A Community Bilevel Service
Treating Obesity Hypoventilation

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- 500,000 people – 11.5% of NZ population
- Culturally diverse – Māori 16%; Pacific 23%; Asian 22%; Other 38%
- Young 24% < 14 Years
- Growing – 10 000 new residents each year
- Decile 9 and 10 – 34% of CMDHB population
- Health disparities with Pacific and Māori
- Obese BMI > 25 – 66%; BMI > 30 – 33%(2006/7)
• Mostly achieved better than required on health targets (x smoking and heart/diabetes checks)
• Innovative – good ideas are encouraged
• Multi cultural staff – speaking patients languages
• Focus on services into the community and involving community in designing these.
Obesity Hypoventilation Syndrome

Combination of

- **Obesity** - body mass index of > 30 kg/m²
- **Daytime hypercapnia** - PaCO₂ > 45 mmHg in the absence of other known causes of hypoventilation
- Sleep disordered breathing
What does this mean?

- Ventilatory failure – breathing response is blunted, and not strong enough to rid the body of CO$_2$.
- Rise in CO$_2$ levels is detrimental in the body and ultimately can lead to coma and death.
- Caused by mechanical restriction of ventilation due to excessive fat around the chest wall.
- Aetiology not clearly understood.
Main function of the Lungs is to exchange O$_2$ and CO$_2$ between blood and air to maintain normal levels of arterial O$_2$ and CO$_2$.

How is arterial PO$_2$ and PCO$_2$ are kept within normal limits?

By controlling ventilation
Signs and Symptoms

- Fatigue
- Excessive daytime sleepiness
- Morning headaches
- Frequent hospitalisations
- Minor infection results in complex hospital stay
- Heart failure
Bilevel Service

- Started by innovative accident in 2001
- Approx 65 patients now
- 2007 formal clinics set up – NP led
- Patients predominantly Pacific and Māori, young - mid to late 40’s, morbidly obese – ave BMI 50’s to 60’s.
- 3 patients with Pompe’s, SLE, MS, Kyphoscoliosis, Bronchiectasis, mostly OSA/OHS or pure OHS
Case study - Evan

- First presented to EC – May 2012
- 29 year old 220kg Cook Island Maori man (BMI 61.6) presented with coma secondary to type 2 respiratory failure on a background of 1/52 history of URTI
- ABG on admission - it is arterial!!
- pH 7.17 ;pCO2 15.1 ;pO2 3.8 ;BE +5
Case study - Evan

- Bilevel in ICU, off bilevel after 48 hours
- Discharge ABG - pH 7.36; pCO2 9.1; pO2 6.1; Bicarb 38
- With sleep study planned for 6/52
- Apnea Hypopnoea Index (AHI) of 136.5 events/hour with an average desaturation of 9.1% consisted of mainly clear-cut obstructive apnoeas occurring while the patient was supine.
- Average oxygen saturation was 85.1% and nadir was 67% during NREM.
Case Study - Evan

- CPAP was applied for a limited period via a F&P Zest nasal mask from 0030 hours to 0300 hours. All events were abolished at 10cmH2O but due to increasing PaCO2 levels the titration was abandoned and a diagnostic study resumed.

- The study is of adequate severity to confirm severe OSAS with baseline hypoxia, presumably due to hypercapnia. After introduction of CPAP there is REM rebound and a predictable fall in baseline saturation likely due to hypercapnia. CPAP was then withdrawn.
Case Study - Evan

- Initially started on CPAP
- DNA’d sleep clinic twice
- Eventually engaged and then changed to bilevel during another hospitalisation when he came in unwell, but not moribund
- Cholecystitis – ongoing problem, complicated by obesity
- Lost 15 kg
- In progress
Case study – Jack

- 45 years
- Māori man with bronchiectasis – not eligible for lung transplant
- Oxygen dependant (uses portable as well as LTOT)
- Started on bilevel
- Originally kg
- Pulmonary rehab
- Last year – weightloss of ......

Case study - Jack

- Where to next?

- Sleep study and likely change to CPAP
Obesity Hypoventilation

- Lose the weight and lose the bilevel
- Lose the weight and the problem is solved
- But – Obesity is a complex issue and not as simple as it first appears.
Case study - Toa

- 50 year old, Samoan,
- started bilevel 2004
- 153kg – 30 kg weightloss 2011

“no longer has severe obstructive sleep apnoea and obesity hypoventilation syndrome.

She still does have mild OSA with an overall AHI of only 8.1 per hour.”
Case study - Toa

- However, Dec 2013 – 158kg
History of Non Invasive Positive Pressure Ventilation (NIPPV) (NIV)

- NIPPV - 1870 by Chaussier, who used a bag and face mask to resuscitate neonates.
- 1936, Poulton and Oxon “pulmonary plus pressure machine,” a vacuum cleaner blower and a mask to increase the alveolar pressure and thus counteract the increased intrapulmonary pressure in patients with heart failure, pulmonary edema, Cheyne-Stokes breathing, and asthma.
- 1940s, intermittent positive pressure breathing devices developed for high-altitude aviation. Motley, Werko, and Cournand used these devices to treat acute respiratory failure in pneumonia, pulmonary edema, near-drowning, Guillain-Barré syndrome, and acute severe asthma.
The iron lung was an early form of NIV and used in the treatment of polio
• 1980’s Continuous positive airway pressure (CPAP) for obstructive sleep apnea, followed by noninvasive positive-pressure volume ventilation in neuromuscular diseases.

• Bilevel positive pressure devices (ie, with separate inspiratory and expiratory pressures) initially for obstructive sleep apnea and then for diverse neuromuscular diseases.
Bilevel Ventilators

- Two pressures - inspiratory positive airway pressure, or IPAP and a lower pressure during expiration EPAP.
- The difference (gradient) key importance in maintaining alveolar ventilation and reducing Paco2.
- The IPAP acts as pressure support to augment the patient’s effort, maintain adequate alveolar ventilation, unload respiratory muscles, decrease the work of breathing, and control obstructive hypopnea.
- EPAP (CPAP) is set to maintain upper airway patency, control obstructive apnea, improve functional residual capacity, and prevent microatelectasis.
Bilevel Ventilators
Back up rates

- Although the patient should be able to maintain spontaneous breathing on bilevel, a backup rate option can be set for those whose ventilation during sleep may be significantly impaired (e.g., those with neuromuscular diseases, complex sleep apnea, central apnea in congestive heart failure, or obesity-hypoventilation syndrome)
1997 study in patients with COPD and acute respiratory failure, Nava et al found that NIPPV was no more expensive and no more demanding of staff resources than invasive mechanical ventilation in the first 48 hours of ventilation.

Further, after the first few days of ventilation, NIPPV put fewer time demands on physicians and nurses than did invasive mechanical ventilation.
References


Ambrosino N, Vagheggini G. Noninvasive positive pressure ventilation in the acute care setting: where are we? Eur Respir J 2008;31:874–886


References


