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Case Series

Noninvasive Ventilatory Support to Avert Airway Tubes: A Retrospective Review of Emergency Cases

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Abstract

Introduction: The treatment paradigm for all dyspneic patients by emergency services is to administer oxygen (O_2) and possibly bi-level positive airway pressure at spans less than 15 cm H_2O. The O_2 for hypercapnic patients often results in hypercapnic coma, then resuscitation, intubation, and when unweanable, either tracheostomy or death via extubation to morphine and O_2. However, intubation can be averted for patients with only weak respiratory muscles by providing noninvasive ventilatory support (NVS) at 18 to 20 cm H_2O and mechanical in-exsufflation (MIE) at 50 to 60 cm H_2O to clear the airways. The following cases demonstrate this.

Conclusion: Up to continuous NVS and MIE can avert intubation for managing ventilatory pump failure in Emergency Departments. This has the potential to preserve quality of life and prolong life without resort to tracheostomy tubes and millions of dollars of subsequent nursing expenses.

Keywords: *Noninvasive Ventilation; Noninvasive Ventilatory Support; Tracheostomy; Muscular Dystrophy; Emergency Medicine; CO_2 Narcosis; Respiratory Failure; Mechanical Insufflation Exsufflation*

Introduction

Patients with progressive neuromuscular diseases and ventilatory pump failure (VPF) eventually have ineffective cough peak flows (CPF) below 270 to 300 L/m [1,2], which result in an increased risk of upper respiratory tract infections (URIs) developing into pneumonias and acute on chronic respiratory failure (ARF). These patients become symptomatic, dyspneic, seek emergency services, and are almost always administered supplemental oxygen (O_2) without the clinician having knowledge of the patient's carbon dioxide (CO_2) levels [3], and possibly low pressure support bi-level inspiratory (IPAP) and expiratory positive airway pressure (EPAP). For many of these episodes CO_2 levels soar and they are intubated, can become unweanable largely because of the tube itself, then are only offered extubation to continuous tracheostomy mechanical ventilation (CTMV) (SOCIT) or palliative care with extubation to death on morphine and O_2 (SOCID). However, tracheotomies detract from quality of life [4], and many, if not most patients eventually die from complications of the tubes themselves [5,6]. This is the fate of most patients with severe VPF who are never offered both continuous noninvasive ventilatory support (CNVS) and mechanical insufflation exsufflation (MIE) at optimal settings of 50 to 60 cm H_2O via the upper airways and 60 to 70 cm H_2O via airway tubes to clear the airways [7].

Case Presentation

The following cases presented to emergency services with dyspnea and often symptoms of morning headaches, hypersomnolence, and orthopnea. The patients avoided intubation by having the O_2 discontinued and being placed on CNVS and MIE.

Case 1

Figure 1

Case 1: A currently 49 year old with muscle weakness since 2 years of age was diagnosed with Becker muscular dystrophy at 8 years of age and became wheelchair dependent by age 15. He began sleep nasal NVS in March 2013 on assist-control mode with an active ventilator circuit set to 1100 ml, rate 12, with fiO_2 21%. During an April 6, 2016 visit he reported to be air stacking ventilator delivered volumes to deep lung volumes every 15 minutes throughout the day to relieve dyspnea, but did not want to use NVS continuously into daytime hours to become "ventilator dependent". His VC was 580 ml, end-tidal (Pet)CO_2 was 57 mm Hg, and pulse oxyhemoglobin saturation (SpO_2) 93-94%. Told he needed mouthpiece NVS (Figure 1) during the daytime he only continued regular air stacking. On April 13, 2016 he went too long without air stacking, became obtunded, was brought to a local emergency department (ED) where his $PaCO_2$ was 77 mm Hg before O_2 therapy, became unresponsive on 100% fiO_2 with the CO_2 increasing to 177 mm Hg in minutes with a pH of 6.98 and HCO_3^- 41 meq. During resuscitation efforts his father called me and noted that his portable ventilator was in the trunk of his car. He was told to place him on it at his usual nasal NVS settings. His CO_2 and SpO_2 and cognitive status normalized in minutes and he was discharged home 30 minutes later. During subsequent annual visits he was using mouthpiece NVS daytime and nasal NVS for sleep on the same settings of 1100 ml, rate 12. His $PetCO_2$ ranged from 37 to 45 mm Hg and SpO_2 remained normal at annual visits over the next 10 years. His last recorded vital capacity in 2025 was 220 ml. Since his unassisted cough flows were only 100 L/m, he uses MIE at 60 cm H_2O to clear airway secretions during colds and for lung volume recruitment. His glossopharyngeal ("frog breathing") maximum single breath capacity (GPmaxSBC) is 1250 ml. He now uses GPB for ventilator free breathing along with mouthpiece NVS (Figure 1) throughout the day and nasal NVS for sleep and has so far continued to do so for 10 years with no further hospitalizations.

Case 2

One year after spinal instrumentation for scoliosis surgery, a 15 year old boy with DMD developed pneumonia and ARF from having ineffective cough flows during a URI. Placed on O_2 in a local New York City ED his $PaCO_2$ increased to 88 mm Hg. He was placed on 6 cm

H_2O bi-level pressure support with minimal improvement and sent home using supplemental O_2. The next month he returned to the ED with a $PaCO_2$ of 99 mm Hg and was diagnosed with anoxic encephalopathy. The physicians advised morphine and O_2. We were called and advised discontinuing the O_2 and to use bi-level at NVS settings of IPAP 18 and EPAP 2 cm H_2O but the clinicians refused to increase the bi-level. Refusing intubation, he was transferred from the New York ED to our clinic when his parents signed him out against medical advice. He was accompanied by a specifically trained respiratory therapist on the NVS bi-level settings. Upon arrival, we discontinued the O_2, began nasal NVS on volume assist control mode from a PLV100™ ventilator, active circuit, portable volume cycling at 800 to 1000 ml volumes, and instructed him and his parents in using NVS and MIE with oximetry feedback to maintain SpO_2 over 94%. He regained normal cognitive function. His SpO_2 and $PetCO_2$ normalized and we discharged him home where after 1 week, at home he weaned to sleep only NVS. Over the next 2 years he became CNVS dependent on settings of 1300 ml, rate 12/min, and remained so for 20 years as his VC decreased to 120 ml.

At age 35 he developed abdominal pain and his parents brought him to another New York City ED. The triage nurse referred to his ventilator as "bi-PAP" and the physicians removed it from him, without his consent, because it was "not hospital equipment". Removed from full NVS he immediately became apneic. His father rushed to place him back on the PLV100™ but he and his wife were forcibly removed under guard. The physicians then placed him on bi-level at 5 cm H_2O pressure support (IPAP 10, EPAP 5 cm H_2O) with 100% fiO_2, on which he arrested and died despite attempts to intubate him. His parents found him deceased 2 hours later when the guards went to supper. The abdominal pain he presented for was never worked up.

Case 3

A 38 year old man with limb girdle muscular dystrophy, was a patient of ours since 6 years of age. At age 33 in 2013 his $PetCO_2$ was 45 mm Hg but sleep mean SpO_2 was 94% with a mean $PetCO_2$ 51 mm Hg. He refused NVS. Three years later his VC had decreased to 640 ml; he was markedly overweight; $PetCO_2$ was 57 mm Hg and SpO_2 90-93% but he still refused NVS. Five months later he presented to our ED complaining of being unable to take a deep breath. His venous CO_2 was 63 mm Hg, HCO_3^- 36 mEq/L, and SpO_2 73-80%. We advised him, his mother, and the ER physician to administer NVS and MIE and not supplemental O_2 [3]. However, one hour later at 5 PM he was placed on 2 L/m O_2 via nasal cannula. At 7AM the next morning, still in the ED, he was comatose with a $PaCO_2$ of 188 mm Hg, pH 6.90, and was intubated. Subsequently we were called for assistance. We increased the ventilator settings to normalize his CO_2 during the day and extubated him to CNVS while still in the ED. He subsequently weaned to sleep NVS. From then on he used NVS, then CNVS as he has for the next 8 years.

Case 4

At man, age 57 in November 2016, developed limb weakness and was diagnosed with amyotrophic lateral sclerosis (ALS) in March 2017. He lost consciousness in a pulmonologist's office with a $PaCO_2$ of 68 mmHg and was brought to our ED on May 1, 2017. We placed him on NVS, he awoke, and his blood gases normalized. Only 2 weeks later he became nasal CNVS dependent, alternating nasal interfaces day and night, using a Trilogy ventilator on Vt 1060 ml, rate 16, with positive inspiratory pressures of 30 to 40 cm H_20. Airway secretions were expelled using MIE for effective exsufflation flows over 250 L/m despite his VC decreasing from 400 ml in 2017 to 90 ml in 2018 and by November of 2019, 40 ml. In December of 2019 his SpO_2 decreased below 40% and he was rescued repeatedly by MIE at home. In January of 2020 he died from an apparent mucus plug. Thus, after 2 years and 8 months of CNVS a tracheostomy was needed but refused and with death inevitable so we prescribed O_2 along with the CNVS for comfort care.

Discussion and Conclusion

Cases 1-3 demonstrated mildly hypercapnic people with neuromuscular disorders becoming CO_2 narcotic within minutes of administration of supplemental O_2 [3]. In each case, this was expeditiously corrected by using nasal NVS to avoid intubation in two cases and to permit extubation without resort to tracheotomy in the ED for case 3. Case 4 also developed CO_2 narcosis without supplemental

O_2 but was also spared intubation by being placed on CNVS and using MIE to clear secretions. These four patients, spared intubation and tracheotomies, have thus far survived using CNVS for a total of over 40 patient-years that would otherwise have been spent on CTMV and with nursing care, either at home or institutionalized, at over $500,000 per year.

In 2017 we published 316 cases of patients with VPF who were intubated for ARF after receiving supplemental O_2 instead of NVS and MIE that might have averted intubation [3]. The O_2 was delivered with no knowledge of the patient's CO_2 for 310 of them and it depressed hypoxic ventilatory drive to precipitate CO_2 narcosis. Even obtunded, patients' CO_2 and O_2 levels can be normalized by providing CNVS, and MIE to clear airway secretions. If only too weak to breathe, they can also be extubated or decannulated of tracheostomy tubes to CNVS and MIE as needed with essentially 100% success [8-10].

Typically, for Case 2, neither his physicians, nor the neurologist and two pulmonologists who defended them in a losing court case understood that ventilatory support could, and was being provided noninvasively. They did not understand that even bi-level PAP could have been used at NVS settings with pressure support greater than 16 cm H_2O rather than the 4 cm H_2O they used, nor did they understand that MIE needs to be used to clear the airways at the effective 40 mm Hg (55 cm H_2O) pressures used when it was first on the market from 1953 through 1967.

In one year four of our CNVS users with Duchenne dystrophy presented to EDs with chest colds but instead of it being understood that they were dependent on NVS, they were removed from their ventilators without their consent. Two died within minutes and the third suffered a cardiopulmonary arrest, severe anoxic encephalopathy, tracheotomy, and spent the last two months of his life in a nursing home. It is crucial for ED clinicians to understand NVS and that it should be provided for patients who are only too weak to breathe and that O_2 is not a substitute for it.

These four successful outcomes using CNVS and MIE to avoid airway tubes were facilitated by the expertise of specifically trained respiratory therapists using NVS and MIE at effective settings that has not been reported elsewhere in the United States. Conventionally, patients fail extubation due to retained secretions and are often labeled "ventilator unweanable" or "NIV failure" and are subjected to tracheotomies without MIE or it being used at effective H_2O settings. However, as demonstrated in this review, MIE can reliably simulate effective coughing by providing effective exsufflation flows to expel secretions to avoid intubation and can facilitate extubation without resort to tracheostomy tubes.

Thus, emergency personnel should be alert to the possibility of hypercapnia in wheelchair users with weak upper limb muscles, with or without overt respiratory symptoms. If hypercapnia is suspected, NVS should be administered for symptomatic patients, via mouthpiece or nasal interface to re-normalize CO_2 and return ambient air SpO_2 saturation to normal, 95%. If airway secretions and weak cough flows are suspected, MIE should be provided at 50 to 60 cm H_2O pressures via the upper airways, 60 to 70 cm H_2O pressures via airway tubes as it was successfully used when first on the market in the 1950s and 1960s [6], as well as currently in centers that entirely avoid resort to tracheostomy tubes for muscular dystrophy and other such patients [8-11]. CNVS and MIE can not only reverse respiratory failure but also prolong quality lives and avoid the half million dollar per year costs of nursing care for TMV users. For EDs, CNVS and MIE need to become the first-line intervention for neuromuscular respiratory failure.

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