Noninvasive Positive and Negative Pressure Effects in Upper Airway Respiratory Failure

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Introduction

Noninvasive ventilation (NIV) is becoming more common in patients with acute respiratory failure, such as exacerbations of chronic obstructive pulmonary disease or acute heart failure [1]. One of the main goals of NIV is to prevent endotracheal intubation, which reduces the risks of invasive ventilation [2]. NIV failure rates range between 5% and 50%, with the majority of these patients requiring endotracheal intubation.

The endotracheal tube bypasses the upper airway during invasive ventilation, and the cuff of the endotracheal tube creates an airtight seal in the trachea. On the other hand, the upper airway may affect how ventilation works during NIV [1].

Upper Airway

The nose, oral cavity, pharynx, and larynx make up the upper airway. The upper airway is used for chewing, swallowing, speaking, and smelling, and its principal roles are to act as an air conductor, humidify and warm the inspired air, and keep foreign materials out of the tracheobronchial tree [3]. The nose and oral cavity are

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mostly static in their conducting role, but the pharynx and larynx are mostly muscular organs that can change the upper airway's patency [4].

During inspiration, glottic constriction increases upper airway resistance, which may impede effective breathing.

The presence of negative pressure in the upper airway and flow during inspiration results in a phasic respiratory activity of the posterior cricoarytenoid muscle above tonic levels, which results in glottic widening during inspiration and decreases resistance to airflow in isolated piglet upper airways. The inspiratory muscles are successfully unloaded as a result of this response. During expiration, positive pressure and flow cause phasic activation of the thyroarytenoid muscle, which causes glottic constriction and hence higher resistance to the expiratory flow. As a result, at least in an animal model, respiratory flow patterns influence upper airway muscle activity [5].

The upper airway is a collapsible tube vulnerable to closure during breathing because this region of the breathing apparatus is surrounded by a complex anatomical arrangement of skeletal muscles and soft tissues, unlike other regions of the respiratory tract such as the trachea and bronchi that are supported by a more rigid cartilaginous structure. The muscular and soft tissue composition of the pharyngeal airway provides the necessary support for a variety of essential non-respiratory functions such as vocalization, suckling, chewing, and swallowing, i.e., behaviors that require dynamic changes in airway size to move air, liquids, and solids. However, this property of a collapsible tube compromises the essential respiratory function of the upper airway; the airway must remain open during breathing, in all postures, to allow for adequate lung ventilation and gas exchange [5].

From a mechanical standpoint, the upper airway behaves as a Starling resistor, in which the pharyngeal airway represents the collapsible segment and is situated between two noncollapsible structures (larynx and nasopharynx). The flow pattern depends on the forces applied inside and outside the collapsible segment. The transmural pressure gradient is the net pressure difference between all of these opposite forces. The collapsing forces are represented by the negative inspiratory transmural pressure gradient and the pressure applied by upper airway tissue. The contraction of upper airway stabilizing muscles (upper airway dilators) is the main dilating force, the other being represented by tracheal traction. So, the amount and timing of the neuromuscular activation process of the muscles that help keep the upper airway stable, as well as the mechanical properties of the tissues in the upper airway, play a big role in deciding how stable the upper airway is [6].

Apart from the influence of the extent of phasic activation of upper airway muscles, the dynamic profile of this phasic activity plays a key role in the maintenance of upper airway patency. The upper airway muscles are activated first and reach their peak value before the respiratory muscles. Phasic activity and the preactivation delay increase with increasing central respiratory activity and with decreasing upper airway pressure. This activation pattern decreases upper airway resistance and prevents upper airway inspiratory collapse. The occurrence of upper airway obstruction in normal awake subjects when this preactivation of upper airway stabilizing muscles is lost (as with diaphragmatic pacing, phrenic nerve stimulation, or iron

lung ventilation) further supports the importance of the upper airway muscle preactivation pattern in maintaining upper airway patency. The link that exists between ventilatory and upper airway stability could result from the common activation process of respiratory and upper airway stabilizing muscles originating from the central pattern generator that would be responsible for the fine-tuning in the amplitude and activation pattern of these different muscle groups [6].

The Upper Airway's Interaction with Noninvasive Ventilation

In awake lambs, the influence of NIV on glottal constrictor (thyroarytenoid) and dilator (cricothyroid) muscle activity was studied. Both the thyroarytenoid and cricothyroid muscles are active during spontaneous breathing, with the thyroarytenoid muscle activity peaking at the end of inspiration. However, when pressure support is used during NIV, inspiratory cricothyroid activity decreases, but thyroarytenoid muscle activity increases. As evidenced by respiratory inductance plethysmography [7], this causes glottal constriction and limited breathing.

A subsequent study demonstrated that increased glottal constrictor muscle activity during NIV depends mainly on the activation of bronchopulmonary receptors. After a bilateral vagotomy, the increase in inspiratory activity of the thyroarytenoid muscle that was seen before when the support got stronger during NIV did not happen [8].

In humans, there is limited evidence of a similar response to NIV. Rodenstein and colleagues subjected healthy volunteers to escalating amounts of NIV assistance while monitoring their glottis with a fiber-optic bronchoscope. The narrower the glottic aperture and the greater the airway resistance, the higher the amount of support. This effect resulted in a gradual decline in the percentage of tidal volume reaching the lungs, which was attributed at least in part to the glottis' behavior [9]. In conclusion, research on both animals and people shows that positive pressure breathing makes the upper airways less open when trying to breathe in.

Maintaining Airway Patency in the Upper Airway

The upper airway collapse in patients with obstructive sleep apnea is a typical indication of NIV (either with PEEP or as simple CPAP). According to the fundamental physiologic concept, positive upper airway pressure splints open the collapsed upper airway structures during sleep. Even though adding inspiratory positive pressure can help some people, CPAP on its own is often enough for most.

Positive pressure and negative pressure techniques are used to provide noninvasive ventilation. Positive pressure is put on the airway to directly fill the lungs with air, and negative pressure is put on the abdomen and thorax from the outside to pull air into the lungs through the upper airway.

By avoiding intubation, the risk of upper airway trauma, patient discomfort, and the need for sedation are all reduced. It also keeps the airway clear and helps the

patient swallow; it also allows oral patency and intermittent ventilation, which lets the patient eat, drink, and talk normally. It also lets the patient take breaks from ventilation, which lets them eat, drink, and talk normally [10].

Monitoring Muscles of the Upper Airway

During mechanical ventilation, the need for monitoring inspiratory muscle activity has been emphasized in the literature [11]. Less is known about the role of upper airway activity monitoring during NIV. This is likely because it is hard to see how these patients' upper airways work.

Activation of intrinsic laryngeal muscles alters glottis opening, which affects flow resistance into and out of the lungs. Because the phasic activity of upper airway dilator muscles goes up when breathing is restricted, like in patient-ventilator asynchrony, it could be clinically important to watch how upper airway dilator muscles work when breathing in [12].

MR imaging with tagging of the upper airway in healthy patients revealed that not only the genioglossus muscle but also nonmuscular soft tissues surrounding the upper airway move before the commencement of inspiratory flow. The movement of particular reference sites on the genioglossus muscle was greater during normal inspiration than during loaded inspiration, suggesting that the increased muscle activity during loaded inspiration causes rigidity of the upper airway rather than dilatation [13]. Furthermore, it has been proven that nonmuscular soft tissue movement impacts upper airway patency. The movement of nonmuscular soft tissue and the activation of the genioglossus muscle have a complex interplay.

Although electromyography of laryngeal muscles (such as the genioglossus or cricothyroid muscle) is possible during NIV [14], electromyography does not provide information on nonmuscular soft tissue movement. As a result, additional procedures should be utilized to assess upper airway patency. Magnetic resonance imaging is the most reliable way to get information, but it is also the most expensive and takes the most time, especially for people with NIV.

Although the upper airway can be viewed with ultrasound, the utility of using this technology to measure upper airway patency has not been investigated [14]. Endoscopy has also been used to measure upper airway patency, but it should preferably be utilized at several levels in the upper airway.

Conclusion

Future research and clinical relevance increasing the success rate of NIV are critical from a clinical standpoint. In contrast to invasive ventilation, NIV relies heavily on the upper airway as a conductor of air. Recent research shows that during NIV, it is important for the ventilator to have synchronization with the muscles in the upper airway to make sure there is enough ventilation.

The effects of NIV on upper airway physiology in patients with acute respiratory failure are currently unknown. When the ventilator cycles in time with the upper airway, it is fair to predict that ventilation efficiency will improve. The neuronal respiratory drive is linked to upper airway patency. As a result, better synchronization between the ventilator and respiratory drive may increase ventilation by reducing wasted ventilation at the upper airway level. At this time, it would be premature to provide recommendations on how to adjust the level of assistance, the level of positive end-expiratory pressure, and the flow pattern to improve upper airway patency in patients with acute respiratory failure [7].

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