Rev Port Pneumol. 2016;22(5):262–265

revista portuguesa de
PNEUMOLOGIA
portuguese journal of pulmonology
www.revportpneumol.org

ORIGINAL ARTICLE

Quantitation of oxygen-induced hypercapnia in respiratory pump failure

M. Chiou[a], **J.R. Bach**[a,*], **L.R. Saporito**[a], **O. Albert**[b]

[a] *Department of Physical Medicine and Rehabilitation, Rutgers New Jersey Medical School, Newark, NJ, USA*
[b] *Respiratory Critical Care Department, King Fahad Medical City, Riyadh, Saudi Arabia*

Received 26 January 2016; accepted 5 March 2016
Available online 23 April 2016

KEYWORDS
Neuromuscular disease;
Noninvasive ventilation;
Permissive hypercapnia;
Supplemental oxygen therapy

Abstract
Purpose: To determine the likelihood that clinicians know carbon dioxide levels before administering supplemental oxygen to patients with neuromuscular disorders, to quantitate the effect of oxygen therapy on carbon dioxide retention, and to explore hypercapnia contributing to the need to intubate and use of continuous noninvasive ventilatory support to avert it.
Basic procedures: A retrospective chart review for patients with neuromuscular disorders intubated or having intubation averted by using continuous noninvasive ventilatory support with carbon dioxide known pre- and during oxygen administration.
Main findings: For only 2 of 316 patients who were intubated did clinicians know carbon dioxide levels prior to administering oxygen. For four cases, intubation was averted by continuous noninvasive ventilatory support and mechanical insufflation–exsufflation despite severe hypercapnia and acidosis. After initiating oxygen therapy, patients' carbon dioxide partial pressures increased 52.1 ± 42.0 mm Hg in over as little as 20 min.
Principal conclusions: Clinicians should attempt to use continuous noninvasive ventilatory support and mechanical insufflation–exsufflation rather than supplemental oxygen to normalize blood gases for neuromuscular ventilatory failure and should be prepared to intubate hypercapnic patients for whom oxygen is administered.

Abbreviations: ABG, arterial blood gas; CMV, continuous mandatory ventilation; CNVS, continuous noninvasive ventilatory support; CO_2, carbon dioxide; CTMV, continuous tracheostomy mechanical ventilation; EPAP, expiratory positive airway pressure; $EtCO_2$, end-tidal carbon dioxide; FEV1, forced expiratory volume-one second; FiO_2, fraction of inspired oxygen; ICU, intensive care unit; IPAP, inspiratory positive airway pressure; MIE, mechanical insufflation–exsufflation; NMD, neuromuscular disease; NVS, noninvasive ventilatory support; O_2, oxygen; O_2 sat, oxyhemoglobin saturation; $PaCO_2$, partial pressure of carbon dioxide in arterial blood; PaO_2, partial pressure of oxygen in arterial blood; PAP, positive airway pressure; SMA, spinal muscular atrophy; URI, upper respiratory tract infection; VC, vital capacity; Vt, tidal volume.

* Corresponding author.
E-mail address: bachjr@njms.rutgers.edu (J.R. Bach).

http://dx.doi.org/10.1016/j.rppnen.2016.03.005

Introduction

Although administering supplemental oxygen (O_2) to hypercapnic patients with cardiopulmonary disease can decrease dyspnea, pulmonary hypertension, polycythemia, exercise intolerance, permit greater ventilator-free breathing particularly for chronic lung disease patients, and prolong life,[1–5] it can also depress hypoxic ventilatory drive and exacerbate hypercapnia.[2,3] With the advancing respiratory muscle weakness of patients with neuromuscular disease (NMD), however, it can render noninvasive ventilation ineffective,[6] which can cause carbon dioxide (CO_2) narcosis, coma, and acute on chronic respiratory failure.

In intensive care units (ICUs), since it is far more common to encounter patients with lung disease than NMDs, it is generally assumed that O_2 administration is harmless for anyone with respiratory symptoms and that NMD patients must accommodate elevated baseline CO_2 levels anyway so emergency services tend to administer O_2 immediately to all such symptomatic patients without first determining CO_2 levels.[7] The only publication that has thus far quantitated O_2-induced hypercapnia in NMD is that of Gay and Edmonds in 1995,[8] and they could only identify the pre- and post-O_2 delivery CO_2 levels for eight patients including three with polymyositis, three with motor neuron disease, and one each with inflammatory motor neuropathy and chronic poliomyelitis. The patients received low-flow O_2 and within 0.8–144 h, three became obtunded and required intubation but subsequently recovered; and two died when intubation was declined. Neither noninvasive ventilatory support (NVS) nor mechanical insufflation–exsufflation (MIE) was used to either avoid intubation or facilitate successful extubation.

Bi-level positive airway pressure (PAP) is generally used in acute and chronic care at less than full ventilatory support settings. However, many patients with little or no vital capacity (VC) require continuous NVS (CNVS) at volume or pressure settings that fully support respiratory muscle function. Two studies reported 166 patients with respiratory pump failure who required intubation after having been administered O_2 supplementation.[9,10] Another 61 patients underwent tracheotomy after being intubated following O_2 administration for acute on chronic respiratory failure.[11] None of these 227 patients had been offered CNVS or MIE[10] to either prevent intubation or facilitate extubation prior to transfer to a specialized unit for extubation or decanulation to CNVS and MIE. The purpose of this case series is to determine the likelihood that clinicians obtain CO_2 levels before administering O_2 to patients with NMD, to quantitate subsequent CO_2 levels as a contributing factor in the need to intubate, and to describe the use of CNVS and MIE to reverse hypercapnia and avert invasive airway intubation.

Methods

Consecutive outpatients to a NMD clinic from 1996 to 2015 were screened for having received O_2 therapy, undergoing intubation then possibly tracheotomy, then being extubated or decanulated to CNVS and MIE. In addition, we screened for others whose hypercapnia had been documented either by arterial blood gas sampling or by end-tidal CO_2 ($EtCO_2$) measurements before O_2 therapy but who were spared intubation by using CNVS and MIE. The MIE was administered by both hospital staff via invasive airway tubes and by the patients' relatives post-extubation/decanulation in the acute care setting up to every 20–30 min during waking hours via oronasal interfaces at insufflation and exsufflation pressures of 50–60 cm H_2O until ambient air oxyhemoglobin saturation (O_2 sat) baseline remained greater than 94%.

Results

Of 2804 patients with NMD, 316 were identified who had undergone intubation, then 61 of 316 tracheotomy. All were subsequently extubated or decanulated to CNVS and MIE.[9–11] Of these, only nine cases had had CO_2 levels available both pre- and during O_2 administration, and for only cases 1 and 4 was the CO_2 known by the clinicians who administered the O_2 and intubated the patients. For cases 2, 3, 7, and 9 whose hypercapnia worsened during O_2 therapy, intubation was averted by using CNVS and MIE.

Case presentations

Case 1: A 27-year-old woman with nemaline rod myopathy had failed extubation following scoliosis surgery at age 10, but she was successfully extubated to CNVS and MIE upon transfer to our intensive care unit (ICU).[9,10] From age 10 to 27, she used bi-level PAP at an inspiratory (I)PAP of 18 cm H_2O, expiratory (E)PAP of 4 cm H_2O, and rate 13 breaths/min only during intercurrent upper respiratory tract infections (URI) despite having a VC as low as 340 mL. In December 2014, she presented to emergency services with a URI and partial pressure of CO_2 in arterial blood ($PaCO_2$) of 47 mm Hg despite continuous use of bi-level PAP. Then, she was started on supplemental O_2. Over a 2-h period, her $PaCO_2$ increased to 67 mm Hg. She became obtunded, was intubated, and failed one extubation attempt to IPAP 8 cm H_2O, EPAP 4 cm H_2O, and supplemental O_2. After nine total days of intubation, she was transferred to our ICU, where she was successfully extubated to CNVS and MIE. Her $PaCO_2$ remained below 40 mm Hg, and she was discharged home 4 days later, where she eventually weaned to nocturnal nasal NVS.

Case 2: A 32-year-old wheelchair-dependent man with Becker muscular dystrophy began to use nocturnal nasal NVS in November 2013 to treat fatigue, sleepiness, difficulty concentrating, and hypercapnia. He had a VC of 550 mL and an $EtCO_2$ of 77 mm Hg. His symptoms cleared and diurnal $EtCO_2$ decreased to 54 mm Hg, but since he refused to use NVS during daytime hours, he again developed mild confusion and presented to emergency services in February 2014. He was placed on 100% fraction of inspired O_2 (FiO_2) and became obtunded over the next 20 min. An arterial blood gas (ABG) revealed a $PaCO_2$ of 177 mm Hg, bicarbonate of 41 meq/L, and pH of 6.98. The patient was manually resuscitated and immediate intubation was recommended. However, the father insisted that the author (Bach) be called first, and when it was discovered that the patient's portable ventilator was in the trunk of his car, the father retrieved it and administered nasal CNVS at a volume-preset of 1150 mL and rate 12 breaths/min. A few minutes later, the patient transitioned to mouthpiece CNVS and, 20 min later, was discharged home with normal O_2 sat, fully coherent, and in no distress.