Mechanical Insufflation-Exsufflation (MI-E) vs High-Frequency Chest Wall Oscillation (HFCWO)

Rationale, Technology and Clinical Use

Introduction

The following annotated bibliography was authored by Jane Braverman, PhD and supported by Hill-Rom.

Inherited or acquired neuromuscular diseases (NMD) affect millions of Americans at some time during their lives. Excluding peripheral neuropathies, the estimated prevalence of the most common NMDs exceeds 400,000 [1]. Scores of disorders affecting motor neurons and peripheral nerves have been identified [2,3]. NMDs are characterized by progressive atrophy and weakness of skeletal muscle, skeletal-spinal deformities, limb contractures, and restrictive lung disease leading to poor respiratory function [2-6]. Among those with significant systemic involvement, including diaphragmatic and/or intercostal muscle weakness and progressive chest deformities, acute respiratory tract infections and chronic respiratory insufficiency are the chief cause of morbidity and mortality [4-7]. Weak cough and consequent retention of airway secretions is the principle contributing factor [2-7]. An effective cough is necessary for airway clearance and protection against foreign matter including respiratory pathogens [2-7]. A variety of secretion mobilizing and cough augmentation modalities are part of standard protocols to enhance secretion clearance in cough-deficient NMD patients [7-10]. When used appropriately, such techniques have been shown to be both safe and effective [8,9]. This review is focused upon a discussion of the rationale underlying use of two of these techniques, mechanical insufflation-exsufflation (MI-E) and high-frequency chest wall oscillation (HFCWO). It includes, in a question and response format, a comparison of each modality in terms of intended use, mechanisms of action, patient selection criteria.

I. Rationale for secretion mobilization and cough assistance in NMD

Healthy respiratory function requires open airways to permit efficient gas exchange and effective mechanisms to keep airways free of accumulated secretions and harmful inhaled particulate matter. These mechanisms, part of a complex pulmonary defense system, include mucociliary clearance (MCC) and cough. In simplified terms, MCC involves coordinated interaction between airway mucous and tiny hair-like structures called cilia that work to entrap bacteria and other particulate matter and sweep secretions upwards and outwards from smaller to larger airways until they reach an accumulation point in the central bronchus. Cough is essential to complete MCC [8,10]. When sufficient mucous is accumulated in the central bronchus, a cough reflex is triggered, permitting final clearance by expectoration or swallowing. Abnormal secretion accumulation in the bronchial tree is caused by a complex array of pathologies that occur in the presence of restrictive and/or obstructive lung disease. Secretion mobilization and cough augmentation interventions are implemented to compensate for deficits in MCC and/or cough mechanisms to achieve healthy clearance [3, 8-10].

II. NMDs and secretion clearance dysfunction

NMDs weaken respiratory muscle strength and distort thoracic skeletal structures in varying patterns and degrees. As a result, patients with these disorders may develop restrictive pulmonary disease, ineffective cough, atelectasis and chronic respiratory insufficiency. Ineffective cough limits ability to clear secretions from the tracheobronchial tree. Poor secretion clearance affects normal mucous function, disrupting certain physical, biological, and chemical components of the pulmonary defense system. Retention of increasingly large volumes of pulmonary secretions leads to consequences including airway obstruction, increased work of breathing (WOB), hypoxia, frequent episodes of pneumonia and, ultimately, irreversible lung damage and respiratory failure [3-7]. The majority of episodes of respiratory failure in patients with severe NMD are a result of ineffective cough during acute respiratory infection [4-7].

Q. How do NMDs lead to impaired secretion clearance?

A. In NMD, factors contributing to impaired airway clearance include:

- **Restrictive lung disease:** Restrictive lung disease is characterized by static or diminished lung volumes and vital capacities. In NMD, the respiratory muscles may be weak and the spine and thorax deformed, reducing ability to take a deep breath, to generate expiratory force, and to cough effectively [3-10].

- **Ineffective cough:** In NMD, diminished inspiratory capacity as a result of diaphragm weakness or thoracic deformity, poor coordination of the bulbar muscle impairing closure of the glottis and ability to build up intrapleural pressure and poor coordination of the expiratory muscles diminishes expulsive force and mucous shearing, resulting in secretion retention [3-10].

- **Dysphagia/aspiration/gastroesophageal reflux:** Dysphagia, or difficulty in swallowing, is a consequence of anatomical abnormalities or weakness of the muscles associated with swallowing. Gastro-esophageal reflux occurs when a defective lower esophageal sphincter allows stomach contents to surge into the esophagus. Affected NMD patients are at risk for aspiration of secretions, vomitus or foreign bodies into the lungs. Serious, sometimes fatal pneumonia may result [5,7].

- **Immobility:** Patients with severe NMD mobility limitations cannot exercise as necessary to maintain aerobic capacity, bellows function, and lung volume necessary for effective cough [1].

Q. What is an effective cough?

A. Cough consists of three phases: 1) an inspiratory gasp, 2) a compressive phase and 3) an expulsive phase. An effective cough requires ability to inspire up to 85-90% of total lung capacity, intact bulbar muscle function to accomplish rapid closure of the glottis for approximately 0.2 seconds, and subsequent contraction of abdominal and intercostal (expiratory) muscles sufficient to generate intrapleural pressures of > 190 cm H_2O. When the glottis opens, an explosive decompression generates a transient peak cough expiratory flow (PCEF). In normal adults, expiratory volumes range from 360-720L/min or more [3-10]. PCEF below 162 L/min considered ineffective. [1,7,8].

Q. How is cough effectiveness measured?

A. Cough effectiveness is assessed in the clinical setting using a measure called Peak Cough Expiratory Flow (PCEF). This involves measurement of expiratory flow during a cough using a peak flow meter device called a pneumotachometer. Patients are asked to inspire to total lung capacity and then forcibly expire, either through a mouthpiece or facemask attached to the device [8,9].

III. MI-E machines and HFCWO devices perform different functions, operate on different principles and involve different mechanisms of action.

Q. What is the theoretical principle supporting MI-E therapy?

A. The application of positive pressure to the airway, followed by an abrupt transition to negative pressure, will produce a high expiratory flow from the lungs, thus increasing PCEF sufficient to stimulate a secretion-clearing cough effect [5,7,9,13].

Q. What is the theoretical principle supporting HFCWO therapy?

A. Oscillation of the chest wall creates an expiratory airflow bias that moves secretions from peripheral airways towards the central bronchus and trachea. Oscillations also result in transient increases in airflow at low lung volumes, thus improving gas-liquid interactions to decrease sputum viscosity and accelerate secretion mobilization [12].

Q. What is the function of MI-E in respiratory care?

A. MI-E (cough assist) devices are designed to augment cough in patients with cough function insufficient to clear bronchial secretions from their central airways. Candidates for MI-E devices are typically those with NMDs involving respiratory and/or bulbar muscle dysfunction. In many of these patients, MCC mechanisms are intact and secretions advance normally from peripheral lung regions to the central bronchus. However, because they lack effective cough function, secretions accumulate, resulting in airway obstruction and associated pathological consequences. NMDs characterized by progressive loss of cough function include but are not limited to Duchenne (DMD) and certain other muscular dystrophies, amyotrophic lateral sclerosis (ALS), spinal muscular atrophies, types I and II (SMAI and SMA II) and spinal cord injury. *MI-E IS NOT a substitute for HFCWO in patients with significant MCC dysfunction [5,6,13].*

Q. What is the function of HFCWO in respiratory care?

A. HFCWO devices are designed to mobilize excess or retained bronchial secretions from smaller to larger airways to the central bronchus. At that collection point, final clearance is typically accomplished by cough or suctioning. Candidates for HFCWO are those in whom secretion clearance is compromised by primary or acquired defects in the MCC component of the pulmonary defense system. In such patients, secretions accumulate in and obstruct peripheral lung regions and smaller bronchi because ciliary activity is absent or ineffective and/or impeded by secretions that are excessive and/or abnormally thick, thin or sticky. *HFCWO IS NOT a substitute for cough assistance therapy in cough-deficient patients [5,8,9,13].*

Q. How do MI-E devices work?

A. An MI-E therapy cycle consists of delivery of inspiratory pressure, expiratory pressure and pause. Therapy typically involves the use of preset insufflation pressure (of +35 to + 45 cm H_2O), followed by a rapid shift to preset negative exsufflation pressure (- 35 to - 45 cm H_2O) and then a pause before the next MI-E cycle. These maneuvers generate peak and sustained cough flows sufficient to move secretions from central airways towards the mouth for clearance by expectoration or suctioning. MI-E may be administered noninvasively using either a mouthpiece or a facemask or may be connected directly to a tracheostomy tube. *MI-E is effective in the six central airway divisions not clearable by MCC [11].*

Q. How do HFCWO devices work?

A. HFCWO devices typically consist of a garment and a connecting hose or hoses that attach to an air pulse generator. The air pulse generator creates oscillating air pressures that are delivered to the garment via the interconnecting hose/s. The rapid inflation and deflation of the garment against the user's chest creates oscillatory airflow within the peripheral airways that help to loosen, thin, and mobilize bronchial secretions from smaller to larger airways for clearance by swallowing, coughing or suctioning [12]. *HFCWO cannot clear secretions from the six central airway divisions without the support of natural or assisted cough or suctioning [11].*

IV. Patient selection criteria: Which patients are appropriate candidates for MI-E, HFCWO or a combination of both therapies?

Q. Which categories of patients can benefit from MI-E alone?

A. A significant proportion of NMD patients, including those with hereditary disease and acquired neuromotor dysfunction, suffer progressive loss of cough function and resulting high risk for respiratory failure from complications of secretion retention. In such patients effective cough is necessary for clearance of airway secretions during both when stable and ill with respiratory infections. Therapeutic goals are to ease work of breathing and prevent atelectasis, pneumonia and progression to respiratory failure. Cough is considered ineffective when PECF is < 270 L-min-1 [ref]. These patients may be candidates for MI-E. For patients with PECFs < 160 L-min-1 [8]. Other candidates for MI-E include those who have failed MV weaning attempts owing to mucous-obstructed central airways [8,9,13].

Q. Which categories of patients can benefit from HFCWO alone?

A. *HFCWO meets airway clearance needs in patients with absent or impaired MCC function but with normal cough function, or those for whom tracheal suctioning already is part of routine care.* HFCWO is a standard of care for patients with obstructive lung diseases who suffer from pulmonary secretion retention. This includes but is not limited to cystic fibrosis (CF), immotile cilia syndromes (ICS) and bronchiectasis arising from a broad range of underlying causes [10,11]. It also includes certain patients with neuromuscular disorders who are unable to mobilize secretions from peripheral to central lung regions [10,11].

Q. Are there patients for whom combined MI-E and HFCWO therapy is appropriate?

A. *MI-E and HFCWO may be appropriate complimentary therapies for patients with both impaired cough and MCC mechanisms.* Such patients include those with NMD and who have gone on to develop significant secretion-related disease, including those that have significant secretion retention and/or have a history of recurrent respiratory infection. In severe cases, these patients develop lung damage secondary to absent or inadequate secretion clearance and/or cough clearance. These patients suffer from persistent atelectasis, hypoxia, mucous impaction or plugging, an accelerating cycle of recurrent respiratory illnesses and other manifestations of chronic lung disease. Patients meeting these criteria may require HFCWO to accomplish MCC and MI-E for cough clearance [3,5,10,13].

Notes

1. McDonald CM. Physical activity, health impairments, and disability in neuromuscular disease. *Am J Phys Med Rehabil* 2002; 81 (Suppl): S 108–120.
2. Gozal D. Pulmonary manifestations of neuromuscular disease with special reference to Duchenne muscular dystrophy. *Pediatr Pulmonol* 2000; 29: 141–150.
3. Benditt JO, Boitano LJ. Pulmonary issues in patients with chronic neuromuscular disease. *Am J Resp Crit Care Med* 2013; 187 (10): 1046–1055.
4. Panitch HB, The pathophysiology of respiratory impairment in pediatric neuromuscular disease. *Pediatrics* 2009; 124: Suppl 4: S215–218.
5. Bach JR. Mechanical insufflation/exsufflation: has it come of age? A commentary. *Resp J* 2003; 21(3): 385–386.
6. Perrin C, Uterborn JN, Ambrosio CD, Hill NS. Pulmonary complications of chronic and neuromuscular diseases and their management. *Muscle Nerve* 2004; 29: 5–27.
7. Chatwin M. Mechanical aids for secretion clearance. *Internat J Resp Care* 2009 (Autumn/Winter): 50–53.
8. Homnick DN Mechanical insufflation-exsufflation for airway mucus clearance. *Resp Care* 2007; 52 (10): 1296–1305.
9. Boitano LJ. Management of airway clearance in neuromuscular disease. *Resp Care* 2006; 51 (8): 913–922.
10. Finder JD Airway clearance modalities in neuromuscular disease. *Paediatr Resp Rev* 2010; 11 91): 31–34
11. Bach JR, Barrow SE, Goncalves M, et al. A historical perspective on expiratory muscle aids and their impact on home care. *Am J Phys Med Rehabil* 2013; 92 (10): 930–941.
12. Hansen L, Warwick W. High-frequency chest compression system to aid in clearance of mucus from the lung. *Biomed Instrum Technol* 1990; 24:289–294.
13. Wolfe LF, Joyce NC, McDonald CM, Benditt JO, Finder J. Management of pulmonary complications in neuromuscular disease. -2012- *Phys Med Rehabil Clin N Am.* 829–853.

ORDER NUMBER 187604 rev1 19-JUNE-2014 ENG – US

For further information about this product or a service, please contact your local Hill-Rom representative or visit our webpage:

800.426.4224 option 3

www.vitalcough.com

Enhancing outcomes for patients and their caregivers: Hill-Rom