



Glottic patency during noninvasive ventilation in patients with chronic obstructive pulmonary disease



We read with interest the study by Eline Oppersma et al. (2018) on the patency of the glottis in eight patients with acute exacerbation of chronic obstructive pulmonary disease (COPD) requiring non-invasive ventilation (NIV). In their introduction, the authors suggest that neurally adjusted ventilator assist (NAVA) may limit the decreased patency of the upper airways observed with other modes of non-invasive positive pressure ventilation (in animals and humans) which may be accountable in part for some NIV failures.

No differences were found both in glottis angles during inspiration and at peak inspiratory effort for all subjects between PSV low and high and NAVA low and high. Moreover, no closure of the glottis were observed during NIV. According to these results, the authors concluded that the glottis of human adults with acute exacerbation of COPD is not influenced by increasing inspiratory pressure levels during NIV.

These conclusions may be criticised. The patency of the upper airways, and especially the glottic aperture, represents a part of an integrated central mechanism coordinating the activity of all dilating upper airways muscles and inspiratory pump muscles. We have previously shown in normal awake subjects that the glottic aperture was significantly greater in spontaneous ventilation (range 35–55°) than during noninvasive positive pressure ventilation (range 0–40°) and that reappearance of a diaphragmatic EMG activity after periods of diaphragmatic silence under NIV was systematically accompanied by glottis widening (Jounieaux et al., 1995a, 1995b). These observations led us to conclude that, in absence of respiratory muscle activity, the glottic aperture represents the main factor regulating ventilation during NIV in awake humans. During NAVA mode, because the level of inspiratory support and the cycling of the ventilator are proportional to the electrical activity of the diaphragm (Sun et al., 2017), the glottis could not play any role in the airflow regulation. Indeed, Oppersma et al. (2018) found results similar to ours with vocal cords angles ranging from 35° to 55° when diaphragmatic muscle is active. Thus, the glottic behaviour strongly depends on absence or presence of a diaphragmatic muscle activity, explaining the differences observed in the previous animal models: absence of glottic closure with NAVA (Hadj-Ahmed et al., 2012) and glottic closure with PSV when no diaphragmatic muscle activity is observed (Moreau-Bussière et al., 2007).

When NIV provides all the ventilatory needs (i.e. no spontaneous respiratory efforts and a silent diaphragmatic EMG), we have also shown that the glottic aperture decreases through multiple influences:

vigilance state, sleep stages, delivered minute ventilation, CO₂ levels and mechanical factors including inspiratory flow (Jounieaux et al., 1995a, 1995b and Parreira et al., 1997). Because all these parameters were not recorded in their study, Oppersma et al. (2018) could not draw a general conclusion on the upper airways behaviour in COPD patients with acute exacerbation during NIV.

From a practical point of view, more studies are required to explain the 5–40% failure of NIV observed in such patients. If NIV is used to assist patients (persistence of a spontaneous diaphragmatic activity), failure may be related to insufficient mechanical ventilation and not to glottic interactions whereas if NIV is used to rest diaphragm (no more spontaneous diaphragmatic activity), failure may be related to glottic closure. The upper airways affair during NIV is far from being closed.

References

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