

EDITORIAL

Mechanical insufflation/exsufflation: has it come of age? A commentary

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In 1852, the English physician MERYON [1] described how a 16-yr-old male with what was to become known as Duchenne muscular dystrophy, died from acute respiratory failure during a febrile episode with "profuse secretion of mucus from the trachea and larynx". One-hundred and fifty years later, over 90% of episodes of respiratory failure in patients with muscular dystrophy are caused by ineffective coughing during intercurrent chest colds [2]. Most people with neuromuscular diseases (NMDs) still die prematurely or are hospitalised and undergo tracheotomy, because of failure to assist respiratory muscles to prevent respiratory failure. The study by CHATWIN *et al.* [3] of cough augmentation with mechanical in-exsufflation (MI-E) is an important step towards ending this perennial trend.

Over 700 patients with NMDs, postpoliomyelitis, high-level spinal cord injury, and other conditions with respiratory muscle weakness or paralysis have used noninvasive positive pressure ventilation delivered *via* mouthpieces [4] and nasal interfaces [5] for continuous ventilatory support, in many cases, for over 40 years [2, 5–9]. However, it would have been impossible for these patients to have avoided tracheotomy had they been unable to effectively clear airway secretions during intercurrent chest colds. Whereas the mucociliary elevator is generally intact in patients with NMD unless they undergo tracheotomy, the cough is not. LEITH [10] reported that a normal cough requires that the vocal folds enlarge airway diameter and the inspiratory muscles inspire up to ~85–90% of total lung capacity. There is then a rapid and firm closure of the glottis for ~0.2 s. Both glottic opening and closure require intrinsic laryngeal (bulbar) muscle contractions. Subsequent contraction of abdominal and intercostal (expiratory) muscles results in intrapleural pressures of ≤ 18.62 kPa (140 mmHg). Upon glottic opening, this causes an explosive decompression that generates transient cough peak flows (CPF) of $360\text{--}1,200$ L·m⁻¹. The expiratory flow is facilitated by active gradual abduction of the vocal cords [10]. Thus, inspiratory, expiratory, and bulbar muscle function is required for effective CPF.

Total expiratory volume during normal coughing is 2.3 ± 0.5 L [10]. Irrespective of expiratory muscle function, CPF are decreased for patients whose inspiratory muscles cannot generate tidal volumes $>1,500$ mL [11]. Thus, to optimise CPF for patients with low vital capacities, coughing needs to be preceded by the delivery of deep insufflations or effective air stacking. To air stack, the patient takes a deep breath and then receives consecutively delivered volumes of air from a volume-cycled ventilator or a manual resuscitator. The patient stacks the volumes under a closed glottis until the lungs are maximally expanded. If the lips or cheeks are too weak to

permit air stacking, stacking is done *via* a nasal interface or lip seal. The ability to air stack and the extent that the maximum insufflation capacity exceeds the vital capacity is a measure of glottic and bulbar competence [12]. Thus, patients hold large volumes of air under pressures of 40–70 cmH₂O and perform this routinely, three times a day, in many cases, for ≥ 40 yrs. No barotrauma or complications have ever been reported from air stacking. Patient's who cannot air stack to optimal lung recoil volumes because of bulbar muscle dysfunction need to be provided with single, deep insufflations *via* an oronasal interface for the inspiratory component of an effective cough. Many patients use mechanical insufflation at 40–60 cmH₂O for this.

When the intercostal and abdominal muscles are too weak to generate sufficient thoracoabdominal pressures, their function can be assisted by some combination of a tussive squeeze and an abdominal thrust as well as by the application of negative pressure exsufflation (mechanical assistance) to the airway upon glottic opening. A manually assisted cough is the coughing out of a maximally air stacked volume of air with the aid of an abdominal thrust timed to glottic opening. The resulting cough flows are the (manually) assisted CPF. Manually and mechanically assisted coughing can be combined to produce effective CPF. In one study, all 43 postextubation patients with CPF (unassisted or assisted) >160 L·m⁻¹ remained successfully extubated, whereas all 15 with CPF under this level had to be re-intubated within 48 h [13]. Thus, it seems that ≥ 160 L·m⁻¹ of CPF are needed to clear airway secretions from central airways.

Given that noninvasive ventilation and assisted coughing can be effective even in the absence of any inspiratory or expiratory muscle function [4, 7, 9], when, then, does bulbar dysfunction become a limiting factor for noninvasive approaches and when is MI-E indicated? With the exception of children, and patients with bulbar amyotrophic lateral sclerosis (ALS), assisted CPF can almost always be increased to >160 L·m⁻¹, and therefore, be at least minimally effective for eliminating airway secretions. For ALS patients, however, glottic closure and therefore, air stacking can be lost and assisted CPF become unmeasurable. Once glottic function is severely diminished, patients must receive all nutrition *via* gastrostomy tubes and chest colds, and eventually, aspiration of saliva results in pneumonia and respiratory failure. Once aspiration of saliva reaches the point of causing persistent oxyhaemoglobin desaturation, acute respiratory failure is only days to weeks away unless the patient undergoes tracheotomy. With loss of laryngeal muscle function and glottic control, the upper airway collapses during expiration and eventually during inspiration. Airway collapse can render MI-E, even with a timed abdominal thrust, totally ineffective [14], and respiratory failure can then only be prevented by tracheotomy.

At the opposite extreme, MI-E is usually unnecessary when inspiratory and expiratory muscles are completely paralysed but bulbar muscles are completely intact as for many high-level spinal-cord injured patients. In this case, manually

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assisted coughing can often result in extremely effective CPF, often $>500 \text{ L}\cdot\text{m}^{-1}$, because of intact air stacking ability and effective lung recoil. Therefore, MI-E is indicated when inspiratory, expiratory and bulbar muscles are dysfunctional but the latter are not completely paralysed and glottic closure is still possible. This is the case for most patients with NMDs.

The study by CHATWIN *et al.* [3] demonstrates that MI-E can increase CPF from marginal levels of $169 \text{ L}\cdot\text{m}^{-1}$ (unassisted) to the much more effective $235 \text{ L}\cdot\text{m}^{-1}$. It is because of this that such patients can survive without resorting to tracheotomy. However, the investigators achieved these flows without providing concomitant abdominal thrusts and suboptimal pressures were used. Although the investigators used pressures that were "comfortable", this is irrelevant for efficacy during respiratory tract infections when airways actually need to be cleared. Mask pressures of $15 \text{ cmH}_2\text{O}$ are found to be almost completely ineffective and are never used clinically.

Comparing cough flows with airway secretion management by chest therapy techniques is like comparing apples and oranges. Patients with irreversible airway obstruction, such as occurs in chronic obstructive pulmonary disease or tracheal stenosis, may not be able to generate adequate CPF with or without the Cough-AssistTM (J.H. Emerson Company, Cambridge, MA, USA) but may benefit from airway vibration. However, chest vibration techniques, no matter how effective in mobilising peripheral secretions, will not result in their expulsion without an effective cough flow. All too often this can only be achieved by manually or mechanically assisted coughing. Thus, MI-E is not an alternative approach to facilitating secretion clearance but an often vital method for expelling secretions from central airways for patients with weak respiratory muscles but some bulbar muscle function. While it will not be effective when bulbar muscles are completely paralysed, it is not appropriate to exclude patients with significant bulbar dysfunction. If bulbar function is intact, MI-E is not needed because CPF can be greatly increased by abdominal thrusts following air stacking.

It has been demonstrated that mechanical insufflation/exsufflation is critical for avoiding hospitalisations, pneumonias, episodes of respiratory failure, and tracheotomy for patients with Duchenne muscular dystrophy [7], spinal muscular atrophy [8], and nonbulbar amyotrophic lateral sclerosis [9]. Many neuromuscular disease patients can become continuously dependent on noninvasive ventilation for years without ever being hospitalised by using oximetry for feedback to maintain effective alveolar ventilation and using mechanical insufflation/exsufflation during intercurrent chest colds [7–9]. While this approach to management is routine in several centres, clinicians who do not understand how patients can be 24-h ventilator dependent without tubes, without ventilator-free breathing ability, and with the need to increase cough flows to prevent respiratory failure, sometimes demand double-blinded controlled studies [15]. The authors anticipate a long wait before such studies can be ethically designed. In

the meantime, it may be more rational to quantitate what should already be intuitively obvious, which is that the higher the cough flow, the more effective the cough and the less likely that airway secretion accumulation will result in respiratory failure.

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