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Successful Use of Combined High-frequency Oscillatory Ventilation, Inhaled Nitric Oxide, and Prone Positioning in the Acute Respiratory Distress Syndrome

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MANAGEMENT of patients with acute respiratory distress syndrome (ARDS) is a significant challenge to clinicians. Recently, concern has been expressed that conventional strategies using high airway pressures may contribute to lung injury and perhaps to multisystem organ failure in patients with ARDS.[1] Consequently, other strategies of respiratory support have been used in the hope of improving gas exchange while avoiding ventilator-induced lung injury, including prone positioning, high-frequency ventilation, inhaled nitric oxide (INO), and partial liquid ventilation. Because of the complexity of this illness, although individual interventions may not result in improved outcome, combined modalities as part of a comprehensive treatment strategy may become an important feature of future investigations. The strategies mentioned have been used in combination in animals, but rarely in humans.[2-4] We present a case report of the successful use of high-frequency oscillatory ventilation (HFOV), prone positioning, and INO in a patient with severe ARDS.

Case Report

A 56-yr-old man was brought to the emergency department of a community hospital with respiratory failure caused by a drug overdose and aspiration of gastric contents. His medical history was significant for bipolar affective disorder and type 2 diabetes mellitus. He had overdosed on a number of medications, including a benzodiazepine, an antipsychotic, and an antidepressant. He underwent intubation in the emergency department and was transferred to the intensive care unit. His sputum and blood cultures were subsequently positive for *Staphylococcus aureus*, and bronchoalveolar lavage showed herpes simplex virus. He was treated with appropriate antibiotics and supportive care. His chest radiograph initially showed a focal infiltrate, but this progressed to diffuse bilateral infiltrates. One week after admission, he was transferred to our intensive care unit in a tertiary care university-affiliated hospital for further management of severe ARDS. On day 1 in our intensive care unit, conventional mechanical ventilation was continued. A pressure-control mode was used with a peak airway pressure of 30 cm H_2O and a positive end-expiratory pressure of 15 cm H_2O. Delivered tidal volume was 450 ml (5.5 ml/kg). While breathing a fraction of inspired oxygen (Fio_2) of 0.5, his blood gas showed a pH of 7.33, an arterial carbon dioxide tension ($Paco_2$) of 61 mmHg, an arterial oxygen tension (Pao_2) of 64 mmHg, a bicarbonate concentration of 30 mEq/l, and an arterial oxygen saturation (Sao_2) of 93%. His oxygenation status worsened, and he required an Fio_2 of 1.0 to maintain an Sao_2 of 90% or more. The patient was already deeply sedated, and a neuromuscular blocking agent was administered. INO was initiated, and within a short period, his Fio_2 was reduced to 0.55 (see table 1 for a summary of ventilator settings, oxygenation, and ventilation at the time of initiation of INO, HFOV, and prone positioning). His respiratory status, however, continued to worsen. Despite a peak inspiratory pressure of 40 cm H_2O, tidal volumes decreased to 300 ml, and worsening hypercapnia developed. Because of concern about high peak pressures as well as the increasing $Paco_2$ and increasing Fio_2 requirements, the patient was placed on a high-frequency oscillatory ventilator (3100 B; Sensormedics, Yorba Linda, CA). The mean airway pressure was initially set at 32 cm H_2O, 3 cm H_2O above the mean airway pressure applied during conventional mechanical ventilation. A few hours later, blood gas with an Fio_2 of 0.7 showed an improved $Paco_2$. The following morning, because of worsening hypoxemia, the mean airway pressure was increased to 36 cm H_2O, but with further deterioration, the patient was placed in the prone position. Shortly thereafter, the patient's oxygenation improved, and the Fio_2 and the mean airway pressure were reduced to 0.5 and 32 cm H_2O, respectively. The oscillatory frequency throughout his course was 4–6 Hz. Because of this excellent response, the patient was placed in the prone position every 6–8 h and left prone for 6–8 h at a time. After 4 days of combined prone positioning and HFOV, he was returned to conventional mechanical ventilation and kept in the supine position. He was gradually weaned from INO, and INO was discontinued after a total of 9 days. During the next month, he was gradually weaned to supplemental oxygen *via* tracheostomy and was then transferred to the ward. No evidence of multisystem organ failure nor any complications related to mechanical ventilation developed. He was subsequently discharged from hospital.

Discussion

Despite profound gas exchange abnormalities, the most common cause of death in patients with ARDS is sepsis and multisystem organ failure.[5] Patients with ARDS have significantly reduced respiratory system compliance; hence, it is often difficult to support oxygenation and ventilation adequately without subjecting them to potentially harmful transpulmonary pressures and tidal volumes. There is a concern that ventilator strategies used in the treatment of these patients not only result in local damage to the lung, but may promote more widespread inflammation, which contributes to

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Table 1. Summary of Ventilatory Modes and Arterial Blood Gases

ICU Day	Time	INO (ppm)	Patient Position	Vent Mode	P_{aw} (cm H_2O)	F_{IO_2}	pH	$Paco_2$ (mmHg)	Pao_2 (mmHg)	Spo_2 (%)	Pao_2/F_{IO_2} (mmHg)	OI
3	17:00	0	Supine	PCV	20	1.0	7.35	67	68	94	68	29
4	04:30	20	Supine	PCV	27	0.55	7.34	70	59	90	107	25
5	10:20	10	Supine	PCV	29	1.0	7.27	82	59	89	59	49
5	12:20	20S	Supine	HFOV	32	0.7	7.37	61	48	86	73	47
6	04:30	20	Supine	HFOV	35	1.0	7.25	90	47	86	47	74
6	08:10	10	Prone	HFOV	32	0.6	7.24	98	73	91	122	26
6	17:15	12	Supine	HFOV	32	0.95	7.32	76	56	89	59	54
7	13:15	10	Prone	HFOV	32	0.5	7.45	59	61	93	122	26

ICU = intensive care unit; INO = inhaled nitric oxide; ppm = parts per million, P_{aw} = mean airway pressure; F_{IO_2} = fraction of inspired oxygen; $Paco_2$ = arterial carbon dioxide tension; Pao_2 = arterial oxygen tension; Spo_2 = oxygen saturation measured by pulse oximetry; OI = oxygenation index; PCV = pressure control ventilation; HFOV = high frequency oscillatory ventilation.

multiorgan failure.[1] A number of strategies have been developed that can support ventilation in patients with ARDS while potentially reducing exposure to these harmful effects. Two such strategies are HFOV and prone ventilation.

High-frequency oscillatory ventilation is one of a number of high-frequency ventilatory modes that has been investigated in the setting of ARDS. The mean airway pressure is generally set 3–5 cm H_2O higher than the mean airway pressure applied during conventional mechanical ventilation, and the alveolar pressure is well above the pressure at which derecruitment of lung units is thought to occur. The variations, or oscillations, around this mean airway pressure are believed to be dissipated and not transmitted to the alveolar epithelium. This theoretically allows the recruitment of lung units without overdistention.

Studies in neonates and children have shown improvements in oxygenation and reduction in chronic lung disease using HFOV; however, no mortality benefit has been demonstrated.[6–8] In adults, studies are limited to case series in which HFOV has been used as a rescue strategy.[9,10] HFOV has been used safely and seems to improve oxygenation; however, randomized control trials assessing secondary outcome measures are lacking.

The prone position has been investigated for a number of years as a therapeutic intervention in ARDS. There have been several uncontrolled trials showing that oxygenation can safely be improved in patients who are turned prone,[11–13] and two randomized controlled trials are currently underway. Patients in the supine position have a pleural pressure gradient that increases dorsally because of the weight of the lung and mediastinal structures. In ARDS, this gradient is exaggerated because of inflammation and edema present in the lung. Prone positioning reduces the pleural pressure gradient as the mediastinal and abdominal organs move ventrally. This allows for recruitment of dorsal alveolar units at any given alveolar pressure. In addition, blood flow is redistributed away from shunt regions, thus increasing areas with a normal ventilation/perfusion ratio.[14]

Another concern in ARDS is severe hypoxemia. Although the major cause of death in patients with ARDS is multiple organ failure, a proportion of patients may die as a result of hypoxemia. In addition, high oxygen levels can theoretically result in promotion of diffuse alveolar damage and possibly impact on long-term lung function. Both HFOV and prone positioning may improve oxygenation by means of alveolar recruitment. Nitric oxide, on the other hand, is a strategy that may improve oxygenation by selectively improving perfusion to well-ventilated areas of the lung, thus improving ventilation/perfusion matching. Recently, a large randomized trial showed acute improvements in oxygenation without mortality benefit.[15]

Given the mechanisms of action and potential benefits of each of these interventions alone, it is possible that together they may have an additive or even synergistic effect. There have been a few small studies that have evaluated such combined modalities, but to our knowledge, none have shown successful use of INO, HFOV, and prone ventilation in patients with ARDS. In the case described, despite high driving pressures, $Paco_2$ continued to increase, and oxygenation deteriorated despite high levels of positive end-expiratory pressure. The use of INO was followed by an improvement in oxygenation. The use of HFOV allowed improved ventilation and adequate oxygenation while potentially exposing alveoli to lower pressure excursions than those during conventional mechanical ventilation. Turning the patient prone led to further oxygenation improvement, allowing further reductions in mean airway pressure. Although no definite conclusions about outcome can be drawn from a single case report, we have described one patient in whom multiple modalities were used safely and effectively in combination. Patients with ARDS clearly have many complex and dynamic physiologic derangements. During their course in the intensive care unit, these patients often receive hundreds of interventions. Therefore, it is not surprising that trials examining a single intervention have failed to show an improvement in clinical outcome. Future studies should give consider-

ation to combining modalities with complementary physiologic endpoints and perhaps should test a comprehensive treatment strategy compared with usual care.

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Anesthetic Management for Patients with Postpolio Syndrome Receiving Electroconvulsive Therapy

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ALTHOUGH acute poliomyelitis has been virtually eliminated in the United States because of a successful vaccination program, it is estimated that there are between 250,000 (Driscoll *et al.*[1]) and 300,000 (Dalakas *et al.*[2]) survivors of acute poliomyelitis in this country. Many of these survivors are susceptible to the development of postpolio syndrome 25–30 yr after the initial infectious episode.[3] Patients with postpolio syndrome may have severe respiratory sequelae and neuromuscular dysfunction. Thus, they are a significant challenge to anesthesiologists when they undergo operative intervention secondary to coexisting disease. We present a patient with a major depressive disorder and postpolio syndrome who was scheduled to undergo electroconvulsive therapy (ECT).

Case Report

The patient is a 62-yr-old man admitted for ECT to treat major depression refractory to medical management. He had acute poliomyelitis in 1946 and recovered, but postpolio syndrome was diagnosed in 1996, with progressive weakness of both lower extremities. He was wheelchair bound. Other than symptoms related to his postpolio syndrome, his medical history was positive for chronic sinusitis, occasional heartburn, a spinal fusion in 1960 for scoliosis, and shoulder operations in 1995 and 1997, which were performed during general anesthesia without complications. His weight was 70 kg, his blood pressure was 133/73 mmHg, and his pulse was 88 beats/min. Physical examination results were negative except for muscle atrophy and weakness in both lower extremities and decreased reflexes.

We anesthetized the patient on four separate occasions within a period of 8 days to facilitate ECT (table 1). Monitoring consisted of electroencephalography, electrocardiography, blood pressure, pulse oximetry, and neuromuscular response to electrical stimulation. Each time, the patient was preoxygenated using a Mapleson D system and face mask. Esmolol was administered to modify the anticipated sympathetic response to ECT. Anesthesia was induced with 60 mg methohexital (0.85 mg/kg), and as soon as the patient lost consciousness, mivacurium was administered to attenuate the muscular response to ECT. When application of electric current was completed, the patient underwent ventilation with bag and mask until adequate spontaneous recovery of respiratory function. The ECT-induced seizures on these four occasions ranged in duration from 20–57 s. Blood pressure and pulse rate did not vary significantly during any of the four treatments. For the first two treatments, neostigmine and glycopyrrolate were not administered until the patient showed some respiratory effort. For the third and fourth treatments, neostigmine and glycopyrrolate were

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