

RESPIRATION AND THE AIRWAY

Ventilation strategies for front of neck airway rescue: an *in silico* study

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Abstract

Background: During induction of general anaesthesia a ‘cannot intubate, cannot oxygenate’ (CICO) situation can arise, leading to severe hypoxaemia. Evidence is scarce to guide ventilation strategies for small-bore emergency front of neck airways that ensure effective oxygenation without risking lung damage and cardiovascular depression.

Methods: Fifty virtual subjects were configured using a high-fidelity computational model of the cardiovascular and pulmonary systems. Each subject breathed 100% oxygen for 3 min and then became apnoeic, with an obstructed upper airway. When arterial haemoglobin oxygen saturation reached 40%, front of neck airway access was simulated with various configurations. We examined the effect of several ventilation strategies on re-oxygenation, pulmonary pressures, cardiovascular function, and oxygen delivery.

Results: Re-oxygenation was achieved in all ventilation strategies. Smaller airway configurations led to dynamic hyperinflation for a wide range of ventilation strategies. This effect was absent in airways with larger internal diameter (≥ 3 mm). Intrapulmonary pressures increased quickly to supra-physiological values with the smallest airways, resulting in pronounced cardio-circulatory depression (cardiac output < 3 L min⁻¹ and mean arterial pressure < 60 mm Hg), impeding oxygen delivery (< 600 ml min⁻¹). Limiting tidal volume (≤ 200 ml) and ventilatory frequency (≤ 8 bpm) for smaller diameter cannulas reduced dynamic hyperinflation and gas trapping, preventing cardiovascular depression.

Conclusions: Dynamic hyperinflation can be demonstrated for a wide range of front of neck airway cannulae when the upper airway is obstructed. When using small-bore cannulae in a CICO situation, ventilation strategies should be chosen that prevent gas trapping to prevent severe adverse events including cardio-circulatory depression.

Keywords: airway management; airway obstruction; apnoea; cannot intubate; cannot oxygenate; front of neck airway; oxygenation simulation

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Editor's key points

- In cannot intubate, cannot oxygenate scenarios, emergency front of neck airway access can be life-saving in the face of hypoxaemia.
- Optimal ventilation strategies are not clear for small-bore emergency front of neck airways that ensure effective oxygenation without risking lung damage and cardiovascular depression.
- This computational simulation study examined the effects of various ventilation strategies on re-oxygenation, pulmonary pressures, cardiovascular function, and oxygen delivery in 50 virtual subjects.
- Re-oxygenation was achieved in all ventilation strategies, but smaller airway configurations led to dynamic hyperinflation.
- When using small-bore cannulae for emergency front of neck access, ventilation strategies should be chosen that prevent gas trapping and cardiovascular depression.

During a 'cannot intubate, cannot oxygenate' (CICO) situation, the anaesthetist is unable to establish a patent airway through manipulation or conventional airway adjuncts and devices. In the apnoeic patient, failure to manage the airway effectively for an extended period can result in severe hypoxaemia, progressing potentially to organ injury and death. In clinical practice, this scenario is rare: approximately 1:12 500 cases of general anaesthesia and 3–8:1000 tracheal intubation attempts in the emergency department.^{1,2}

Guidance on airway management generally includes emergency tracheostomy/cricothyroidotomy as a rescue intervention.^{2,3} Despite evidence that the insertion of larger-diameter airway devices is safer in these settings, the anatomical topography of insertion below the cricoid cartilage and the invasiveness and urgency of the procedure necessary to establish the airway often leads to use of small diameter rescue airway cannulas and tubes. Small-diameter airways produce high airflow resistances.⁴ Owing to incomplete exhalation, dynamic hyperinflation (commonly referred to as 'gas trapping' or 'stacking') can occur.^{5,6} This results in accumulation of intrathoracic gas volume, with increasing intrathoracic pressure, leading to cardiovascular depression and pulmonary injury. Because of known complications associated with over-distension and excessive ventilatory pressures,^{1,7} concerns have been raised regarding the safety of ventilation through small airway devices when upper airway obstruction impedes the escape of excess gas from the lungs.^{1,8–14} Clinically, this scenario can occur where a tumour, swelling, haematoma, or other causes of airway crowding leads to obliteration of the periglottic airspace; the need for urgent front of neck access is more likely to arise in these settings.

Because CICO scenarios are rare, it is difficult to perform structured clinical research on optimal ventilation strategies for these situations. As a result, there is scarce evidence to provide specific guidance on how to achieve rapid re-oxygenation without risking lung damage and cardiovascular depression caused by pulmonary over-distension. Animal studies,^{15–17} mechanical lung models,^{4,18–20} and clinical data¹³

have been used to investigate the mechanics of ventilation via small airways and identify possible pitfalls in achieving safe oxygenation in this extreme situation. However, systematic clinical investigations to assess this scenario would be difficult to recruit and ethically challenging to perform. In addition, mechanical models are limited in their capacity to reproduce human physiology. Large animal models present ethical limitations and intricate differences in their physiology, making it difficult to draw conclusions for extreme cases.

To address these challenges, high-fidelity computer simulation of pathophysiological states and specific clinical interventions can be a powerful tool to inform future investigations or practice while circumventing the need to put patients at risk or to use animal models for research.

We evaluated strategies for achieving re-oxygenation after airway rescue (i.e. re-opening the airway through a front of neck access with ongoing upper airway obstruction) using computer simulation of the integrated respiratory and cardiovascular systems. In particular, we investigated which combinations of tidal ventilation modalities assured safe intrapulmonary pressures when ventilating via narrow bore, front of neck airway devices.

Methods**Computational model**

This study uses the Interdisciplinary Collaboration in Systems Medicine (ICSM) simulation suite based upon the Nottingham Physiology Simulator.^{21–24} The ICSM computer simulation is a highly integrated high-fidelity computational model of a number of organ systems. The model has been extensively validated, including in the simulation of apnoea and hypoxaemia in adults.^{25–27} Recently, the ICSM simulation suite has been extended in order to include the physiological mechanisms underlying apnoeic oxygenation²⁸; we previously used the updated model for an investigation of the influence of supraglottic oxygen supplementation on the effectiveness of airway rescue.²⁹ A detailed description of the ICSM simulation suite is provided in the Supplementary material.

Virtual subjects and protocol

A cohort of 50 *in silico* subjects was created, as described,²⁹ to represent the spectrum of physiology in an otherwise healthy population. These subjects were developed by establishing credible physiological ranges for key model parameters based on data reported in the literature. The virtual subjects were generated using randomly permuted configurations of the parameters within these ranges (Supplementary Table S1).

For all virtual subjects, we simulated rescue ventilation with five different airway sizes (varying internal diameter [ID] and length based on real-world medical devices), as follows:

- A. ID 1.8 mm, length 6.3 cm, representing the Ravussin 13G cannula (VBM Medical, GmbH, Sulz, Germany)
- B. ID 2.0 mm, length 7.5 cm, representing the Emergency Transtracheal Airway Catheter (Cook Medical Inc., Bloomington, IN, USA)
- C. ID 3.0 mm, length 5.8 cm, representing Patil's Airway³⁰
- D. ID 4.0 mm, length 6.3 cm, representing the Quicktrach cannula 4 mm ID (VBM Medical, GmbH)

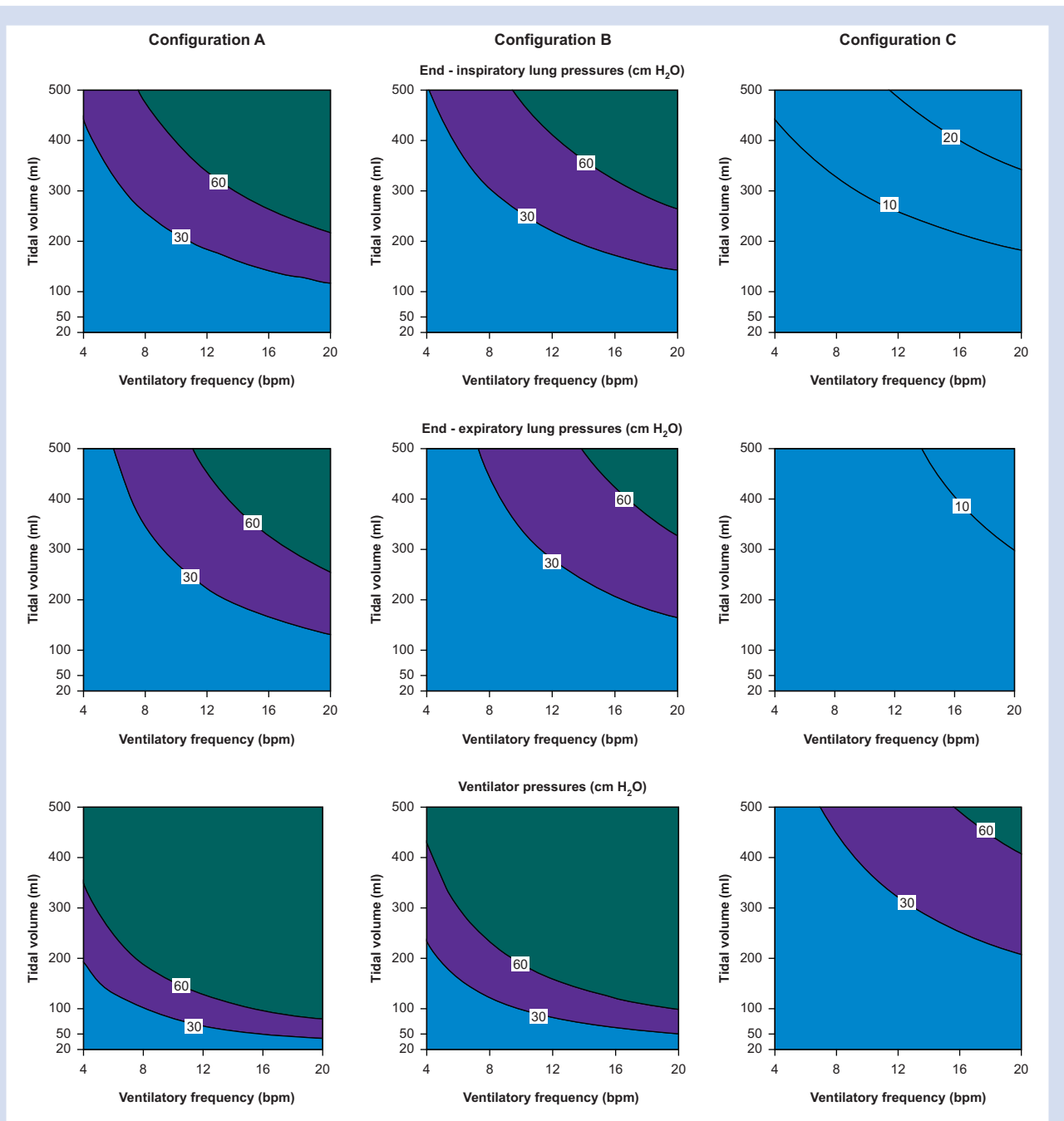


Fig. 1. Airway pressures calculated over the cohort of 50 *in silico* subjects. Mean values are shown for end-expiratory lung pressure, end-inspiratory lung pressure and ventilator pressure (bottom panels) for airway configurations A (inner diameter 1.8 mm, length 6.3 cm), B (inner diameter 2.0 mm, length 7.5 cm), and C (inner diameter 3.0 mm, length 5.8 cm). Standard deviations have not been reported for graphical reasons and because they were not significant. Levels of pressures were represented with different colour coding; blue represented ‘safe’ pressures (<30 cm H₂O),³² purple represents pressures 30–60 cm H₂O, and green represented high pressures (>60 cm H₂O). These thresholds have been chosen because of clinical consideration and studies showing a proportional relationship between driving pressures and potential patient harm in other patient cohorts³³ where a plateau pressure >30 cm H₂O might be acceptable under certain conditions.

E. ID 6.0 mm, length 9.0 cm, representing the Melker cannula 6 mm ID (Cook Medical Inc.)

The 50 virtual subjects underwent pulmonary denitrogenation (pre-oxygenation) during resting, tidal breathing with

an inspired oxygen fraction (FiO₂) of 100% for 3 min. Induction of general anaesthesia was simulated, with apnoea and upper airway obstruction commencing simultaneously. Apnoea continued until the arterial oxygen saturation (SaO₂) reached 40%. At that time, the airway was opened (via front of neck

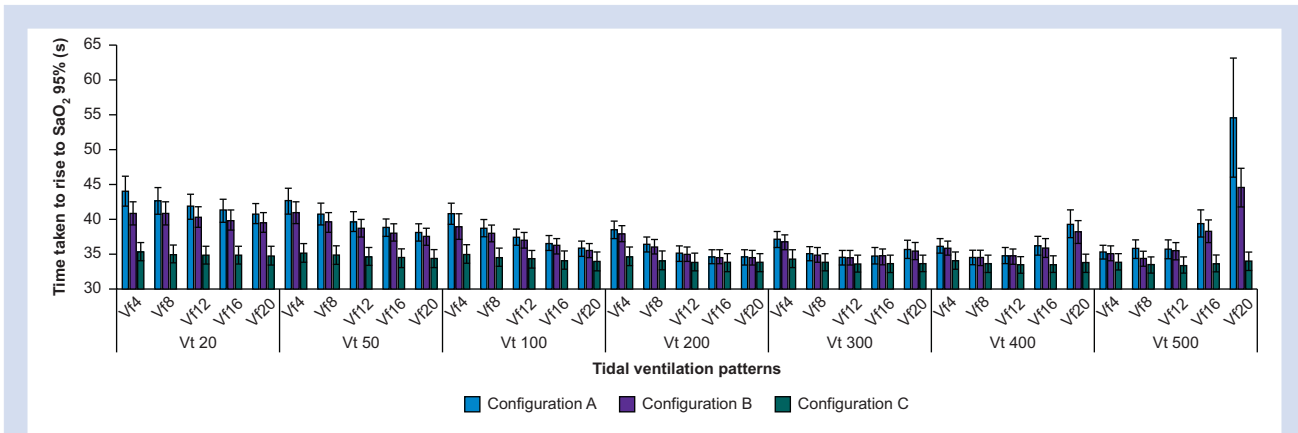


Fig. 2. Time taken to achieve arterial haemoglobin oxygen saturation (SaO_2) of 95% in the small-bore airway configurations (A, B, and C) during all patterns of tidal ventilation studied. Vf, ventilatory frequency (bpm); Vt, tidal volume (ml).

access with each of the above device permutations) and various patterns of tidal ventilation with 100% oxygen were provided for 5 min.

The ventilation patterns comprised:

- Tidal volumes: 20, 50, 100, 200, 300, 400, and 500 ml
- Ventilatory frequencies: 4, 8, 12, 16, and 20 bpm
- Inspiratory/expiratory ratio (I/E): 1:2

The above patterns of tidal ventilation, in every combination, were repeated in all small-bore configurations considered (devices A, B, and C). For the large-bore configurations (devices D and E) gas trapping was highly unlikely to occur.³¹ As our initial results indicated that gas trapping was indeed absent for all combinations of tidal volumes and ventilatory frequency in configurations D and E, the maximum tidal ventilation with a tidal volume of 500 ml and ventilatory frequency of 20 bpm was simulated as a control with the full bank of virtual subjects.

In a second step, we assessed safe intrapulmonary pressures for a wider range of airway size. To study the relationship between airway length and diameter, we ran a separate protocol with generic airway configurations (lengths 4–10 cm and IDs 1–4 mm). A grid of these configurations was assessed for resulting ventilator and airway pressures during ventilation with a tidal volume of 100 ml and ventilatory frequency of 8 bpm.

A total of 7100 individual simulations were conducted to examine all the above scenarios.

Data collection

The following simulation outputs were recorded every 5 ms: end-inspiratory and end-expiratory lung pressures (calculated as the volume-weighted mean of alveolar compartmental pressures), ventilator pressure, lung volume, arterial haemoglobin oxygen saturation (SaO_2), arterial partial pressure of oxygen (PaO_2), arterial partial pressure of carbon dioxide (PaCO_2), arterial oxygen delivery (DO_2), cardiac output (CO), and mean arterial pressure (MAP). Data are presented as mean (standard deviation).

Model simulations were executed on a 64-bit Intel Core i7 3.7 GHz Windows 10 personal computer, running Matlab version R2018a.v9 (MathWorks Inc., Natick, MA, USA).

Results

Figure 1 shows the mean values, calculated over the cohort of subjects, of end-inspiratory and end-expiratory lung pressures, and ventilator pressures for the small-bore configurations (A, B, C), during the application of the various patterns of tidal ventilation examined. When tidal volume was ≤ 200 ml and ventilator frequency was ≤ 8 bpm, the mean end-inspiratory lung pressure remained 'safe' (i.e. less than 30 cm H_2O) in all airway configurations. In the smallest configuration (A) when tidal volume was 200 ml and ventilator frequency was 8 bpm, although the ventilator pressure increased to 63.2 (0.7) cm H_2O , end-inspiratory lung pressure was 21.8 (0.8) cm H_2O . For larger tidal volumes (i.e. 300 and 400 ml) only a ventilatory frequency of 4 bpm assured a mean end-inspiratory lung pressure < 30 cm H_2O in all airway configurations. The largest tidal volume studied (500 ml) with a ventilatory frequency of 4 bpm resulted in a mean end-inspiratory lung pressure < 30 cm H_2O in all airway configurations except in A.

The oxygenation goal after airway rescue of SaO_2 95% was achieved in < 1 min for all patterns of ventilation studied, even when small tidal volumes and ventilatory frequencies were used (Fig. 2). In the smallest airway configurations (A and B), using larger tidal volumes ≥ 300 ml with ventilatory frequency 20 bpm caused re-oxygenation to be slower, whereas in airway configuration C the time to reach SaO_2 95% was similar in all tidal ventilation patterns used.

In the large-bore configurations (D and E), despite a large minute ventilation (500 ml \times 20 bpm), no excessive intrapulmonary pressure was observed.

Example time courses of lung volume, PaO_2 and SaO_2 in one representative subject under varying ventilation strategies and airway sizes are illustrated in Figure 3. The different experimental phases of pre-oxygenation, obstructed apnoea, and ventilation via the rescue airway are demonstrated. Right and left panels show courses with the provision of the maximum (500 ml \times 20 bpm) and minimum (20 ml \times 4 bpm) patterns of the tidal ventilation, respectively. The middle panel shows the pattern of 200 ml \times 8 bpm that still assured end-inspiratory pressure < 30 cm H_2O in all airway configurations. When the maximum tidal ventilation pattern (500 ml \times 20 bpm) was applied, lung volumes showed a pronounced

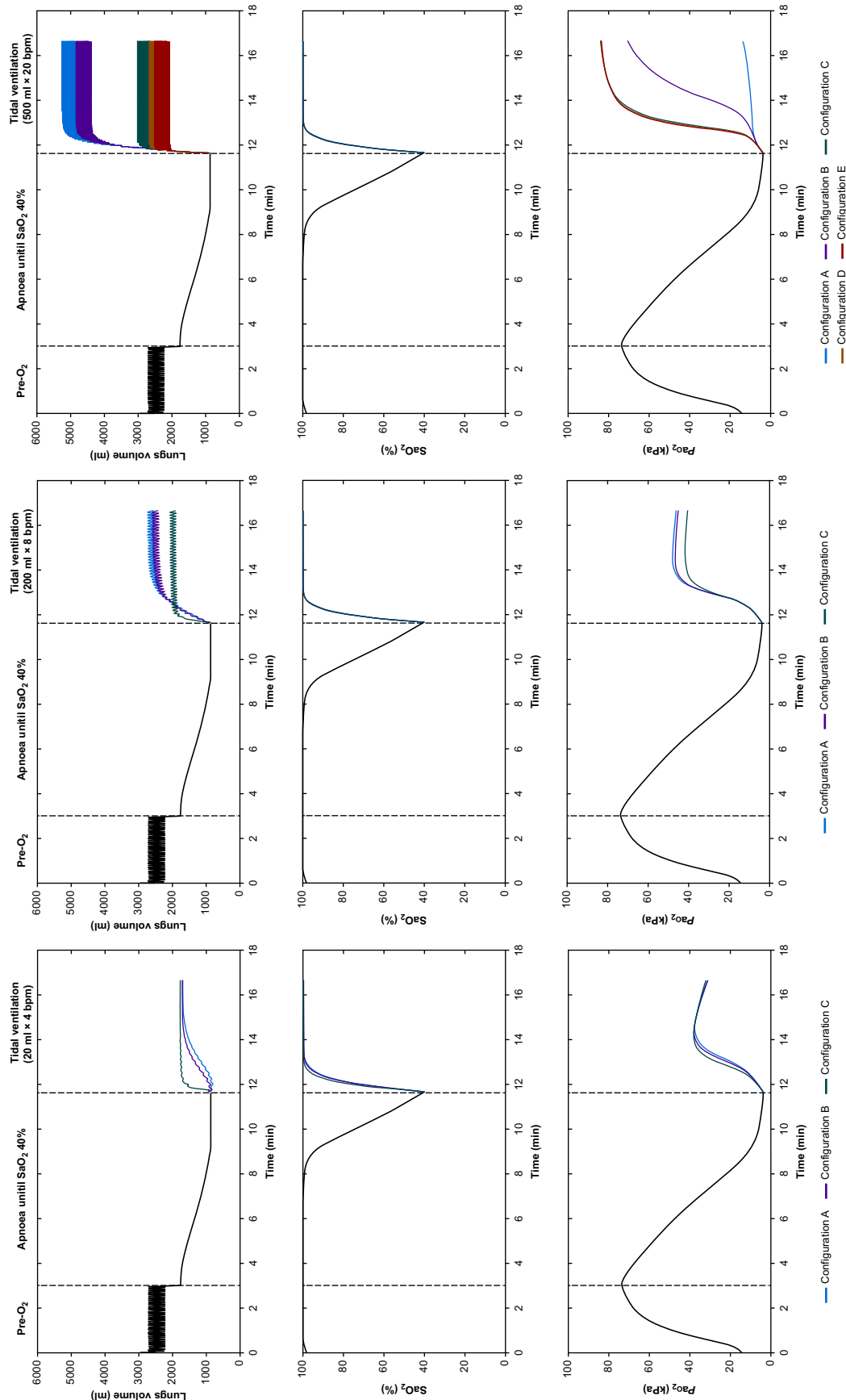


Fig. 3. Time-course of lung volumes, PaO₂ and SaO₂, during pre-oxygenation, apnoea, and airway opening with different provisions of tidal ventilation in a representative subject; right panel: tidal volume 20 ml and ventilatory frequency 4 bpm; middle panel: tidal volume 200 ml and ventilatory frequency 8 bpm; left panel: tidal volume 500 ml and ventilatory frequency 20 bpm.

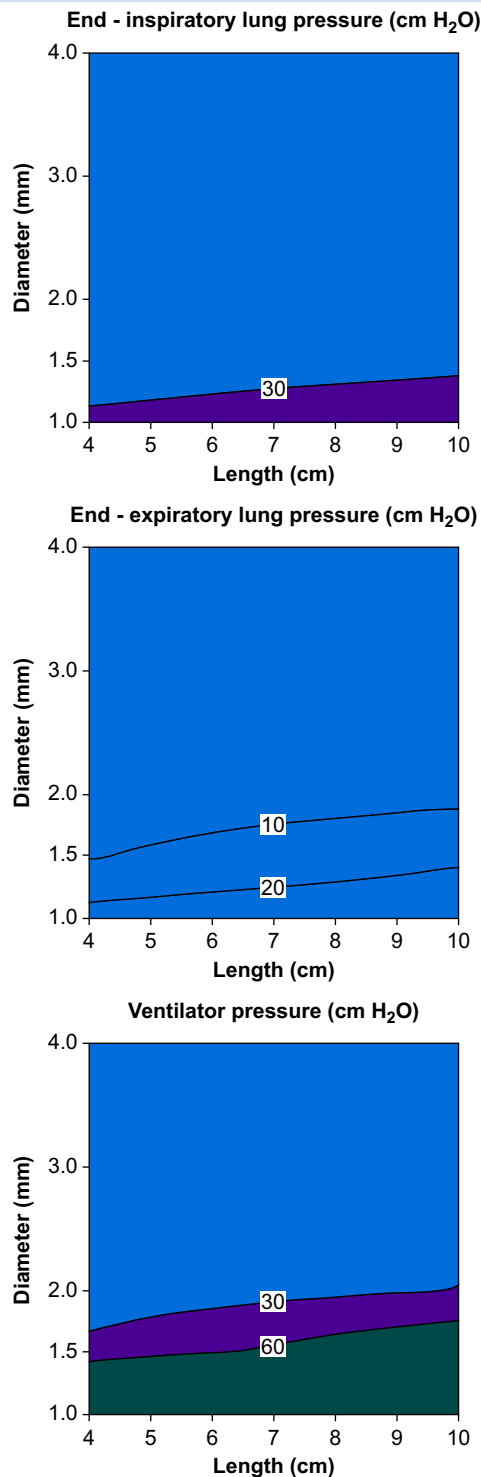


Fig. 4. End-inspiratory lung pressure, end-expiratory lung pressure and ventilator pressure for various airway lengths and diameters, with tidal volume 100 ml and ventilatory frequency 8 bpm. Blue areas: pressure <30 cm H₂O; purple areas: pressures 30–60 cm H₂O and green areas: pressures >60 cm H₂O.

increase above functional residual capacity only in the smallest-bore airway configurations (A and B), indicating that for airway configurations with a diameter <3 mm, smaller tidal ventilation patterns should be used to avoid gas trapping and over-distension of the lung.

In all configurations, re-oxygenation was achieved even with the minimum pattern of tidal ventilation. Comparable time courses occurred in all modelled subjects.

Figure 4 shows the mean values, calculated over the cohort of the subjects, of end-inspiratory lung pressures (top panel), end-expiratory lung pressures (middle panel), and ventilator pressures (bottom panel) for a variation of airway lengths (4–10 cm) and diameters (1–4 mm) with a fixed minute ventilation (tidal volume, 100 ml; ventilatory frequency, 8 bpm). When airway diameter was 2 mm or more, end-inspiratory lung pressure remained <30 cm H₂O for all airway lengths.

Haemodynamic parameters (i.e. DO₂, CO, and MAP), calculated during the 5 min of the tidal ventilation, for the small-bore airway configurations (A, B and C) are reported in Figure 5. DO₂ was >800 ml min⁻¹ in configuration C for all patterns of tidal ventilation, whereas it dropped to 600 ml min⁻¹ in configurations A and B as tidal volume and ventilatory frequency increased with a correspondent decrease in CO <3 L min⁻¹ and in MAP <60 mm Hg. In particular, in configuration A, DO₂ dropped to 600 ml min⁻¹ for tidal volume of 300 ml and ventilatory frequencies of 16 and 20 bpm, and for tidal volumes of 400 and 500 ml and ventilatory frequencies of 12, 16, and 20 bpm. In configuration B, DO₂ dropped for tidal volume of 300 ml and ventilatory frequency of 20 bpm, and for tidal volumes of 400 and 500 ml and ventilatory frequencies of 16 and 20 bpm.

In the large-bore configurations (D and E), DO₂, CO, and MAP remained >900 ml min⁻¹, 4.3 L min⁻¹, and 85 mm Hg, respectively, in all configurations.

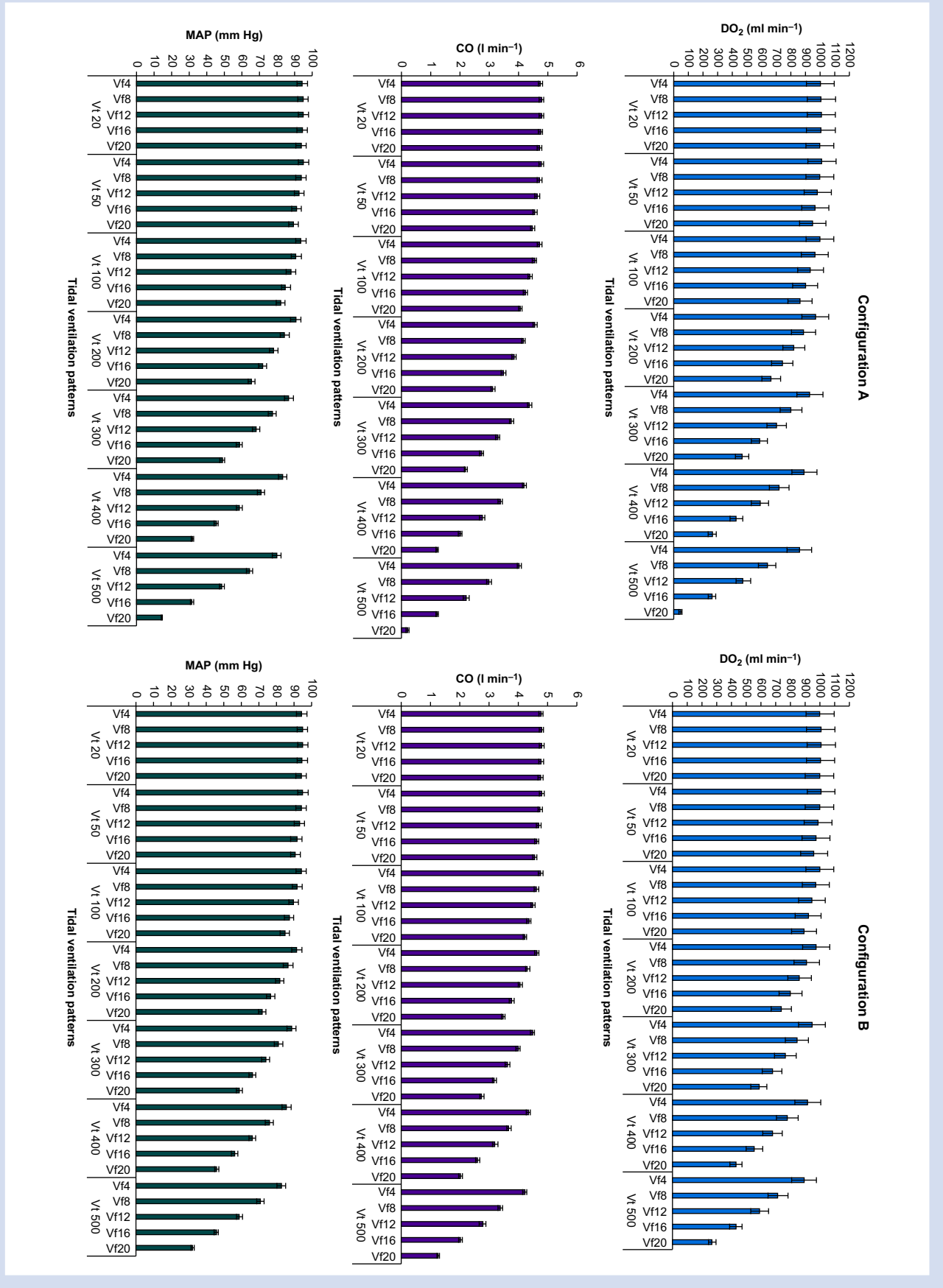
As shown in Figure 6, although there was a significant increase in CO₂ when small tidal volumes were applied (i.e. ≤200 ml), oxygenation was assured in all airway configurations and for any pattern of ventilation considered at the end of the 5 min rescue ventilation period.

Discussion

In this computational modelling study, we simulated a CICO emergency scenario in a robust and representative cohort of 50 virtual subjects. Both choosing larger airway diameters for front of neck access, or limiting minute ventilation for smaller diameter cannulae, reduced gas trapping. *In vivo*, dynamic lung hyperinflation leads to alveolar over-distension and consequent barotrauma, which are common complications of this rescue approach.^{1,2}

Our study provides new evidence that, in the case of front of neck airway rescue with an obstructed upper airway, larger airway configurations (ID ≥3 mm) do not lead to increased airway pressures, gas trapping, or excessive ventilation pressures (<30 cm H₂O).³⁴ For ID <3 mm, gas trapping becomes more pronounced as minute ventilation increases. In configurations A and B, ventilation caused excessive intrapulmonary pressures because of over-distension. This increase in lung volumes is a result of dynamic hyperinflation in the context of the use of small-bore cannulae, but not in the larger-gauge airway configurations. This behaviour reflects clinical experience, in which reports from transtracheal jet ventilation note barotrauma as a known adverse event associated with front of

Fig. 5. Arterial oxygen delivery (DO₂), cardiac output (CO), and mean arterial pressure (MAP) in the small-bore configurations (A, B, and C) at the end of tidal ventilation provision. Before opening of the obstructed airway (at the end of apnoea), DO₂, CO, and MAP were 227.2 (22.9) ml min⁻¹, 2.7 (0.1) L min⁻¹, and 57.2 (2.7) mm Hg, respectively. Vf, ventilatory frequency (bpm); Vt, tidal volume (ml).



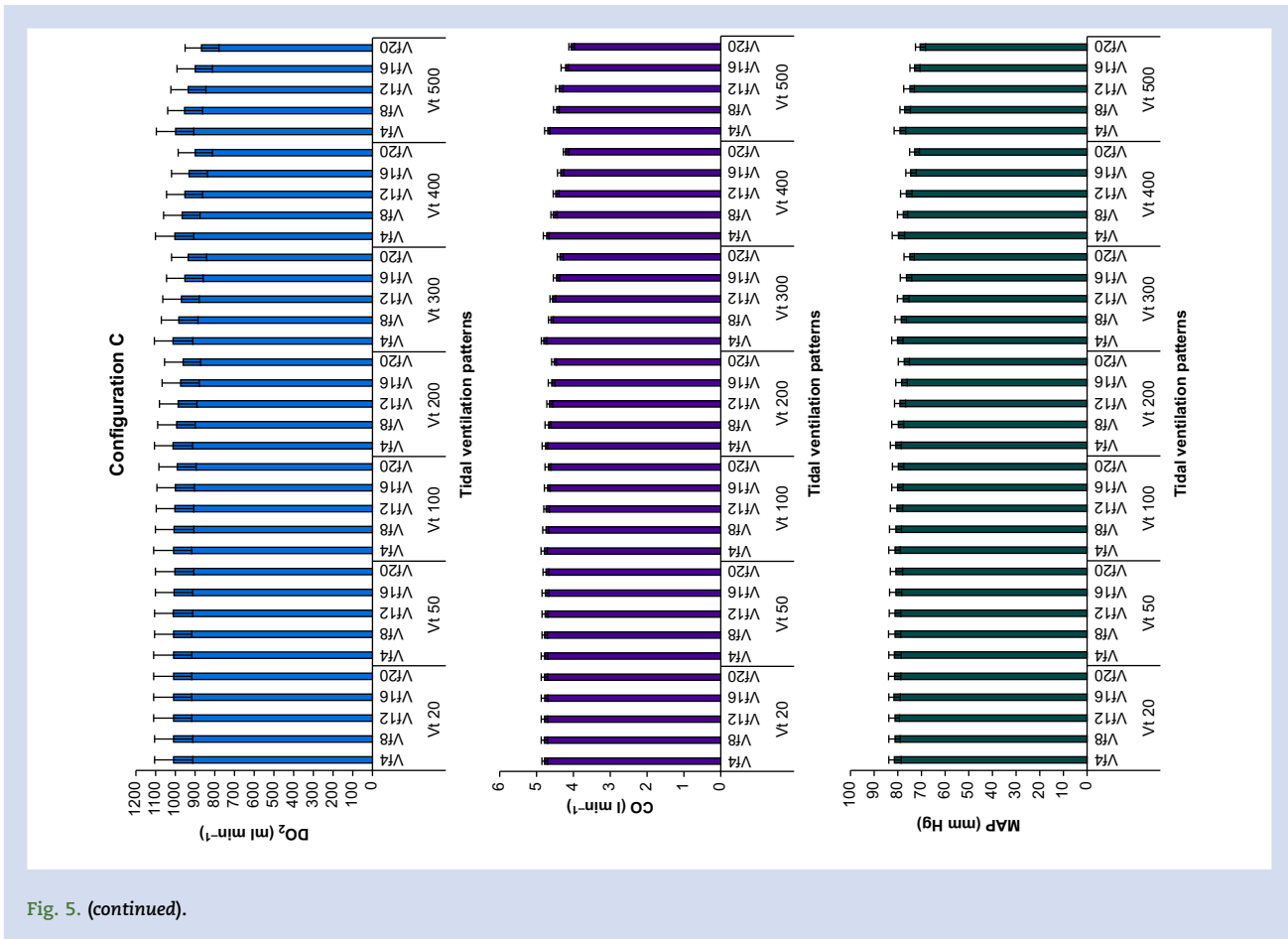


Fig. 5. (continued).

neck access.^{8–12} The gas trapping behaviour in the current study matches the findings of previous studies in mechanical lung models that investigated delayed expiration because of smaller diameters.⁴ The increase in intrapulmonary pressure resulted in a decrease in mean arterial pressure, CO, and oxygen delivery (Fig. 5). Particularly for the maximum minute ventilation scenarios in configurations A and B, severe cardiovascular depression was observed. This is in agreement with clinical experience where excessive intrapulmonary pressures can precipitate drastic cardio-circulatory compromise.

High ventilator pressures did not necessarily translate to high intrapulmonary pressures (e.g. in configuration A), using a tidal volume of 200 ml and a ventilatory frequency of 8 bpm, ventilatory pressure was 63.2 (0.7) cm H₂O whereas end-inspiratory lung pressure was 21.8 (0.8) cm H₂O. Other authors quote pre-systemic pressures in bar or psi. For consistency of units within the simulation, we used cm H₂O. Ward and colleagues³⁵ showed that in a canine model a pre-systemic pressure translating to 3163 cm H₂O resulted in an airway pressure of only 22 cm H₂O with 80% upper airway obstruction. Biro and colleagues³⁶ made similar observations in a clinical study of 10 patients undergoing jet ventilation. In their case, driving pressures of 2099 and 3099 cm H₂O resulted in a peak inspiratory pressure of 12.9 and 17.7 cm H₂O in complete upper airway obstruction, respectively. The significantly greater discrepancy between the driving pressure and

the pressure delivered to the airway could be explained by different airflow dynamics. In the work of Biro and colleagues,³⁶ the jet ventilation port of a rigid bronchoscope was used, the geometry of which was not specified. However, owing to the tracheobronchial geometry of an adult patient, significantly longer tubing can be expected. In Ward and colleagues³⁵ work, a longer 13G cannula was used that has a higher resistance. A linear relationship between cannula length and resistance can be inferred from Figure 4. Incomplete airway obstruction in the study of Ward and colleagues would also have allowed passive gas escape. This was shown by Sasano and colleagues¹⁸ to prevent build-up of airway pressures on a mechanical lung model.

Considering that safe oxygenation was possible via passive provision of high FiO₂,²⁹ it is not surprising that even with minimal minute ventilation, safe oxygenation (SaO₂ 95%)³⁷ was achieved. As CO₂ removal is efficiently achieved with higher minute ventilation (Fig. 6) pursuing this goal would likely result in more pronounced gas trapping in smaller airway configurations. As re-oxygenation is the primary goal in an emergency front of neck access scenario, a volume-restricted ventilation strategy using FiO₂ of 100% might be safer in small-bore cannulas, with tolerance of CO₂ accumulation.

The main limitation of our modelling approach is the inability to account for variations of airflow dynamics, such as dislodgement of the cannula/device, misplacement into surrounding soft tissue or dynamic fluctuations of airway

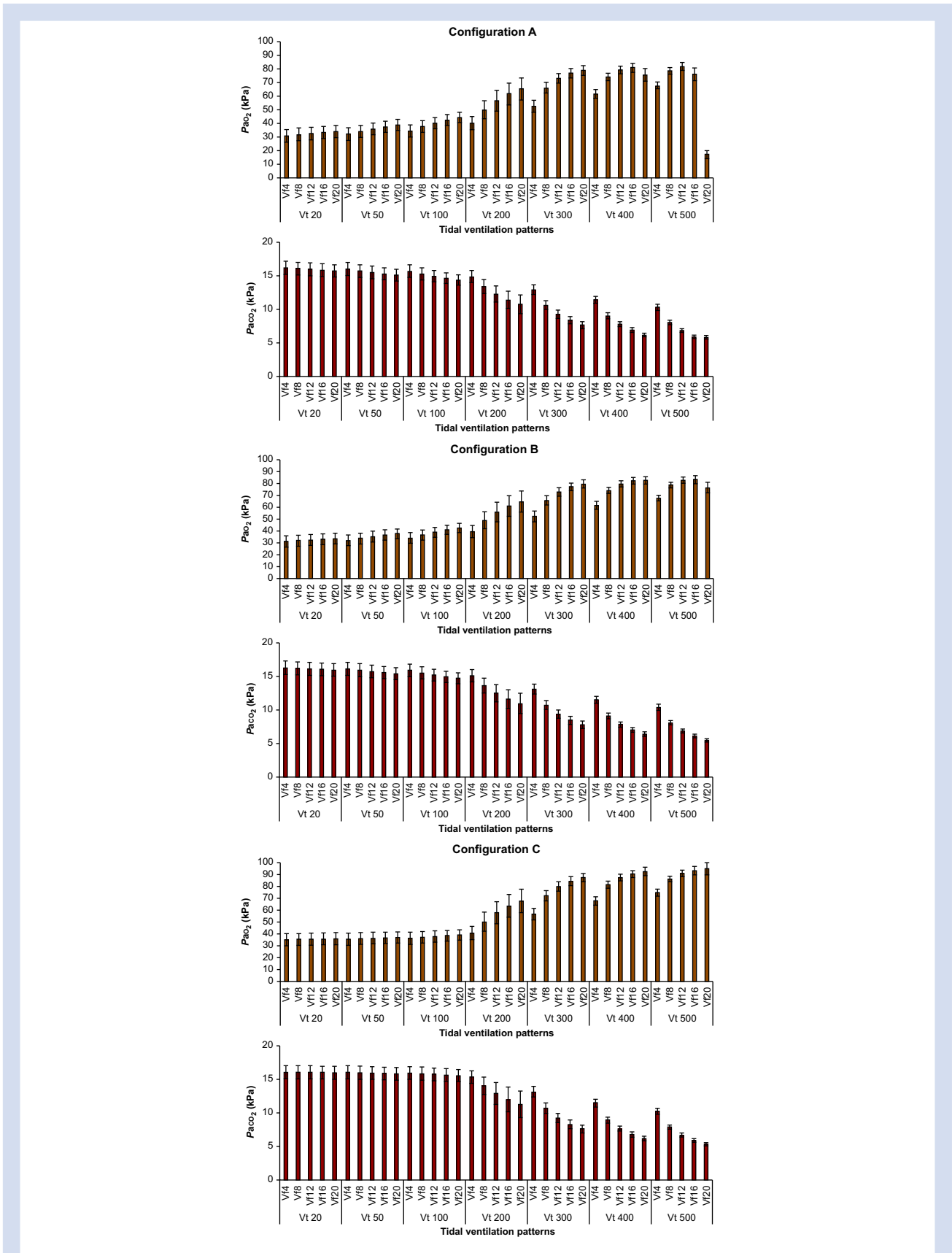


Fig. 6. Arterial partial pressure of oxygen (P_{aO_2}) and carbon dioxide (P_{aCO_2}) calculated in the small-bore configurations (A, B, and C) at the end of tidal ventilation provision. Before opening of the obstructed airway (at end of apnoea), P_{aO_2} and P_{aCO_2} were 3.8 (0.1) kPa and 11.1 (1) kPa, respectively. Vf, ventilatory frequency (bpm); Vt, tidal volume (ml).

resistance, such as bronchial secretions that could fully or partially occlude the small-bore devices. Similarly, we cannot simulate geometry of the airway devices as curvature or kinks might contribute to airflow resistance.³⁸ Also, the orientation of the airway device within the trachea cannot be simulated. It is difficult to gauge the effect these variations might have on the safety evaluation of the tested ventilation strategies. Furthermore, the simulated patients are assumed to be free of cardiovascular morbidity; the effect of the more extreme intrathoracic pressures on pre-existing cardiac disease cannot be derived from our investigation. The simulated complete upper airway obstruction is a specific scenario that might clinically not be as frequent as partial airway patency, allowing some escape of gas through a largely obstructed upper airway.

Unlike previous work,²⁹ we used 50 virtual subjects instead of 100, because in this study the protocol considered a very large number of simulations owing to the large number of permutations of rescue ventilation strategies. However, as the virtual subjects were generated using wide-ranging, random configurations of the key model parameters, 50 virtual subjects should be considered suitable and adequate to describe a wide spectrum of pathophysiology. Similarly, concessions were made in the simulated combinations of tidal volume and ventilatory frequency to deal with limitations in computational power. The permutations presented cover a wide range of clinically feasible ventilation targets reaching moderately high minute ventilation, and volumes less than series dead-space volume. Simulations were conducted on a desktop PC; use of high-performance computing would allow for wider coverage of potential treatment and subject permutations.

Future studies should investigate more extensive variations in the I/E ratio. Additionally, adaptation of the model allowing active inhalation and exhalation^{15–17,39} could inform the discussion about devices that facilitate this type of ventilation, potentially reducing gas trapping. Finally, it will be useful to study partial (and absent) upper airway obstruction while simulating small-bore airway ventilation, in particular the dynamic venting of pressure from the lungs against an increased airway resistance from partial obstruction. Simulation of incomplete upper airway obstruction will most likely affect the tendency for gas trapping, the efficiency of ventilation/oxygenation and of CO₂ removal, as suggested previously.⁴⁰

Author's contributions

Design of study: ML, CN, AD, JGH

Simulation runs: ML

Interpretation of data: ML, CN, AD, DGB, JGH

Writing and final approval of manuscript: ML, CN, AD, DGB, JGH.

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Declarations of interest

JGH is associate editor-in-chief of the *British Journal of Anaesthesia*. JGH accepts fees for the provision of advice to the police,

crown prosecution service, coroners and solicitors. The other authors have no conflicts to declare.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.bja.2021.01.030>.

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