sleep
    - Nocturia
    - Headache

<table>
<thead>
<tr>
<th></th>
<th>NIV</th>
<th>CPAP</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oxygen therapy, %</td>
<td>24</td>
<td>24</td>
<td>15</td>
</tr>
<tr>
<td>Oxygen flow, mean (SD), L/min</td>
<td>2.1 (1)</td>
<td>1.8 (0.8)</td>
<td>2 (0.8)</td>
</tr>
<tr>
<td>Pressures, mean (SD), cm H2O</td>
<td>IPAP</td>
<td>20 (3.3)</td>
<td>20 (3)</td>
</tr>
<tr>
<td></td>
<td>EPAP</td>
<td>7.7 (1.8)</td>
<td>7.8 (1.8)</td>
</tr>
<tr>
<td>Respiratory rate, mean (SD)</td>
<td>14 (3)</td>
<td>14 (3.1)</td>
<td></td>
</tr>
<tr>
<td>Mask, %</td>
<td>Nasal</td>
<td>7</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>Full-face</td>
<td>93</td>
<td>89</td>
</tr>
<tr>
<td>Compliance, mean (SD), h/d</td>
<td>5.3 (2.3)</td>
<td>5.3 (2.1)</td>
<td></td>
</tr>
</tbody>
</table>

Masa - Am J of Resp and CCare Med V (192) N(1) July 1 2015

N= 221
Hypoventilation- OHV therapy

• Which is better CPAP vs NIV?
  – ~57% of patients can be successfully treated with CPAP alone – and only compliance predicts the likelihood of resolving daytime hypercapnea.
  – Initial therapy with NIV does not improve compliance or efficacy (in regards to daytime hypercapnea)
  – NIV should be used when:
    • CPAP fails to resolve hypoxemia or apnea
    • Pressure intolerance occurs

Hypoventilation: OHV

Benefits of NIV
- PaCO2 improves
- ESS Improves
- Mortality is reduced
- Compliance is excellent
- Improves QoL

Where NIV fails
1) Pascaline Priou et al CHEST 2010; 138(1):84–90
3) Vassiliki Tsolaki et al Respiration 2011;81:402–410

Apneas may resolve but arousal index may get worse
PAT signal as a marker of endotheial related damage does not imporve
Arterial Stiffness does not improve
Cytokines do not improve – CRP, Leptin, RANTES, IL-6/8, TNF-α, Adiponectin,
Hypoventilation: Back Up Rate

<table>
<thead>
<tr>
<th>Ventilator Data</th>
<th>S Mode</th>
<th>S/T Mode Low BURR</th>
<th>S/T Mode High BURR</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RR, /min</td>
<td>15 (13, 19.5)</td>
<td>14 (12.5, 16.5)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>22 (21, 22)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.006</td>
</tr>
<tr>
<td>RR, 95th percentile, /min</td>
<td>21 (18.5, 25)</td>
<td>21 (16.5, 23.5)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>25 (22.5, 28.5)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.003</td>
</tr>
<tr>
<td>Leaks, L/min</td>
<td>3.6 (1.8, 7.8)</td>
<td>6 (1.2, 9)</td>
<td>6 (2.7, 15.3)</td>
<td>.6</td>
</tr>
<tr>
<td>Leaks, 95th percentile, L/min</td>
<td>12.6 (7.5, 24.3)</td>
<td>13.2 (3.19.2)</td>
<td>21.6 (8.4, 43.5)</td>
<td>.8</td>
</tr>
<tr>
<td>Vt, mL</td>
<td>600 (500, 825)</td>
<td>600 (475, 675)</td>
<td>425 (400, 587)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.05</td>
</tr>
<tr>
<td>Vt, 95th percentile, mL</td>
<td>750 (625, 1050)</td>
<td>800 (550, 1050)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>9.5 (8.8, 14.7)</td>
<td>.135</td>
</tr>
<tr>
<td>Ve, L/min</td>
<td>10.6 (9.4, 12.6)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>8.7 (8.2, 9.8)</td>
<td>9.5 (8.8, 14.7)</td>
<td>.135</td>
</tr>
<tr>
<td>Ve, 95th percentile, L/min</td>
<td>16 (13.5, 19.5)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>12.8 (11.6, 17.5)</td>
<td>17.1 (12.3, 24.1)</td>
<td>.061</td>
</tr>
<tr>
<td>% cycles triggered by patient</td>
<td>100 (100, 100)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>72 (55.5, 82.2)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>24 (11.2, 36.7)&lt;sup&gt;b&lt;/sup&gt;</td>
<td>.001</td>
</tr>
<tr>
<td>AHI, /h</td>
<td>60 (44, 77)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>19 (9, 40)</td>
<td>12 (3, 26)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>.006</td>
</tr>
<tr>
<td>Central AHI, /h</td>
<td>17.7 (13.7, 37.6)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.8 (0.1, 4.1)</td>
<td>0.2 (0, 4.4)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Mixed AHI, /h</td>
<td>9.5 (4.7, 13.2)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.1 (0, 1.7)</td>
<td>0 (0, 0.6)&lt;sup&gt;d&lt;/sup&gt;</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Obstructive AHI, /h</td>
<td>22 (15.3, 37.3)</td>
<td>17.2 (4.1, 36.5)</td>
<td>8.6 (1.8, 21.6)</td>
<td>.067</td>
</tr>
<tr>
<td>PVA, % TST</td>
<td>11 (7, 20)</td>
<td>8 (2, 32)</td>
<td>6 (0, 31)</td>
<td>.729</td>
</tr>
<tr>
<td>Mean Pco2, mm Hg</td>
<td>44 (40, 47)</td>
<td>44 (41, 50)</td>
<td>47 (38, 52)</td>
<td>.117</td>
</tr>
<tr>
<td>Average Spo2</td>
<td>92 (91, 94)</td>
<td>92 (91, 93)</td>
<td>92 (91, 94)</td>
<td>.575</td>
</tr>
<tr>
<td>Minimal Spo2</td>
<td>79 (73, 85)</td>
<td>82 (79, 86)</td>
<td>83 (77, 86)</td>
<td>.590</td>
</tr>
<tr>
<td>Spo2 &lt;90%, % TST</td>
<td>13 (4, 27)</td>
<td>11 (5, 27)</td>
<td>7 (2, 23)</td>
<td>.670</td>
</tr>
<tr>
<td>ODI ≥4%, /h</td>
<td>59 (52, 71)&lt;sup&gt;a&lt;/sup&gt;</td>
<td>26 (15, 39)</td>
<td>19 (7, 36)&lt;sup&gt;c&lt;/sup&gt;</td>
<td>.002</td>
</tr>
</tbody>
</table>

Olivier Contal et. al. CHEST 2013; 143(1):37–46
Hypoventilation and VAPS

Hard to demonstrate benefit

- N=50 (bmi ~50)
- RCT:
  - AVAPS (ST)
  - vs
  - Philips mode ST
- CO2 improved for all pt’s
  - No advantage to VAPS
- NOTES:
  - Goal of TarVolume of 8-10 cc / g IdBwt
  - PC did not seem to make a difference
    - but Ti time was not reported

Murphy, PB Thorax 2012;67:727-734
Hypoventilation: Supplemental Oxygen

O2 and Mortality in OHV

• Oxygen therapy alone increases CO2
  – Both 28% and 50% FIO2 elevate CO2
    • PaCO2:
      – RA =53, 28% =56, 50% =58

• Oxygen therapy when used in combination with NIV also worsens mortality

Survival curve by Kaplan-Meier analysis for the overall population (N = 130) and for patients with (n = 20) and without (n = 110) supplemental oxygen.

1) Pascaline Priou et al CHEST 2010; 138(1):84–90
Hypoventilation: No OSA

- N= 86 (23% of screened pt’s with obesity and hypercapnea)
  - 79% female
- Randomized to NIV or Life style changes
- PaCO2 in the daytime reduced – dropping 6 points on average
- Oxygen improved on NIV
  - AHI(3%) 14→11
  - Mean SAO2 88 → 94%
- Improvements were modest:
  - ESS / Tiredness
  - AI and TST / Unrefreshing sleep
  - Healthcare resources utilization
  - Mental well being (VAWS/SF-36)

Neuromuscular diseases involving respiratory function

- motor neuron disease (amyotrophic lateral sclerosis)
- poliomyelitis and postpolio syndrome
- myasthenia gravis including myasthenic syndrome
- acute inflammatory demyelinating polyradiculoneuropathy (Guillain–Barre’ Syndrome)
- phrenic neuropathy
- muscular dystrophies including myotonic dystrophies
- congenital myopathies
SDB in NMD – The Brain Stem

1) Brain Stem activity that controls ventilation involves multiple nuclei.
2) Respiratory timing and chemosensitivity are gated through these areas.
3) These areas can be impacted by central neurodegenerative conditions such as MSA, or combined central and peripheral neurodegeneration such as ALS.

### TABLE 1. Brainstem groups involved in control of respiration

<table>
<thead>
<tr>
<th>Localization</th>
<th>Nucleus</th>
<th>Primary neurotransmitter(s)</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pontine respiratory group</td>
<td>Medial parabrachial/Kölliker Fuse</td>
<td>Glutamate, GABA</td>
<td>Phase switch between inspiration and expiration</td>
</tr>
<tr>
<td>Dorsal respiratory group</td>
<td>Lateral parabrachial</td>
<td>Glutamate, GABA</td>
<td>Inspiration</td>
</tr>
<tr>
<td></td>
<td>Nucleus solitary tract</td>
<td>Glutamate, GABA, glycine</td>
<td>Integration of respiratory and cardiovascular inputs and initiation of respiratory reflexes</td>
</tr>
<tr>
<td>Ventral respiratory group</td>
<td>Bożtinger complex</td>
<td>Glutamate, GABA</td>
<td>Expiration</td>
</tr>
<tr>
<td></td>
<td>PreBożtinger complex</td>
<td>Glutamate, GABA</td>
<td>Rhythmogenesis</td>
</tr>
<tr>
<td></td>
<td>Rostral VRG</td>
<td>Glutamate</td>
<td>Chemosensitivity</td>
</tr>
<tr>
<td></td>
<td>Caudal VRG</td>
<td>Glutamate</td>
<td>Inspiration</td>
</tr>
<tr>
<td></td>
<td>Nucleus ambiguus</td>
<td>Acetylcholine</td>
<td>Expiration</td>
</tr>
<tr>
<td></td>
<td>Raphe pallidus</td>
<td>Serotonin</td>
<td>Laryngeal motoneurons</td>
</tr>
<tr>
<td>Putative chemosensitive and modulatory areas</td>
<td>Arcuate nucleus</td>
<td>Glutamate, serotonin, acetylcholine</td>
<td>Modulation</td>
</tr>
<tr>
<td></td>
<td>A5, A6, A1 group</td>
<td>Norepinephrine</td>
<td>Chemosensitivity</td>
</tr>
<tr>
<td></td>
<td>LDT/PPT</td>
<td>Acetylcholine</td>
<td>Chemosensitivity</td>
</tr>
</tbody>
</table>

GABA, gamma-aminobutyric acid; LDT, laterodorsal tegmental nucleus; PPT, pedunculopontine nucleus; VRG, ventral respiratory group; REM, rapid eye movement.

Upper to Lower Motor Neurons:

- Originate in the motor region of the cerebral cortex or the brain stem.
- Carry motor information down to motor neurons that are not directly responsible for stimulating the target muscle.
- Connect the brain to the appropriate level in the spinal cord.
Issues in NMD control of ventilation in sleep.

- Obstructive sleep apnea
  - Pseudoobstructive apnea:
    - Thoraco abdominal paradox occurs due to diaphragm weakness not upper airway obstruction
- Central apnea
  - Pseudocentral apnea:
    - Obstruction of the airway is present but does not become apparent as weakness of the diaphragm prevents development of flow strong enough to collapse the upper airway.

Issues in NMD control of ventilation in sleep.

- Central apnea
  - Traditional central apnea:
    - may occur when associated brain stem involvement may occur
    - These events are more common in Stage REM
  - Central hypoventilation:
    - Most common in NREM sleep and associated with diaphragm weakness
    - Reduction hypocretin reduce ventilatory control in specific settings such as myotonic dystrophy or myesthenia gravis

- Central hypoventilation in NREM
- Central apnea in REM
When to start NIV in NMD: Overnight CO2 testing

- When looking at mortality of many trials the overall finding:
  - **Peak nocturnal TcCO2 ≥49 mmHg** should be considered as one of the criteria to start HMV in patients with NMD.

  - AASM: TcCO2 >55 mmHg for ≥10 minutes or increase in TcCO2 ≥10 mmHg
  - Ward: Peak TcCO2 ≥49 mmHg
  - Simonds: mean TcCO2 >50 mmHg
Role of polysomnography in NMD

- Waiting for sx’s is too late
- Abnormal PFT’s is too late

Does NIV therapy make a difference?

- Retrospective data
- N= 929
- Survival NIV = 28 mo. vs 15 mo in no NIV
- Even bulbar pt’s had a benefit

Indications for PSG in SMA

• Standard of Care Guidelines:
  – Spinal Muscular Atrophy
    • Hypoventilation is an issue
      – not discrete central or obstructive apnea
    • Most specialists recommend annual monitoring for patients whose FVC is $\leq 65\%$, in order to initiate NIV.
      – monitor with overnight pulse oximetry is used by some.
      – monitoring with polysomnography is optimal because it allows for ETCO2 monitoring for hypoventilation.
    • If the patient is placed on noninvasive ventilation
      – Re-evaluation with polysomnography annually
        » Growth, scoliosis, weakness

*J Child Neurol* 2007; 22; 974
## Indications for PSG in DMD

### American Thoracic Society

- **Standard of Care Guidelines:**
  - Duchene Muscular Dystrophy
    - PSG yearly once a patient requires a wheel chair
    - PSG to pre operatively especially in evaluation of kyphoscoliosis
    - NOTE: these recommendations are from the pre steroid era.

### Parent Project Muscular Dystrophy

- **DMD Care Considerations Working Group:**
  - Duchene Muscular Dystrophy
    - Diagnostic PSG with capnography for signs and symptoms of obstructive sleep apnoea or sleep-disordered breathing
    - Nocturnal NIV with rate
      - FVC <50%
      - MIP <60 cm H2O
      - Awake SpO2 <95%
      - Awake PCO2 >45 mm Hg

---


Lancet Neurol 2018; 17: 347–61
Indications for PSG/NIV in ALS

**AAN Guidelines**
- Measured upright or supine and these are OR’s not AND’s
  - Dyspnea / Orthopnea
  - Sniff nasal pressure < 40 cm H2O
  - Maximal inspiratory pressure (MIP) < −60 cm H2O
  - Abnormal nocturnal oximetry
  - Forced or Slow vital capacity < 50%

**More is More Philosophy**
- Early initiation of therapy at FVC of 70%.
  - improved survival
  - As compared to FVC < 50%
    - early initiation of NIV improves survival to 2.7 years vs. 1.8 years.
  - To help facilitate early initiation of NIV
    - MIP criterion
    - FVC attained in the supine position
    - overnight oximetry testing


General indications for PSG/NIV in Neuromuscular Disease

• Respiratory Assist Device “RAD” criterion from CMS:
  – Restrictive Thoracic Disorders
    • neuromuscular disorders and chest wall disorders

• Criterion:
  – EITHER:
    • FVC<50%
    • MIP>-60
    • PaCO2>50
    • Oximetry <88% for >5 min on a recording of at least 2 hours
Indications for PSG in NMD: “The Don’ts”

- Don’t delay the start of NIV awaiting a PSG
- Don’t order a PSG unless the sleep lab has a protocol for NMD
- Don’t order a PSG unless the lab has physical accommodations for those with disabilities
  - Lifts, Hospital beds, Adaptive call system, Care giver space, Bed side commodes, Suction
- Don’t get diagnostic data if any of the RAD criterion have been documented.
Developing your protocol:
What are your goals

No Agreements but options include:

- “Rest”
- Improve ventilation
- Resolve hypoxemia
- Resolve central apnea
- Improve sleep quality
- Optimize / personalize settings for comfort

Concerns: what is rest?

- Options:
  - Ride the back up rate –IE no spontaneously triggered events
  - Reduce WOB
    - Recruit atelectasis
    - Reduce rapid shallow breathing
Developing your protocol:
How to extend the montage /CO2

ETCO2 vs TCM

• There was a constant, and usually close, relationship, between PtCCO2 and PETC02
• PtCCO2 monitoring is best for
  – children who would not tolerate a nasal sampling tube
  – Those with moderate to severe partial airway obstruction,
  – Those with tachypnea,
  – Those with increased physiologic dead space in whom PETC02 underestimated Ptcco. (CHF)

Developing your protocol:
How to extend the montage/ Diaphragm assessment

• To get reliable results your patient should have a body that look like this

http://lsc.univ-evry.fr/~vvigne/doku.php?id=emgd

This work is realised in collaboration with T. Similovski and C. Strauss from Hospital La Pitié-Salpêtrière
NMD Extended Montage EMG

1. External nares
   - levator alae-nasi muscles

2. Posterior triangle of the neck
   - anterior scalene muscle

3. 1/2 between the chin and the hyoid
   - genioglossus among submental muscles

4. 3rd intercostal space
   - Parasternal intercostal muscle

25 year old with achondroplastic dwarf with kyphoscoliosis. Extended montage EMG showing increase in accessory muscle use with ineffective ventilation and disynchrony.