Review

Clinical review: Ventilatory strategies for obstetric, brain-injured and obese patients

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Abstract

The ventilatory management of patients with acute respiratory failure is supported by good evidence, aiming to reduce lung injury by pressure limitation and reducing the duration of ventilatory support by regular assessment for discontinuation. Certain patient groups, however, due to their altered physiology or disease-specific complications, may require some variation in usual ventilatory management. The present manuscript reviews the ventilatory management in three special populations, namely the patient with brain injury, the pregnant patient and the morbidly obese patient.

Introduction

The principles of ventilatory management of patients with acute respiratory failure are supported by good evidence, including pressure limitation to avoid ventilator-induced lung injury [1] and regular assessment for discontinuation of ventilatory support [2]. The ventilatory approach in certain patient groups, however, may require some variation in usual management or in attention to unique issues or complications. This requirement may be related to altered physiology or disease-specific complications, and many of these patient groups have been excluded from traditional large studies due to this potential practice variation. The present manuscript reviews the ventilatory management in three special populations, namely the patient with brain injury, the pregnant patient and the morbidly obese patient.

The brain-injured patient

Physiological considerations

Although a decreased level of consciousness is the primary indication for initiation of mechanical ventilation in up to 20% of patients [3], approximately 20% to 25% of patients with isolated brain injury – both subarachnoid hemorrhage and traumatic brain injury (TBI) – develop acute lung injury/acute respiratory distress syndrome (ALI/ARDS). The presence of ALI/ARDS is associated with a threefold increased risk of death and with a prolonged intensive care unit (ICU) length of stay [4,5]. Furthermore, ALI/ARDS is the most frequent non-neurologic complication of TBI [6].

Several mechanisms have been proposed, but the underlying etiology of this pulmonary dysfunction remains unclear. The most plausible theory involves massive sympathetic discharge. After head trauma, several intracranial complications can occur, including increased intracranial pressure, ischemia or direct trauma to the hypothalamus or mass effect over the medulla. All of these effects may result in massive catecholamine release that produces systemic hypertension, increased peripheral vascular resistance, increased pulmonary artery pressure, pulmonary venous constriction, and stunned myocardium. Rapid development of generalized vasoconstriction leads to a volume shift from the high-pressure systemic circulation to the low-pressure pulmonary circulation. Edema formation is thought to be secondary to increased hydrostatic pressure and also to increased vascular permeability due to endothelial injury [7,8].

Intubation

Endotracheal intubation is clearly a critical and early step in the management of the comatose patient, but attempts at intubation in patients with severe TBI may result in hypoxia and raised intracranial pressure, and can be aggravated by rapid sequence induction [9].

Manipulation of the airway, including laryngoscopy and endotracheal intubation, can result in significant elevations in the heart rate, the mean arterial pressure, the plasma catecholamine levels and the intracerebral pressure (ICP)

ALI = acute lung injury; ARDS = acute respiratory distress syndrome; ICP = intracranial pressure; ICU = intensive care unit; $PaCO_2$ = partial arterial pressure of carbon dioxide; PEEP = positive end-expiratory pressure; TBI = traumatic brain injury.

[10]. Several pharmacologic agents have been studied to determine whether they are capable of attenuating this hemodynamic response to rapid sequence induction and intubation. These agents fall into three groups: lidocaine, β-blockers and opioids. It has been demonstrated that neuromuscular blockade alone, induction of general anesthesia alone, or both together, are not effective in blunting this hemodynamic response [11,12].

A recommended protocol includes preoxygenation for 5 minutes, and use of premedication with intravenous lidocaine and an opioid (for example, fentanyl) in patients who are hemodynamically stable or hypertensive, to decrease the adrenergic response and avoid fasciculation. This is followed by rapid sequence induction with a neuromuscular blocker with a rapid action (for example, succinylcholine) and a sedative agent (for example, thiopental, propofol) [13]. Use of lidocaine as premedication in this situation is not universally accepted, with very little evidence to support its use [14].

Mechanical ventilation

Current recommendations for mechanical ventilation in the brain-injured patient include maintaining the $PaCO_2$ between 35 and 40 mmHg, improving oxygenation while using low positive end-expiratory pressure (PEEP) and brief periods of hyperventilation for emergency treatment of intracranial hypertension [15]. Many patients with TBI develop ALI/ARDS, however, and use of higher tidal volumes with low PEEP is associated with an inflammatory response and exacerbation of ALI/ARDS [5,16]. Guidelines for ventilation in ALI/ARDS recommend low tidal volumes (6 ml/kg), plateau pressures below 30 cmH_2O, and variable levels of PEEP. These settings may produce hypoventilation and hypercapnia, which may increase the ICP. Randomized controlled trials in ALI/ARDS have excluded brain-injured patients, and specific data regarding protective ventilation in these patients are not available. In spite of this lack of information, there are some strategies used in patients with ALI/ARDS that have been tested in brain-injured patients, as described below.

Use of PEEP produces an increase in intrathoracic pressure and a reduced venous return, and may reduce cardiac output. In patients with brain injury, an elevated intrathoracic pressure may also decrease venous drainage from the superior vena cava and increase the ICP, thus reducing the cerebral perfusion pressure. These effects seem to occur only in those patients with hypovolemia and normal respiratory system compliance [15,17,18]. When PEEP produces recruitment, there is little adverse effect on the ICP – whereas in the patient with poor pulmonary compliance, PEEP may increase $PaCO_2$ and raise the ICP [17]. The use of at least 5 cmH_2O PEEP is reasonable in most patients, with higher levels in patients with oxygenation difficulties, and with appropriate monitoring of hemodynamics and ICP [15]. Lung recruitment maneuvers are associated with increased ICP and oxygenation, but with decreased mean arterial pressure, decreased cerebral perfusion pressure, and decreased jugular bulb oxygen saturation for up to 10 minutes [19].

Prone-position ventilation improves oxygenation by increasing lung recruitment, decreasing ventilation–perfusion mismatch and increasing secretion drainage, but does not alter mortality. In neurologic patients, prone positioning is associated with increased ICP and consequently with decreased cerebral perfusion pressure, although oxygenation and respiratory mechanics are consistently improved [20].

High-frequency ventilation is a combination of a high respiratory rate with a very small tidal volume and an elevated mean airway pressure. Use of high-frequency oscillatory ventilation in ALI/ARDS patients has been associated with safe and effective increased oxygenation and with decreased tendency to develop ventilator-induced lung injury, probably due to its ability to avoid overdistention and reduce alveolar derecruitment [21]. Clinical studies of high-frequency ventilation in brain-injured patients have reported a mild to moderate decrease in ICP and an increase in oxygenation and ventilation [22,23].

There is a lack of information regarding other nonconventional modes of mechanical ventilation such as nitric oxide in neurotrauma patients; however, there is a report of the use of pumpless extracorporeal lung assist in five patients with ARDS and severe brain injury. Reduced $PaCO_2$ and subsequent decreased ICP were reported [24].

Weaning

Little data exist to direct the timing and methods of weaning of neurological patients. As a consequence, delayed extubation, a high incidence of ventilator-associated pneumonia and a prolonged ICU length of stay have been reported in patients with TBI [25]. An extubation delay commonly occurs in patients who met standard respiratory and hemodynamic criteria for extubation, due to a decreased level of consciousness (for example, Glasgow coma scale ≤8). Successful extubation is achieved, however, in over 80% of patients extubated with Glasgow coma scale <8, even in those with weak or absent gag or cough [26]. Nevertheless, a small study evaluating protocolized extubation in neurosurgical patients reported that Glasgow coma scale >8 was associated with good prediction of successful extubation [27]. Although controversial, tracheostomy may be considered if after a period of stabilization the patient will require prolonged ventilator assistance [28].

The pregnant patient

Physiological changes

The pregnant woman experiences several physiological changes to the respiratory system. The upper airways may develop edema and hyperemia, contributing to the difficulty in endotracheal intubation of these patients. Changes in the chest wall and lung volumes occur due to the enlarging

uterus, causing a 10% to 25% decrease in functional residual capacity although the total lung capacity decreases only minimally [29]. Lung compliance is unchanged, but the chest wall and total respiratory compliances are reduced [30]. Minute ventilation increases, stimulated by the rising progesterone level. The tidal volume increases and minute ventilation reaches levels as high as 50% above baseline by term [31]. A mild respiratory alkalosis results with compensatory reduction in serum bicarbonate levels ($PaCO_2$ = 28 to 32 mmHg; HCO_3^- = 18 to 21 mEq/l). Oxygen consumption increases in late pregnancy due to the demands of the fetus and maternal metabolic processes, reaching levels up to 33% above baseline by term.

Oxygen delivery to the fetus depends on placental function and oxygen delivery to the placenta (that is, maternal arterial oxygen content and the uterine blood flow). Uterine flow is near maximal in the baseline state, and uterine arterial vasoconstriction can be precipitated by maternal hypotension, by alkalosis (for example, hyperventilation) as well as by endogenous or exogenous catecholamines [32]. Although umbilical venous blood returning to the fetus has a relatively low oxygen tension (25 to 30 mmHg), adequate oxygen content is maintained by the left shift of the oxygen dissociation curve of fetal hemoglobin.

Intubation

Failed intubation is eight times more common in the obstetric population than in other anesthetic intubations [33]. The diminished functional residual capacity and increased oxygen consumption cause a reduced oxygen reserve, producing rapid desaturation in response to apnea or hypoventilation [34]. Preoxygenation with 100% oxygen is beneficial, but respiratory alkalosis should be avoided. The pregnant patient should always be considered to have a full stomach, in view of the delayed gastric emptying and elevated intraabdominal pressure of pregnancy, and appropriate precautions should be taken. Upper airway hyperemia and edema may impair visualization and may increase the risk of bleeding. Nasal intubation should be avoided and a smaller endotracheal tube may be required.

Noninvasive ventilation

Noninvasive ventilation avoids the potential complications of endotracheal intubation, as well as the complications associated with sedation. This modality is well suited to short-term ventilatory support, which may be the case in many obstetric respiratory complications that reverse rapidly. The biggest concern with mask ventilation in pregnancy is the risk of aspiration, due to the presence of increased intra-abdominal pressure, delayed gastric emptying and reduced lower esophageal sphincter tone. Noninvasive ventilation should therefore be reserved for the pregnant patient who is alert and protecting her airway, and where there is an expectation of a relatively brief requirement for mechanical ventilatory support.

Mechanical ventilation

Data on the prolonged mechanical ventilation of pregnant patients in the ICU are limited. Hyperventilation should be avoided as this adversely affects uterine blood flow [35]. The standard ventilatory approach of avoiding excessive lung stretch by pressure and volume limitation, sometimes with permissive hypercapnia, has not been assessed in pregnancy. The usual pressure limits (for example, plateau pressure of 30 cmH_2O) may not be appropriate in the near-term patient, where chest wall compliance is reduced. Transpulmonary pressures may not be elevated at these pressures, and higher pressures may be acceptable in near-term pregnant patients to achieve appropriate tidal volumes. Oxygenation should be optimized to ensure adequate fetal oxygen delivery. Although late pregnancy is associated with a mild respiratory alkalosis, maternal hypercapnia up to 60 mmHg in the presence of adequate oxygenation does not appear to be detrimental to the fetus [36]. Fetal acidemia with associated fetal heart rate changes may occur; these changes do not necessarily indicate fetal hypoxia but may be secondary to the maternal acidosis. If marked respiratory acidosis results from permissive hypercapnia, treatment with bicarbonate may improve both maternal and fetal acidemia.

Other management issues

In the supine position, the near-term gravid uterus produces mechanical effects on the vena cava and aorta, reducing central venous return and decreasing cardiac output. This supine hypotensive syndrome should be considered in any hemodynamically unstable pregnant patient, and they should be positioned on their left side or with the right hip elevated [37].

Pregnancy increases the risks of venous thrombosis due to hypercoagulability and venous stasis. Antithrombotic measures, including physical interventions and heparin prophylaxis, should be utilized.

Radiological investigations are often essential for the assessment and management of the ventilated pregnant patient. Although there are potential risks of exposing the fetus to radiation, shielding the abdomen with lead and using a well collimated X-ray beam can effectively reduce exposure. The adverse effects of exposure of the fetus to radiation include oncogenicity and teratogenicity. A doubling of the risk of childhood leukemia may result from fetal exposure in the range of 20 to 50 mGy (2 to 5 rads). Teratogenicity occurs at radiation exposure greater than 50 to 100 mGy (5 to 10 rads), or somewhat lower in the first trimester. With appropriate precautions, fetal radiation exposure can be limited to safe levels for most procedures, although investigations such as abdominal–pelvic computed tomography will obviously cause significant fetal radiation exposure [38] (Table 1). Every effort should nonetheless be made to minimize uterine exposure, particularly in the first trimester.

Table 1

Risk of fetal radiation exposure resulting from radiological studies in the pregnant patient with respiratory failure

Investigation	Fetal radiation exposure (mGy)
Chest X-ray (with abdomen shielded)	0.01
Ventilation–perfusion scan	
Perfusion	0.1 to 1.0
Ventilation	0.1 to 0.4
Computed tomography pulmonary angiogram	0.1 to 1.0
Computed tomography pelvis and abdomen	30 to 50
Radiation effect on the fetus	
Teratogenicity	50 to 100
Oncogenicity	20 to 50

Little data exist to identify the optimal drugs for prolonged sedation, analgesia or neuromuscular blockade in pregnancy. Benzodiazepines freely cross the placenta and may accumulate in the fetus. Diazepam use in early pregnancy may be associated with a small risk of cleft lip and palate. Midazolam and lorazepam appear to cross the placenta to a lesser degree than diazepam, although the clinical significance of this is unknown. No data exist on the prolonged use of propofol in pregnancy, but it has been used as an induction agent for caesarean section. Congenital malformations have not been demonstrated with the use of narcotic analgesics such as morphine and fentanyl. The majority of non-depolarizing neuromuscular blocking agents cross the placenta, including pancuronium, vecuronium and atracurium, but transfer is unlikely to have clinical effects on the fetus in the short term. If sedative or paralytic agents are used in the pregnant woman, however, this information must be communicated to the neonatologist at the time of delivery, and the need for ventilatory support for the fetus should be anticipated.

The obese patient

Physiological changes

Obesity is defined as a body mass index of 30 to 34.9 kg/m^2, obesity class II as 34.9 to 39.9 kg/m^2 and extreme obesity as a body mass index >40 kg/m^2 [39]. Obesity has been linked to many other conditions such as diabetes, hypertension and dyslipidemia as well as vascular disease, malignancy and liver disease [40]. Patients are also more prone to several other conditions affecting ICU course, including venous thromboembolism, chronic obstructive pulmonary disease and sleep-disordered breathing (Table 2) [41]. Oxygen consumption is increased, and an unusually high proportion of this consumption is spent on the work of breathing even at rest [42]. Lung volumes are abnormal, with reduced expiratory reserve volume and a low maximum voluntary ventilation [43].

Table 2

Physiological effects and risks in the critically ill morbidly obese patient

Respiratory	Reduced lung volumes
	Atelectasis and ventilation–perfusion mismatch
	Increased work of breathing and oxygen consumption
	Obstructive airways disease (mechanical and asthma)
	Obstructive sleep apnea
	Obesity hypoventilation syndrome
Cardiovascular	Coronary artery disease
	Hypertension
	Systolic and diastolic left ventricular dysfunction
	Pulmonary arterial hypertension
	Obesity supine death syndrome
Other	Diabetes mellitus
	Increased risk of venous thromboembolism
	Increase risk of gastric acid aspiration
	Altered drug pharmacokinetics
	Difficult venous access
	Increased risk of renal failure
	Increased risk of pressure ulcers

The alveolar–arterial oxygen difference is also increased [44,45], suggesting ventilation–perfusion mismatch. The functional residual capacity is reduced in class II obesity and extreme obesity due to increased abdominal pressure [45]. Respiratory system compliance is markedly reduced, due to increased chest wall mass and limited diaphragmatic excursion.

The effects on lung volumes and compliance are exacerbated in the supine position. A condition called obesity supine death syndrome has been described, with sudden death occurring due to increased oxygen consumption and worsened hypoxemia on assuming the supine position, in a patient with a hyperactive, borderline hypoxic heart [46].

Airway management

Airway management should be undertaken by an experienced operator and should begin with a detailed assessment looking for features that may suggest difficulty in either intubation, ventilation or tracheostomy. Class II obesity in itself does not imply difficult intubation; as the standard tests taken together cannot reliably predict difficulty [47], however, a high index of suspicion is sensible. The American Society of Anesthesiologists recommends that a preplanned strategy is put in place, all equipment is checked prior to the procedure

and a back-up plan should always be prepared. Awake techniques may be required in some of these patients, especially if there is an increased risk of aspiration. This will usually involve a flexible bronchoscope, but newer rigid devices such as the Airtraq (Prodol Meditec, Las Arenas, Spain) [48] and the Glidescope (Verathon Inc., Bothell, WA, USA) have been used in awake patients. The UK Difficult Airway Society guidelines are highly recommended for a systematic approach to anticipated problems in specific circumstances [49].

Both the American Society of Anesthesiologists and the UK Difficult Airway Society suggest the laryngeal mask airway as their primary rescue therapy in a 'can't intubate, can't ventilate' scenario. If successful, some laryngeal mask airways can then be used as a conduit for a fiberoptic bronchoscope. There are also case reports of the use of the Combitube (Kendall-Sheridan Corporation, Argyle, NY, USA) [50,51], the intubating laryngeal mask [52] and the Proseal laryngeal mask (The Laryngeal Mask Company, Henley on Thames, UK) [53] – the latter two as a primary device [53,54]. These masks should only be employed by operators experienced in their use, and there remains a risk of aspiration, large ventilatory leak and laryngospasm with all of these devices.

Whatever technique is used, adequate preoxygenation is vital and, despite this intervention, obese patients will desaturate more rapidly than usual, due to the physiological changes described above. Preoxygenation in the sitting position may ameliorate this decline [55]. Proper positioning, such as elevation of the upper body with pillows, my also improve the view of the larynx and the ability to bag-mask ventilate if an asleep technique is used [46]. It should always be remembered that in an emergency it is oxygenation – not intubation – that is paramount, and bag-mask ventilation may be difficult and require three hands and the use of oral and nasopharyngeal airways.

Noninvasive ventilation

Noninvasive ventilation is established as a treatment for obesity hypoventilation syndrome [56] and for hypercapnic respiratory failure. The technique has been used successfully in treatment of class II obese patients requiring ventilatory assistance, but an absence of improvement in arterial blood gases within 2 hours should prompt invasive ventilation [57]. There are also reports of use of noninvasive ventilation to preoxygenate patients prior to intubation [58].

Invasive ventilation

The ARDSNet trial currently forms the basis for conventional ventilatory management in the ICU. This trial excluded patients who weighed more than 1 kg per centimeter of height [1]. This does not correlate simply with the body mass index but is likely to have excluded many class II obese patients and extreme obese patients. A secondary analysis of patients included in ARDSNet trials, however, identified over 200 obese patients (body mass index >30 kg/m^2) [59]. Similar outcome benefits of low tidal volumes were seen in this subgroup.

In order to overcome the pulmonary effects of increased abdominal pressure and reduced respiratory system compliance, higher levels of PEEP and plateau pressure may be required [45,46]. In the presence of decreased respiratory system compliance, higher plateau pressures may be necessary to achieve adequate tidal volume and may not be injurious, as the transpulmonary pressure is not increased. The Lung Open Ventilation Study used higher PEEP levels and a plateau pressure of up to 40 cmH_2O with an outcome similar to patients ventilated with the ARDSNet protocol, but obese patients were also excluded from this study [60]. In obese patients, the predicted body weight (based on height) should be used to calculate the tidal volume in order to prevent overdistention. The potential for development of intrinsic PEEP should always be considered and airflow limitation treated. Obese patients may have undiagnosed asthma or an exacerbation of existing asthma, precipitated by low lung volumes and other triggers of airway hyperresponsiveness accompanying obesity (reflux, dyslipidemia, diabetes and hypertension) [61].

There are limited data regarding other modes of mechanical ventilation such as high-frequency oscillatory ventilation in class II obese patients and extreme obese patients, but this mode provides the benefit of increased mean airway pressure and has been used safely in these patients in our institution.

Weaning

Weaning obese patients may be difficult due to the physiological changes described above. Obese patients are at higher risk of prolonged weaning and chronic ventilator dependence, and the postextubation course may be complicated by the presence of sleep apnea [41]. Noninvasive ventilation after extubation may reduce the incidence of reintubation in the obese patient [62]. Early tracheostomy may provide benefits in this patient group. Although most studies looking at outcomes of mechanical ventilation in this obese group have found an increased ICU length of stay, a recent meta-analysis showed there is no difference in mortality between obese and nonobese ICU patients [63]. For this reason, it is important that those caring for obese patients in the ICU are not unnecessarily pessimistic in their outlook, despite the known relationship between obesity and ill health.

Competing interests

The authors declare that they have no competing interests.

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