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Application of pre-hospital continuous positive airway pressure in hypovolaemic shock: A modelling study

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Pre-hospital continuous positive airway pressure after blast lung injury and hypovolaemic shock: a modelling study

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Keywords: Primary blast lung injury, Continuous positive airway pressure, Hypovolaemic shock, mathematical modelling, Computer simulation.

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EDITORS KEYPOINTS

- In non-traumatic respiratory failure, prehospital application of continuous positive airway pressure (CPAP) reduces the need for intubation.

- The authors modelled whether prehospital CPAP may also be beneficial following primary blast lung injury accompanied by haemorrhage.

- The modelling was validated using physiological data obtained from a porcine model of blast lung injury accompanied by haemorrhage.

- Modelling demonstrated that ambient air CPAP improved PaO_2 even with severe haemorrhagic shock, unless 10-15cmH_2O was applied following severe blast injury.

- This modelling study suggests that the clinical benefits of prehospital ambient air CPAP may extend to mass casualty scenarios where blast lung injury is common.

Abstract

Background: In non-traumatic respiratory failure, prehospital application of continuous positive airway pressure (CPAP) reduces the need for intubation. Primary blast lung injury accompanied by haemorrhagic shock is common after mass casualty incidents. We hypothesised that prehospital CPAP is also beneficial following primary blast lung injury accompanied by haemorrhagic shock.

Methods: We performed a computer-based simulation of the cardio-pulmonary response to PBLI followed by haemorrhage, calibrated from published controlled porcine experiments exploring blast injury and haemorrhagic shock. The effect of different CPAP levels was simulated in three *in silico* patients who had sustained mild, moderate or severe primary blast lung injury (10%, 25%, 50% contusion of the total lung) plus haemorrhagic shock. The primary outcome was arterial partial pressure of oxygen (PaO_2) at the end of each simulation.

Results: In mild blast lung injury, 5cmH_2O ambient-air CPAP increased PaO_2 from 10.6 to 12.6 kPa. Higher CPAP did not further improve PaO_2. In moderate blast lung injury, 10cmH_2O CPAP produced a larger increase in PaO_2 (from 8.5 to 11.1 kPa), but 15cmH_2O CPAP produced no further benefit. In severe blast lung injury, 5cmH_2O CPAP increased PaO_2 from 4.06 to 8.39 kPa. Further increasing CPAP to 10-15cmH_2O reduced PaO_2 (7.99 and 7.90 kPa, respectively) due to haemodynamic impairment resulting from increased intra-thoracic pressures.

Conclusions: Our modelling study suggests that ambient air 5cmH_2O CPAP may benefit casualties suffering from blast lung injury, even with severe haemorrhagic shock. However, higher CPAP levels beyond 10cmH_2O after severe lung injury reduced oxygen delivery due to haemodynamic impairment.

Introduction

The application of pre-hospital continuous positive airway pressure (CPAP) for the emergency treatment of acute respiratory failure (ARF) is increasingly used by emergency medical services.[1] In non-traumatic ARF, prehospital application CPAP via a tight-fitting mask reduces respiratory rate and the need for subsequent intubation.[2-4] Additionally, porcine data suggests the potential for decreased mortality, decreased pulmonary oedema, and increased arterial oxygen saturation following toxic lung injury when ambient-air CPAP is administered shortly after injury.[5] Computerised modelling of blast lung injury suggests that the use of ambient-air CPAP improves arterial oxygenation, increases gas exchange and reduces respiratory rate and pulmonary oedema.[6] This study assumes that CPAP in the range used (between 5–10 cmH_2O) will not exacerbate pre-existing pneumothoraces in spontaneously breathing individuals, in accordance with current published guidelines.[7] The authors of both studies supported the use of ambient-air CPAP as a first-aid or self-aid measure following such injury patterns.

The combination of shock and blast lung injury is particularly relevant to the pre-hospital management of casualties in a mass casualty scenario. Before application CPAP under such circumstances, the effect of raising intra-thoracic pressure in casualties that are haemodynamically compromised due to blood loss must be determined to confirm efficacy and safety. However, the design and implementation of randomised controlled trials to answer these questions during mass casualty scenarios is unlikely to be feasible. In this study, we use an existing computerised model of blast lung injury and introduce the capacity to replicate haemorrhagic shock. We then simulate three casualties, each with identical shock state but with increasing degrees of blast lung injury. We assume that the casualties do not

have access to pressurised oxygen in the pre-hospital phase but are able to self-administer or 'buddy-administer' first-aid CPAP masks. Each casualty is then exposed to different levels of ambient-air CPAP, and the subsequent effects on oxygenation and haemodynamics are quantified.

Methods

Study design

This was a computer simulation study, performed on a 64-bit Intel Core i7 3.7 GHz personal computer running Matlab (R2020a).

In-silico model of blast lung injury

The primary blast lung injury simulator employed in this study consists of a bespoke computational model that can represent mechanical and/or spontaneous ventilation, [11-13] trauma induced acute lung injury, [14,15] and ventilator induced lung injury (VILI).[16-18] The pulmonary model comprises of conducting airways and a respiratory zone of 100 parallel alveolar compartments, with a related set of parameters accounting for alveolar compliance, as well as airway and peri-alveolar vascular resistances. The integrated cardiovascular model consists of 19 variably compliant compartments, with pulsatile blood flow and ventilation affected trans-alveolar blood flow. All components are built from mass-conserving functions and solved as algebraic equations, obtained or approximated from the published literature, experimental data and clinical observations. Equations are solved iteratively in series, with each iteration representing a physiological time of 5 milliseconds.

Spontaneous breathing is simulated by incorporating a non-linear pressure signal acting on the lungs that induces a negative pressure gradient between the lungs and the upper respiratory tract, driving the flow of air [see[13] for further details]. The applied pressure at the nasal passage and mouth is assigned to be 0 cmH_2O (relative to the atmospheric pressure) during un-assisted spontaneous ventilation and adjusted to appropriate pressures during continuous positive applied pressure (CPAP) ventilation. Lung trauma due to a blast is simulated in the model by varying two parameters: 1) Extrinsic pressure (P_{ext}, cmH_2O) represents the effective net pressure operating on each alveolar compartment, attributed to factors including alveolar interdependence, presence of adequate levels of pulmonary surfactant or the accumulation of interstitial fluid, and 2) Threshold opening pressure (TOP, cmH_2O), represents the threshold of the airway pressure below which an airway to an alveolar compartment will remain collapsed. As would be expected, the source data also details acute changes in mean respiratory rates in response

to both blast and haemorrhage (Figure 1b), which have been integrated into the simulations.

Haemorrhage model

In order to model haemorrhage, a cardiovascular control module is implemented that produces a realistic baroreflex based autoregulation response, targeting cardiovascular homeostasis. In response to deviation in mean arterial pressure (MAP) from a reference value (MAP_R), the control module adjusts θ, representing three variables of the cardiovascular system: Heart rate (HR), ventricular contractility and peripheral systemic resistance. The value of θ at time t (θ_t) with respect to its value at the preceding time instant, $\theta_{\bar{t}}$, is given by

$$\theta_t = \begin{cases} \theta_{\bar{t}} + \left[\alpha_\theta\{\theta_{max} - \theta_{\bar{t}}\}\dfrac{(MAP_R - MAP)}{20}\right], & if\ MAP < \ MAP_R \\ \theta_{\bar{t}} - \left[\ \alpha_\theta\{\theta_{\bar{t}} - \theta_R\}\right], & if\ MAP \geq \ MAP_R \end{cases}$$

For this study, MAP_R is set to 85 mm Hg. θ_{max} and θ_R define the maximum and reference values of the related variable θ, respectively. α_θ allows for the calibration of the rate of response and is used to match the model outputs to data. Based on the available data, the autoregulatory response has been scaled to reflect measured parameters following both blast exposure and severe haemorrhage, as shown in Table 1. The model iteratively adopts blast and then haemorrhagic physiological parameters as these injuries are suffered by the casualty.

Model validation

The source data used to calibrate and validate the above model was provided by collaborators at the Defence Science and Technology Laboratories, Porton Down, UK. 12 anaesthetised pigs were exposed to blast and haemorrhagic shock under controlled conditions, resulting in a moderate blast lung injury.[8] Fig. 2 compares the model response with the source data. Baseline PaO_2 values are somewhat lower in the pig data than in our *in-silico* subjects, consistent with the fact that the pigs are studied after laparotomy and breathing air spontaneously while anaesthetised. Model responses to PBLI for PaO_2 and

$PaCO_2$ show a good agreement with the data for both the mild and moderate lung injury. In the severe lung injury casualty, PaO_2 value are lower and $PaCO_2$ values higher, than the source data – the increase in $PaCO_2$ demonstrated by the model can be explained by the simulated increase in respiratory rate (see Fig. 1) being insufficient to counteract the effect of the PBLI in this case. Model responses to PBLI for the haemodynamic variables show an excellent match to the data in all cases (note the differences in baseline heart rate between the porcine and human models, as expected). The large reduction of MAP after haemorrhage seen in the porcine data is reinforced by the Barcroft-Edholm reflex, [19] characterised by the reduction of peripheral resistance and heart rate. This reflex is replicated in our model, producing a reduction of MAP to 55 mmHg. After the initiation of this reflex, cardiovascular autoregulatory mechanisms in the model react to try to increase MAP by increasing heart rate and systemic vascular resistance. However, despite heart rate recovering, MAP continues to decrease to 35 mmHg owing to the slow recovery of systemic vascular resistance, the large volume of blood loss, and persistent myocardial impairment (reduced contractility as a result of the blast injury). [19] Overall, the range of responses generated by the computational model provide a realistic spectrum of severity of PBLI and associated responses that agree well with the experimental data from.[8]

Modelling study protocol

Figure 1a illustrates the simulation protocol implemented in this study. Three *in-silico* casualties (healthy young adult males) are created and are initially allowed to reach a physiological steady state within the model for 40 minutes. From this time point up to 55 minutes, the simulation follows the blast injury trial protocol as detailed in the source *in-vivo* data (explained briefly below).[8] At 30, 35, and 40 minutes, three baseline readings are taken. The effects of mild, moderate and severe blast lung injury are then simulated in each of the *in-silico* casualties respectively. This results in an increasing contusional injury (10%, 25%, and 50% of the total lung), reflecting the various degrees of direct lung insult that has been observed in survivors with BLI. Alveolar threshold opening pressures in the contused lung were randomly assigned within a normal distribution to give values consistent with published values.[9,10] At 55 minutes, each casualty suffers a loss of 30% of their blood volume over a 4 minute period. From 60 – 100 minutes, ambient-air CPAP is applied to each casualty replicating a potential first-aid measure in the event of a major incident

overwhelming the emergency medical services. The simulation is repeated for each casualty such that they are trialled with 5, 10 and 15 cmH_2O of CPAP.

Data collection

Throughout the simulation, we recorded heart rate (HR), mean arterial pressure (MAP), arterial partial pressure of oxygen and carbon dioxide (PaO_2 and $PaCO_2$ respectively) and cardiac output (CO). Additionally, the effect of CPAP was evaluated using shunt fraction (derived using the shunt equation) and tissue oxygen delivery (DO_2).

Primary modelling outcome

The primary outcome was the arterial partial pressure of oxygen at the end of each simulation modelling mild, moderate and severe primary blast injury (10%, 25%, 50% lung contusion, respectively).

Statistics

Due to the deterministic nature of the modelling, where the outcome is independent of number of modelling runs unless we change the parameters or modelling boundaries, statistical analysis was not undertaken.

Results

Model validation

Baseline PaO_2 values were lower in the published pig data than in our *in-silico* subjects, consistent with the fact that the pigs are studied after laparotomy and breathing air spontaneously while anaesthetised (Figure 2). Model responses to PBLI for PaO_2 and $PaCO_2$ show a good agreement with the data for both the mild and moderate lung injury. In the severe lung injury casualty, PaO_2 value are lower and $PaCO_2$ values higher, than the source data – the increase in $PaCO_2$ demonstrated by the model can be explained by the simulated increase in respiratory rate (see Fig. 1) being insufficient to counteract the effect of the PBLI in this case.

Model characteristics

Our modelling shows that once haemorrhage stops after 59 minutes, haemodynamic recovery is characterised by the autoregulation of systemic vascular resistance and heart rate compensating for the reduction of MAP caused by blood loss and persistent myocardial impairment due to the blast injury (Figures 3-5). HR increases rapidly and SVR more slowly, with recovery of MAP (although the pre-haemorrhage MAP is not attained).

Mild lung injury

After 10% total lung contusion (Fig. 3), 5 cmH_2O ambient-air CPAP increased PaO_2 from 10.6 kPa to 12.6 kPa. Higher levels of CPAP did not further improve PaO_2 and slightly reduced cardiac output and DO_2.

Moderate lung injury

After 25% total lung contusion (Fig. 4), application of 10 cmH_2O CPAP produced a larger increase in PaO_2 (from 8.5 kPa to 11.1 kPa) than 5 cmH_2O, but a further increase to 15 cmH_2O produced no further benefit.

Severe lung injury

After 50% total lung contusion (Fig. 5), 5 cmH_2O CPAP increased PaO_2 from 4.06 kPa to 8.39 kPa. Further increasing CPAP to 10 and 15 cmH_2O resulted in slightly reduced values of PaO_2

(7.99 and 7.90 kPa, respectively) due to haemodynamic impairment (note smaller increases in cardiac output and DO_2).

Discussion

Ambient-air CPAP is considered to be of significant clinical benefit to casualties suffering lung injury in the pre-hospital environment. This modelling study suggests that this benefit also extends to casualties suffering from blast lung injury with severe haemorrhagic shock. However, high airway pressures can be detrimental to cardiac output through a combination of rising pulmonary vascular resistances increasing right ventricular afterload and diminishing venous return reducing right ventricular preload. [20] This leads to reduced right ventricular output and left ventricular filling. Consequently, improvement in oxygenation can be outweighed by positive pressure ventilation induced reduction in cardiac output leading to poor tissue oxygen delivery. [21] Our results suggest that increasing CPAP levels beyond 5 – 10 cmH_2O does not lead to clinically important improvements in arterial oxygenation, and in our most severely injured casualty model such increases in CPAP began to negatively affect oxygen delivery due to haemodynamic impairment.

Our model showed similar characteristics to an experimental model of porcine lung blast injury and haemorrhage. Model responses to PBLI for the haemodynamic variables show an excellent match to the data in all cases, other than the expected differences in baseline heart rate between the porcine and human models. The large reduction of MAP after haemorrhage seen in the porcine data is reinforced by the Barcroft-Edholm reflex, [19] characterised by the reduction of peripheral resistance and heart rate. This reflex is replicated in our model, producing a reduction of MAP to 55 mmHg. After the initiation of this reflex, cardiovascular autoregulatory mechanisms in the model react to try to increase MAP by increasing heart rate and systemic vascular resistance. However, despite heart rate recovering, MAP continues to decrease to 35 mmHg owing to the slow recovery of systemic

vascular resistance, the large volume of blood loss, and persistent myocardial impairment (reduced contractility as a result of the blast injury). [19] Overall, the range of responses generated by the computational model provide a realistic spectrum of severity of PBLI and associated responses that agree well with the experimental data from.[8]

Our model has a number of limitations. Data from human patients with which the model could be calibrated is scarce, and we therefore relied heavily on data from animal experiments. The model also does not currently include a representation of casualties with pneumothoraces, a limitation that we are currently working to remove. The application of a CPAP mask is intended for fully conscious casualties only. This study therefore applies only to casualties with non-significant head injury that are sufficiently haemodynamically stable to maintain normal cerebration. We recognise that clinical validation of our findings will be difficult given the context, but we feel that this only further emphasises the usefulness of using a computational modelling approach. Further development of the model and validation against additional datasets will allow us to simulate larger numbers of casualties with finer granularity and broader age ranges. However, the results described above represent the best evidence currently available on this subject for health care professionals working in the pre-hospital environment and concerned with the management of mass casualty scenarios.

In summary, our results suggest that in the event of a major incident involving polytraumatised young adults, provision of ambient-air masks providing 5 cmH_2O CPAP should be considered to reduce the need for invasive ventilatory support.

References

1 Nielsen VML, Madsen J, Aasen A, *et al*. Prehospital treatment with continuous positive airway pressure in patients with acute respiratory failure: a regional observation study. *Scand J Trauma Resusc Emerg Med* 2016; 24: 121.

2 Mattera C. Positive Pressure: The physiology of respirations with CPAP. *J Emerg Med Serv* 2011; Suppl 1: 6–10.

3 Plaisance P, Pirracchio R, Berton C, Vicaut E, Payen D. A randomized study of out-of-hospital continuous positive airway pressure for acute cardiogenic pulmonary oedema: Physiological and clinical effects. *Eur Heart J* 2007; 28: 2895–901.

4 Finn JC, Brink D, Mckenzie N*, et al.* Prehospital continuous positive airway pressure (CPAP) for acute respiratory distress: a randomised controlled trial. *Emerg Med J* Advance Access published on March 26, 2021, doi: 10.1136/emermed-2020-210256.

5 Graham S, Fairhall S, Rutter S, Auton P, *et al*. Continuous positive airway pressure: An early intervention to prevent phosgene-induced acute lung injury. *Toxicol Lett* 2018; 293: 120-6.

6 Scott TE, Haque M, Das A, Cliff I, Bates DG, Hardman JG. Efficacy of continuous positive airway pressure (CPAP) in casualties suffering from primary blast lung injury: A modelling study. 41st *Annu Int Conf IEEE Eng Med Biol Soc* 2019; 2019: 4965-8.

7 Mowery NT, Gunter O, Collier BR, *et al*. Practice Management Guidelines for Management of Hemothorax and Occult Pneumothorax. *J Trauma Inj Infect Crit Care* 2011; 70: 510-8.

8 Garner J, Watts S, Parry C, Bird J, Cooper G, Kirkman E. Prolonged permissive hypotensive resuscitation is associated with poor outcome in primary blast injury with controlled hemorrhage. *Ann Surg* 2010; 251: 1131-9.

9 Pulletz S, Adler A, Kott M, *et al*. Regional lung opening and closing pressures in patients with acute lung injury. J Crit Care 2012; 27: 323.e11-323.e18.

10 Gattinoni L, Caironi P, Cressoni M *et al*. Lung recruitment in patients with the acute respiratory distress syndrome. *N Engl J Med 2006;* 354: 1775-86.

11 Laviola M, Niklas C, Das A, Bates DG, Hardman JG. Ventilation strategies for front of neck airway rescue: an in silico study. *Br J Anaesth* 2021; 126: 1226-36.

12 Laviola M, Niklas C, Das A, Bates DG, Hardman JG. Effect of oxygen fraction on airway rescue: a computational modelling study. *Br J Anaesth* 2020; 125: e69-e74.

13 Weaver L, Das A, Saffaran S, *et al*. High risk of patient self-inflicted lung injury in COVID-19 with frequently encountered spontaneous breathing patterns: a computational modelling study. *Ann Intensive Care* 2021; 11: 109.

14 Scott TE, Das A, Haque M, Bates DG, Hardman JG. Management of primary blast lung injury: a comparison of airway pressure release versus low tidal volume ventilation. *Intensive Care Med Exp* 2020; 8: 26.

15 Das A, Haque M, Chikhani M, *et al*. Hemodynamic effects of lung recruitment maneuvers in acute respiratory distress syndrome. *BMC Pulm Med* 2017; 17: 34.

16 Saffaran S, Das A, Laffey JG, Hardman JG, Yehya N, Bates DG. Utility of Driving Pressure and Mechanical Power to Guide Protective Ventilator Settings in Two Cohorts of Adult and Pediatric Patients With Acute Respiratory Distress Syndrome: A Computational Investigation. *Crit Care Med* 2020; 48: 1001-8.

17 Das A, Saffaran S, Chikhani M, *et al.* In Silico Modeling of Coronavirus Disease 2019 Acute Respiratory Distress Syndrome: Pathophysiologic Insights and Potential Management Implications. *Crit Care Explor* 2020; 2: e0202.

18 Saffaran S, Das A, Hardman JG, Yehya N, Bates DG. High-fidelity computational simulation to refine strategies for lung-protective ventilation in paediatric acute respiratory distress syndrome. *Intensive Care Med* 2019; 45: 1055-7.

19 Kirkman E, Watts S. Haemodynamic changes in trauma. *Br J Anaesth* 2014; 113: 266-75.

20 Vieillard-Baron A, Matthay M, Teboul JL *et al*. Experts' opinion on management of hemodynamics in ARDS patients: focus on the effects of mechanical ventilation. *Intensive Care Med* 2016; 42: 739–49.

21 Chikhani M, Das A, Haque M, Wang W, Bates DG, Hardman JG. High PEEP in acute respiratory distress syndrome: quantitative evaluation between improved arterial oxygenation and decreased oxygen delivery. *Br J Anaesth* 2016; 117: 650-8.

Table 1. Parameters of the cardiovascular control model.

θ	θ_{max}	θ_R	Scenario	α_θ
Heart Rate	200 bpm	70 bpm	Normal	0.0005
			Haemorrhage	0.00002
			Blast trauma	0.00003
Contractility	20 mm Hg.ml^{-1}	2.95 mm Hg.ml^{-1}	Normal	0.01
			Haemorrhage	0.0001
			Blast trauma[1]	-
Systemic Resistance	0.2 mm Hg.s. ml^{-1}	0.09 mm Hg.s. ml^{-1}	Normal	0.01
			Haemorrhage[2]	0.0001
			Blast trauma	0.01

[1] In the case of blast trauma, ventricular contractility is reduced by 5.7 mm Hg.ml^{-1}, reflecting the myocardial impairment due to a blast. [2] The data from the porcine study indicates a drop in cardiac output despite an increase in heart rate, suggesting another autonomous reflex overriding the baroreflex. This is likely to be the 'Barcroft-Edholm reflex[19], a reduction in heart rate and a vasodilatory response to sudden onset of blood loss, which is included here as a one-off reduction in peripheral resistance at the beginning of haemorrhage.

Figure Legends

Figure 1. Simulation protocol

(a) The timeline of the implemented simulation protocol.

(b) the values of respiratory rate implemented during the simulation protocol.

Figure 2. Model validation.

Modelled patients' responses to varying levels of severity of PBLI and haemorrhage of 30% total blood volume, compared to responses of 12 pigs to PBLI and haemorrhage of 30% total blood volume (mean ± sd)[8] Refer to Figure 1 for simulation protocol. (A) Partial pressure of oxygen (cmH_2O), (B) Partial pressure of carbon dioxide (cmH_2O), (C) Mean Arterial Pressure (mmHg), (D) Cardiac Output (ml/min), (E) Heart Rate (BPM).

Figure 3. Patient responses to varying levels of CPAP for 10% lung contusion (Mild PBLI) and haemorrhage of 30% total blood volume.

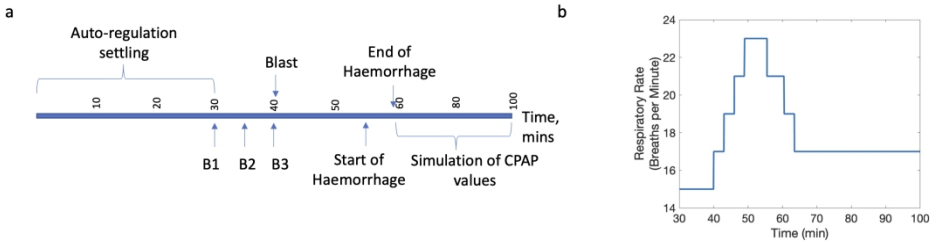
Refer to Figure 1 for simulation protocol. (A) Level of CPAP with ambient air (cmH_2O) implemented at 64 minutes into the simulation protocol, (B) Shunt fraction, (C) Partial pressure of oxygen (cmH_2O), (D) Partial pressure of carbon dioxide (cmH_2O), (E) Mean Arterial Pressure (mmHg), (F) Cardiac Output (ml/min), (G) Delivered Oxygen (ml/min), (H) End Expiratory Lung Volume (ml).

Figure 4. Patient responses to varying levels of CPAP for 25% lung contusion (Moderate PBLI) and haemorrhage of 30% total blood volume.

Refer to Figure 1 for simulation protocol. (A) Level of CPAP with ambient air (cmH_2O) implemented at 64 minutes into the simulation protocol, (B) Shunt fraction, (C) Partial pressure of oxygen (cmH_2O), (D) Partial pressure of carbon dioxide (cmH_2O), (E) Mean Arterial Pressure (mmHg), (F) Cardiac Output (ml/min), (G) Delivered Oxygen (ml/min), (H) End Expiratory Lung Volume (ml).

Figure 5. Patient responses to varying levels of CPAP for 50% lung contusion (Severe PBLI) and haemorrhage of 30% total blood volume.

Refer to Figure 1 for simulation protocol. (A) Level of CPAP with ambient air (cmH_2O) implemented at 64 minutes into the simulation protocol, (B) Shunt fraction, (C) Partial pressure of oxygen (cmH_2O), (D) Partial pressure of carbon dioxide (cmH_2O), (E) Mean Arterial Pressure (mmHg), (F) Cardiac Output (ml/min), (G) Delivered Oxygen (ml/min), (H) End Expiratory Lung Volume (ml).



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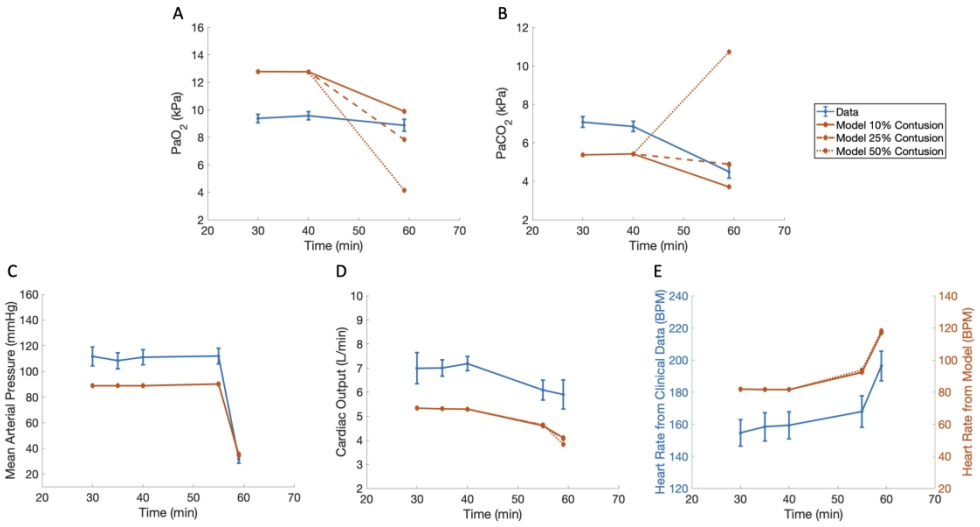

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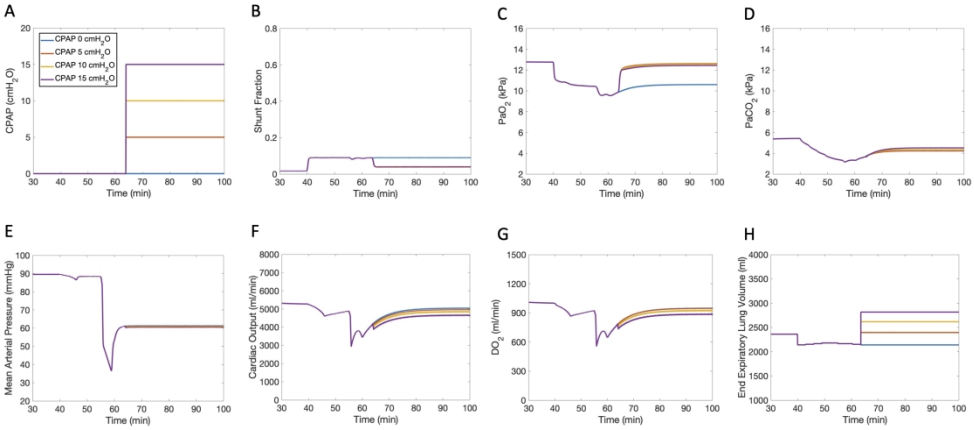

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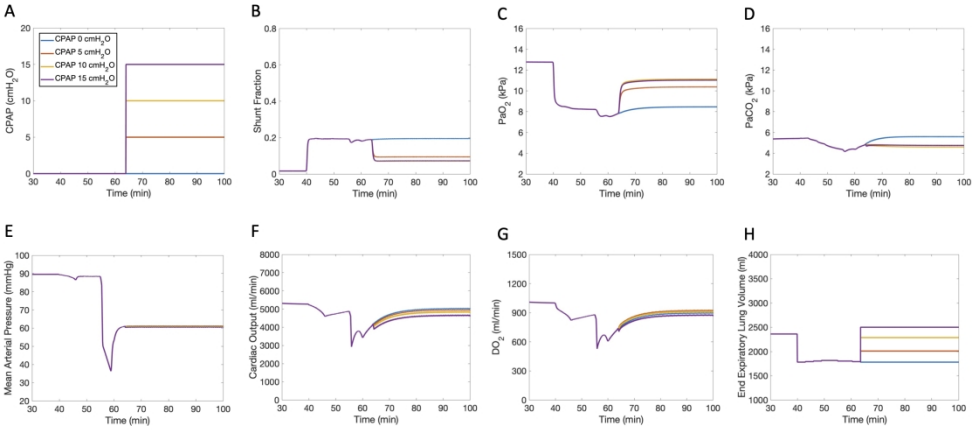

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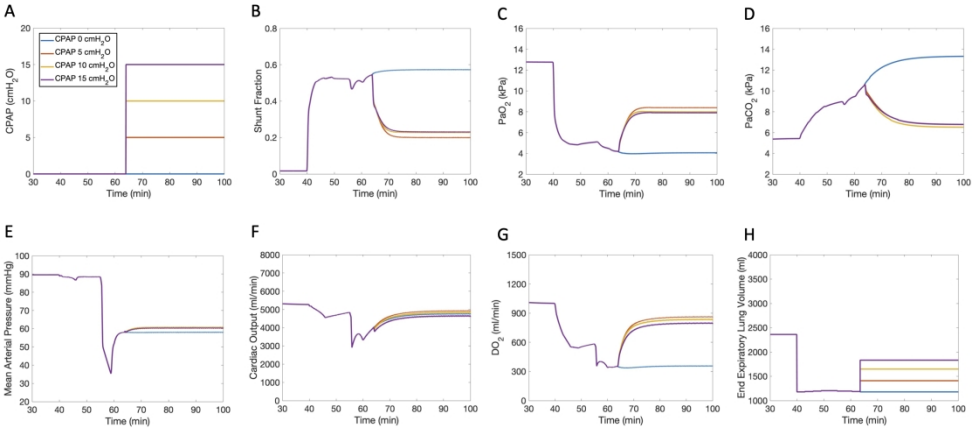

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