Respiratory Dysfunction in Spinal Cord Injury

Brooke Wadsworth
Advanced Physiotherapist
SIU, PAH

The problem

Spinal cord injury affects 80-100 QLDers each year

1. Primary injury occurs
2. Inflammatory process causes oedema which results in ischaemia via compression that leads eventually to spinal cord cell death.
3. Spinal shock – flaccidity, areflexic, all systems shut down (gut, bowels etc.)
4. Cells recover – reflexes & tone return, spasticity
5. Neurological recovery?
What Happens?

• The majority of all newly diagnosed patients with tetraplegia are admitted to ICU and the incidence of intubation ranges from 74%-90%

• Intubation usually for spinal surgery or respiratory deterioration

• Assessment of breathing ability challenging initially (struggle sitting even with abdominal binder, poor endurance so can’t repeat, EBEV & impact of ETT)

• Acutely use Wright spirometer, sustained phonation/voice loudness & then Easyone.

So what?

• Diaphragm responsible for 60-70% of inspiration with intercostal muscles usually accounting for the remainder of inspiration.
  (Winslow, 2003)

• Intercostal muscle flaccid paralysis creates an unstable chest wall so that the negative intrathoracic pressure occurring during inspiration causes paradoxical inward depression of the ribs.

• These forces favouring airway closure may lead to microatelectasis, pneumonia & increased work of breathing.

• Pulmonary compliance deteriorates rapidly because of poor lung expansion & ↓ production of surfactant

  > Fatigue now an issue due to WOB.

• Mucus can block the inflow of air, & the paralysed patient has trouble keeping the airways free of mucus because of weakness of the cough.

• It is during this time that patients are most likely to require intubation (or reintubation!) & ventilation for respiratory support.
  (Claxton et al 1998)

• Damage to cx spine interrupts sympathetic nerve supply to lungs which originates from the upper 6 thoracic ganglia. Parasympathetic innervation arising from vagal nuclei of brainstem remains intact. SCI may have ↑ resting airway tone for this reason & also hypersecretions.

• Higher more complete injuries will result in more significant arrhythmias & these are most common in the first 14 days.
  (Lehmann et al 1987)

• Paralytic ileus may cause vomiting and aspiration

• Pulmonary oedema can affect as many as 50% of individuals with acute tetraplegia.

• The causes are multifactorial & include excessive fluid resuscitation in the presence of hypotension in the acute post injury setting.
  (Lanig & Peterson 2000)

• O₂ requirements- SCI ventilation problem not oxygenation problem!
Ventilation Dysfunction!

• Loss of volume
• Retained Secretions
• Fatigue

The challenge

Only just surviving!!!!... Not thriving!

One in five patients with acute cervical SCI fails extubation. Odds of extubation failure are 2.76 x greater for complete SCI than incomplete SCI.

Respiratory dysfunction is a frequent complication of spinal cord injury. The article discusses the epidemiology of respiratory complications following spinal cord injury, with a specific focus on cervical injuries. The authors present data on the incidence, risk factors, and management of respiratory complications, including pneumonia, retained secretions, and ventilation dysfunction. The research highlights the importance of early intervention to prevent respiratory complications and improve outcomes for individuals with spinal cord injury.
Time since SCI

- Immediately following injury, there is flaccid paralysis of the intercostal & abdominal muscles, with marked paradoxic abdominal & thoracic movement. A reduction in VC to ≈ 20–60% of the predicted value in tetraplegia & 80–90% in paraplegia.
- The paradoxical ventilation is due to the diaphragm contracting against an unstable rib cage & is more marked following cervical injuries.
- Truncal and intercostal tone increase with time, stabilising the rib cage and returning the VC to approximately 60% of the pre-injury level.
- A progressive reduction in the FRC also occurs during this time, associated with atelectasis & basal pulmonary fibrosis.
- RV remains elevated compared with normal population values, especially in the sitting position with the abdomen unbound, although the magnitude of the elevation reduces with time.
- TLC does not appear to recover significantly during this time.

Restrictive pattern

- ↓ VC, IC, PEF
- ↓ TLC
- ↓ ERV due to lack of abdominal muscles
- ↓ FRC (small change) ↑ in lung recoil pressures & ↓ in chest wall recoil forces
- ↑ RV compensation for ↓ in FRC & ERV

Pulmonary function measures in tetraplegia

What matters to PT’s: FVC & FEV₁

- After SCI with higher lesions, especially in people with tetraplegia (Linn et al. 2000; Baydur et al. 2001)
- demonstrate moderate correlation with injury level (Baydur et al. 2001)
- longer duration of injury & smoking are associated with greater loss while incomplete lesions (compared to complete lesions) have lesser degrees of compromise of forced expiratory measures (Linn et al. 2000)
ATS/ERS standard modified for SCI

Spirometry (Kelley et al.)

…to permit excessive back-extrapolated volume and expiratory efforts of less than 6 seconds duration allows for 88% of subjects with chronic SCI to provide acceptable and reproducible spirometric efforts.

MIP/ MEP

Consider using tube-style mouthpiece instead of flange-style as less leak in study of 50 subjects with tetraplegia.

Respiratory muscle strength

Calculation Lower limit of normal (LLN)

Male:
MIP LLN: 62 – (0.15 x age)  62 – (0.50 x age)
MEP LLN: 117 – (0.83 x age)  95 – (0.57 x age)


SNIP

- SNIP measured from RV
- Nasal plug inserted into a nostril. Other nostril unobstructed.
- Detailed instructions can be counter-productive, although a maximal effort is required.

Respiratory muscle strength

Practicalities

- Do you take height in supine as recommended or go by what patient tells you?
- Do you measure weight as recommended or go by what patient tells you?
Cough? How to measure PC

- Standardise instructions
  - Position, lung volume, number of trials

- Choose device and interface
  - Portable spirometer vs paediatric peak flow meter
  - Spirometer more accurate
  - Mask vs mouthpiece
  - Mask recommended

- Use same device and interface.
- Interpret trend, not absolute values.

Obstructive dysfunction

- Damage to cx spine interrupts sympathetic nerve supply to lungs which originates from the upper 6 thoracic ganglia. Parasympathetic innervation arising from vagal nuclei of brainstem remains intact. SCI may have resting airway tone for this reason & also hypersecretions.

- Unopposed parasympathetic activation of the airways has been demonstrated to increase bronchial reactivity and the effect may be reversed with ipratropium bromide, an anticholinergic agent.
  - > Ipratropium bromide pre-treatment abolished airway reactivity following methacholine challenge

- Short term β agonist administration in acute tetraplegia may confer some benefit but no clear evidence of longer-term benefit from bronchodilation in cervical SCI

- The effects of other medications commonly used in the management of SCI, such as baclofen and oxybutynin, should be considered when reviewing airway hyperreactivity in people with tetraplegia.

<table>
<thead>
<tr>
<th>Table 2: Response to Ipratropium bromide in spinal cord injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary Function</td>
</tr>
<tr>
<td>--------------------</td>
</tr>
<tr>
<td>Parameter</td>
</tr>
<tr>
<td>FVC(L)</td>
</tr>
<tr>
<td>FEF(50%)</td>
</tr>
<tr>
<td>FEF(25%)</td>
</tr>
<tr>
<td>FEF(12.5%)</td>
</tr>
<tr>
<td>FEV1/FVC ratio</td>
</tr>
</tbody>
</table>

Values are mean ± SD for postbronchodilator measurements. Numbers in parentheses represent percent change after administration of ipratropium bromide.

Positioning Matters!

• Patients do report less breathlessness in supine compared to sitting during the acute stage after high SCI

• Positioning should be considered when a patient is experiencing breathlessness or weaning from ventilation as the use of supine positioning may seem counterintuitive based on experience in other patient groups

(Adapted from Arora et al. 2012)

• Chen et al. (1990) recorded a 14% ↓ in predicted VC & Linn et al. (2000) showed a statistically significant ↓ in FVC in the tetraplegic person on changing from supine to sitting or standing.

• Conversely, VC of the tetraplegic person rises by 6% when the bed is tipped 15° head down from supine (Cameron et al. 1955).

Aaargh “sit them up to ease their breathing” NO!

Sleep

• OSA is a big deal in SCI!
• OSA appears as a direct consequence of CxSCI > up to 83% prevalent in the 1st year.
• OSA has a prevalence of 40–91% in chronic SCI
• Prevalence in paraplegia similar to able-bodied population (however, the literature is far less comprehensive in this group)
• Neurocognitive impairments including decreased memory & attention have been linked to nocturnal hypoxia in tetraplegic subjects with untreated OSA
• In chronic SCI, most authors have observed associations between increasing age, body mass index (BMI) & neck circumference and OSA prevalence, but these relationships appear weaker acutely.
• OSA in tetraplegia is a biphasic disorder, acutely caused by the cervical SCI that partially resolves only to increase in prevalence again as people with SCI age and gain weight
• An individual with acute tetraplegia and undiagnosed or untreated OSA may struggle to participate in the demanding process of rehabilitation
Breath/Air stacking
Using a modified resus bag to augment patients own ability to inspire deeply.
- Volume recruitment
- Thoracic stretch
- Chest clearance

More air in = more air out!
Assisted cough, trunk flexion = clearance

(Jeong & Yoo 2015, Torres-Castro et al. 2014)
Lung Vol Recruitment Kits – RAPP Australia 03 5284 0208

Respiratory Muscle Training
- Respiratory muscle training in tetraplegia increases
  - vital capacity (slow and combined),
  - inspiratory capacity,
  - inspiratory pressure (muscle strength),
  - maximal ventilatory capacity and
  - expiratory pressures
- All increases are small in magnitude (e.g. 410 ml in VC), but the benefit is consistent
- Similar for all NMD single studies (Cheah 2009, Gozal 1999, etc)

RMT-Conclusions
- No evidence of carry-over after training ceases
- RMT shows a small significant benefit
  - Unable to determine which type of RMT is "best" or what is the dose-response
  - Sensitivity analyses show that RMT is effective in acute tetraplegia, but no MEP benefit acutely
  - almost certainly not a bad idea ....
- Pulmonary mechanics measures necessary to apportion contribution of static recoil versus skeletal muscle strength
The Feasibility of Using Mouthpiece Ventilation Post Extubation for Acute Tetraplegia in the Intensive Care Unit.

Brooke Wadsworth, Peter Kruger, Chris Joyce, Craig Hukins, Gabrielle Ferguson, Jenny Paratz, Tim Geraghty, Duncan Brown, Brooke Duggan, Michele Foster, Adam Semciw

HREC/16/QPAH/688 – SSA/16/QPAH/689

Objective

- To determine the feasibility and effectiveness of the novel use of mouthpiece ventilation (MPV) post extubation in the intensive care unit (ICU) in acute tetraplegia.

The secondary aims of this study are to:

- determine the nature and the amount of assistance required to adequately implement MPV,
- determine if any baseline characteristics are likely to be predictive of ability to use MPV successfully,
- and to obtain preliminary data to inform a power analysis for the design of a definitive study to examine whether MPV has an impact on incidence of reintubation and ICU length of stay (LOS).
So...

- Of the 14 participants, 3 failed extubation.
- 5 considered to have pneumonia at time of extubation – 2 of these failed extubation.
- One extubation failure required a tracheostomy (multi chest trauma)
- Two extubation failures went on to use MPV at second extubation & remained extubated.
- We are getting better! Finesse with initial inspiratory Ti, rise Ti, IPAP.

+ve of MPV

- ↓ chance of skin breakdown
- Better ability to eat & drink
- Better speech than with oro-nasal & nasal mask
- Improved security compared to tracheostomy (GPB)
- Better appearance
- Ability to set pressure or volume mode
- Breath stack- variable breath support depending on demands in AC mode
- Less claustrophobia

-ve MPV

- Difficulty using at night/sleep – need for lip seal
- Air leaks from the mouth or nose
- Gastric distension
- Can’t be used if unable to co-operate or suffers bulbar dysfunction

Mouth intermittent Positive Pressure Ventilation in the Management of Postpolio Respiratory Insufficiency*

John E. Bach, M.D.; Augusta S. Allen, M.D.; George Belknap, M.D.;
Lori Saporito, R.T., and Mathew Leo, M.D.

CHEST  /  81 / 6 / JUNE, 1982  659

Change in type & amount of respiratory assistance in 75 postpolio MPV users from 1950 - publication date. Descriptive educational

Intermittent Positive Pressure Ventilation via the Mouth as an Alternative to Tracheostomy for 257 Ventilator Users*

John E. Bach, M.D.; F.C.C.P.; Augusta S. Allen, M.D.; and
Lori L. Saporito, B.A., B.R.R.T.

(Chest 2003, 123:76-82)

Single centre experience from prior 39 years with descriptive use for weaning, day/night time interface, tracheal incidence for neurological conditions.
Phrenic nerves & diaphragm

**Phrenic nerve pacing**

Electrodes wrapped around the phrenic nerve (one on each side) via a thoracotomy (or potentially a minimally invasive technique).

Electrodes have wires attached to them that pass to a receiving unit that is implanted in the patient’s abdomen (usually) or possibly higher on the chest wall. In order to send a signal to the receiver, a flexible antenna wire is taped to the patient’s skin (over the area of the receiver). The antenna wire is in turn attached to a transmitting device which is a fairly large square black box with some dials or it to adjust things like stimulus intensity and rate etc.

**Direct diaphragm pacing**

Electrodes placed (laparoscopically) into the undersurface of the diaphragm and then bringing the 4 wires out of the skin on the upper chest wall or upper abdomen and attaching them to a plastic “block”. A lead is then attached to the wires that are captured in the block and the lead is finally plugged into a pacing box or transmitting unit.

Pacing - Why?

The whole point of pacing the diaphragm is to make positioning, seating, transfers and community activities more accessible for ventilator dependent quadriplegics so the systems are designed to be portable, unobtrusive and easy to position.

No particular contraindications or precautions other than making sure that the connections to the transmitting (or stimulating) units are kept intact and that satisfactory ventilation is maintained.