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Case Report

Oxygen is Not a Substitute for Noninvasive Ventilatory Support and Mechanical In-exsufflation When Treating Hypoxia in Neuromuscular Disorders: A Case Study

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Abstract

Physicians and emergency services routinely administer supplemental oxygen (O_2) for people with severe respiratory symptoms. If CO_2 levels are elevated, as at times for neuromuscular disorders, this often causes CO_2 to soar and results in intubation, failure to ventilator wean, then tracheotomy or terminal extubation with morphine and O_2. However, the third option when unweanable is extubation to continuous noninvasive ventilatory support (CNVS) with mechanical in-exsufflation (MIE) used to reverse all O_2 desaturations due to airway mucus. In a recent study 320 patients developed coma and required intubation within minutes of O_2 administration causing acute hypercapnic respiratory failure. Once intubated, patients can become ventilator unweanable due to the airway tube itself. For patients with ventilatory pump failure of any cause, hypercapnic respiratory failure can be prevented by using NVS via noninvasive patient ventilator interfaces along with MIE when unassisted cough peak flows are below 200 L/m, and therefore, ineffective. O_2 should not be used as a substitute for NVS and MIE.

Keywords: *Ventilatory Pump Failure (VPF); Cough Peak Flows (CPF); Neuromuscular Diseases (NMDs); Hypoxia*

Introduction

Ventilatory pump failure (VPF) is the inability of dysfunctional inspiratory muscles to maintain lung ventilation. This usually happens along with having ineffective cough peak flows (CPF) for airway clearance. In physiatric practice this is typically seen in people with neuromuscular diseases (NMDs), multiple sclerosis, spinal cord injury, morbid obesity, severe skeletal deformities, advanced age, especially when associated with obesity, malnutrition, immobility, and deconditioning, and in some other neurological conditions. Severe brain disorders can also precipitate ARF, but they typically do not result in VPF as patients often retain the reflex ability to breathe

provided O_2 is avoided and airway clearance optimal [2,3]. Patients with VPF calling emergency services, or presenting to emergency departments (EDs) with dyspnea, whether due to hypoventilation or airway secretions from respiratory tract infections, as well as people under general anesthesia, are typically placed on supplemental O_2. This is done even when oxyhemoglobin saturation (SpO_2) levels are normal and irrespective of blood CO_2 levels. In a recent study, 320 VPF patients were intubated within minutes as administered O_2 caused hypercapnic ARF [1]. While it is widely appreciated that hypercapnic ventilatory failure patients are in danger of ARF if treated with O_2, this is widely ignored for VPF patients for whom O_2 cannot substitute for even continuous noninvasive ventilatory support (CNVS) and mechanical in-exsufflation (MIE) to prevent ARF.

The effects of oxygen

Typically, O_2 administration for hypercapnic patients with only VPF results in intubation, then airway secretions that cause patients to remain ventilator unweanable until the tube is removed to CNVS and MIE [3,4]. The O_2 administration diminishes hypoxic drive and renders oximetry useless as a gauge of alveolar ventilation, airway secretions, and lung pathology. When ventilator weaning parameters and spontaneous breathing trials fail, patients are conventionally offered only tracheotomy or palliative care terminal extubation. Extubation attempts to often inadequate pressure support (PS) bi-level noninvasive ventilation (NIV) such as inspiratory/expiratory 10/5 cm H_2O often result in extubation failure. However, NIV used at full NVS settings, whether by volume or pressure assist-control modes, combined with MIE for airway clearance, can both avert the need to intubate, and if not, avoid resort to tracheotomy and/or death subsequently whether they remain unweanable or not [3-5]. We have reported the extubation of 254 or 257 unweanable VPF patients, with vital capacities (VCs) as low as 0 ml, to CNVS with the only three undergoing tracheotomies, two due to severe concomitant lung disease and one due to cardiovascular instability [3,4]. Once extubation to CNVS and with MIE used for all O_2 desaturations below 95%, all 254 patients unweanable at the time of extubation subsequently weaned back to any pre-hospitalization ventilator use regimen [3,4].

Bi-level positive airway pressure or "NIV"

While bi-level NIV can theoretically provide NVS settings when used at PS levels of 18 to 20 cm H_2O and, thereby, maintain normal O_2 and CO_2 levels for VPF patients, it is usually not used at these settings. In patients with VPF using passive ventilator circuits, the expiratory positive airway pressure (EPAP) prevents full expiration and cannot be turned off. Crescimanno., *et al*. reported for advance bulbar ALS that EPAP leads to increased air leaks, reduced N3 stage sleep and overall sleep quality, increases breathing effort, ventilator auto-triggering, premature cycling, ventilation dyssynchrony, prolonged insufflations and reverse triggering, increases risk of aspiration and reflux, and increases sympathetic myocardial stimulation with decreased heart rate variability and it does not improve SpO_2, time spent with SpO_2 below 90%, or O_2 desaturation index. There was also no evidence that EPAP improved symptoms, which instead worsen as CO_2 levels increased [6].

NVS via active ventilator circuits also allows for complementary interventions including lung volume recruitment, manually assisted coughing [7] and glossopharyngeal breathing that enable ventilator-free breathing even for patients with little to no measurable VC [8]. Dating back to the transition from Iron Lungs to NVS, mostly in 1957, hundreds if not thousands of patients with NMDs have used active circuits for CNVS at 650 to 1800 ml volumes for inspiratory pressures (PIPs) averaging about 20 cm H_2O, all without tracheostomy tubes, including some for over 65 years. MIE delivered at pressures of 50 to 60 cm H_2O via the upper airways and 60 to 70 cm H_2O via airway tubes also facilitates noninvasive alternatives to trach or death [3,4,9-11].

To further prove that tracheostomy is not required for solely being too weak to breathe, we now have 23 patients with spinal muscular atrophy type 1, who have been dependent on nasal CNVS since as young as 3 months of age, most with no measurable VC and only trace volitional eye movements [12]. Despite this, ARF remains the leading cause of morbidity and mortality in patients with NMDs because it is often treated as illustrated in the following case.

Case Presentation

A 28-year-old with Duchenne muscular dystrophy (DMD) was diagnosed at 4 years of age and wheelchair dependent by age 9. At age 15, without access to MIE, he had a pneumonia and began using sleep nasal NVS. Over time he extended NVS throughout the day for CNVS. His VC was 260 ml in February 2025 and SpO_2 was normal but end-tidal CO_2 was 48 mm Hg when not using mouthpiece NVS. His NVS settings on a Trilogy ventilator were: Vt 1350 ml, rate 12, fiO_2 21%, active circuit. He could not generate any CPF. On these nasal and mouthpiece NVS settings, his mean SpO_2 and CO_2 levels remained normal 24 hours a day.

He developed a cold on October 10, 2025 and visited an ED on October 21st. Triage personnel removed him from his CNVS thinking it was "CPAP" and administered supplemental O_2 on which he quickly developed hypercapnic ARF and was intubated like 320 other such recently reported patients [1]. He remained ventilator-unweanable but transfer to our hospital for extubation back to CNVS could not be arranged for lack of funding. On October 23rd it was explained to his mother that he needed to have normal SpO_2 in ambient air and be extubated to his home CNVS settings as described for the 254 other such unweanable patients extubated successfully to CNVS and MIE [3,4]. However, on October 25th, with SpO_2 normal on fiO_2 21%, he was extubated at his local hospital, not to NVS settings, but to fiO_2 40% and volume targeted bi-level at 500 ml with pressure ranges of 12 maximum and 5 cm H_2O minimum for PS and volumes about one-third of what he needed for normal blood gases. Maximum pressure limited to 12 cm H_2O was far below the 20 cm H_2O needed for a full breath so he got much less than the 500 ml target. Instead of being returned to home NVS settings, fiO_2 was increased to 100% and the bi-level settings were increased to a range of 30/5 cm H_2O but the target volume remained 500 ml and his airways were not cleared by MIE. The MIE would also have hyperventilated his lungs, but the treating physicians refused to allow his mother to administer it which she had performed daily for him for lung volume recruitment and for assisted coughing for over 14 years. He suffered a cardiac arrest and an emergency tracheotomy was attempted but he died.

Discussion

In one year, four of our DMD patients dependent on CNVS presented to EDs, only two with respiratory complaints, and were removed from CNVS because the staff thought they were using CPAP or bi-level units, not ventilators. All four died and two resulted in successful lawsuits [13]. Despite being CNVS dependent even before hospitalization and the O_2 causing hypercapnic ARF and intubation, since his ambient air SpO_2 was normal while intubated, his local physicians extubated him to O_2 and grossly inadequate NIV. The O_2 prevented SpO_2 decreasing below 95% to indicate hypoventilation or airway secretions and did not address the causes of the hypoxia but only exacerbated hypercapnia. For 14 years, NVS and MIE had maintained his blood gases normal without O_2 but the treating physicians did not permit NVS nor his mother to use MIE to clear his airways.

Any SpO_2 below 95% should have triggered an evaluation of why the NVS was no longer adequate. First, inspiratory pressures should have been checked for adequate air delivery, then secretions expulsed by MIE. If pressures were low, then NVS leakage should be addressed, interface retention straps tightened, or switching to an oronasal interface. He should have been extubated without O_2. Even fiO_2 of 25% could have allowed CO_2 levels to rise into the 60s without desaturations to alert clinicians.

In a recent study of 486 patients with DMD, all dependent on wheelchairs by age 12 and on NVS/CNVS, only three required tracheostomy tubes but only for severe bronchiectasis, not ventilatory failure. Fourteen were over age 50 and 57 were over age 40. Many had no measurable VC. For 81 consecutive intubations for ARF, all unweanable before and immediately after extubation and none passing ventilator weaning parameters or ventilator weaning trials, all were successfully extubated to CNVS and MIE [3,4,14]. Many of the CNVS users had never been hospitalized for any respiratory difficulties, including some over age 50 [5]. Thus, O_2 does not address the underlying causes of hypoxia or hypercapnia and is not a substitute for NVS and MIE, day or night, for patients with only VPF [1,15].

Conclusion

Physicians frequently encounter patients with only ventilatory pump failure and put them into hypercapnic ventilatory failure by administering O_2 rather than NVS and MIE to renormalize their blood gases. This can have catastrophic effects on quality of life, personal and societal finances, and even survival. O_2 administration has become a reflexive standard practice and a common cause of iatrogenic harm. The case presented illustrates tragic consequences of this practice that typically but unnecessarily results in only the offered options of tracheotomy or terminal extubation. This manuscript presents data from over 500 patients showing that full ventilator setting noninvasive ventilatory support, combined with mechanical insufflation-exsufflation for airway clearance, can prevent need for intubation and enable successful extubation even for intubated patients "unweanable" by conventional criteria [14]. We reported the successful extubation of 254 of 257 ventilator-dependent VPF patients—including some with zero mL of vital capacity—to continuous noninvasive ventilatory support.

In summary, the key clinical insights are straightforward, that is, to recognize VPF as the sole pathology to address, avoid supplemental O_2, and to establish normal blood gases using NVS and MIE for airway clearance with oximetry and CO_2 monitoring for feedback. These interventions require no new technology — only physicians willing to explore a noninvasive treatment paradigm previously unknown to them for the benefit of their patients.

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Conflicts of Interest

The authors have no conflicts of interest to report.

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