

# Oxygenation targets and monitoring in the critically ill: a point prevalence study of clinical practice in Australia and New Zealand

Paul J Young, Richard W Beasley, Gilles Capellier,  
Glenn M Eastwood and Steve A R Webb

The human body has adapted to the oxygen concentration of ambient air (21%) and has a normal  $P_{aO_2}$  of 80–100 mmHg.<sup>1</sup> This corresponds to oxygen saturation of 95%–99% when measured by arterial blood gas analysis (using  $S_{aO_2}$ ) or via pulse oximetry (using  $SpO_2$ ). Many critically ill patients require supplemental oxygen to maintain a normal  $SpO_2$  and, consequently, supplemental oxygen is one of the most common treatments given to patients in the intensive care unit. However, although the administration of supplemental oxygen can be life-saving, the indiscriminate use of oxygen in the ICU environment may be undesirable because it may expose patients to unnecessarily high inspired oxygen concentrations and/or hyperoxaemia, both of which might potentially be harmful.<sup>1</sup> As with other physiological targets, there may be a definable optimal  $SpO_2$  target for critically ill patients<sup>2</sup> that minimises the harms associated with too much or too little oxygen.

The association between arterial oxygen saturations and outcomes in critically ill patients has been explored in retrospective studies.<sup>3–7</sup> Unfortunately, due to the potential for unmeasured confounding effects in such studies, their findings do not provide a robust evidence base to inform clinicians. There are currently only limited data from prospective studies of different oxygen strategies in critically ill patients<sup>8</sup> and it is not clear whether a liberal or a conservative approach to oxygen administration is the most appropriate. Moreover, there appears to be a spectrum of views about what  $SpO_2$  is acceptable to nursing and medical staff monitoring patients in current ICU practice.<sup>9,10</sup> We hypothesised that high  $SpO_2$  values would generally be tolerated in critically ill patients but that low  $SpO_2$  values would be carefully avoided. Our aim was to evaluate clinical practice in adult ICU patients with respect to  $SpO_2$  monitoring, the prescription of  $SpO_2$  targets by doctors, and the upper and lower limits of tolerance of high and low  $SpO_2$  levels by ICU bedside nurses.

## Methods

We undertook an observational, cross-sectional study in 48 Australian and New Zealand centres under the auspices of the Australian and New Zealand Intensive Care Society

## ABSTRACT

**Background:** Many critically ill patients require supplemental oxygen. However, the optimal oxygen saturation measured by pulse oximetry ( $SpO_2$ ) in intensive care unit patients is unknown.

**Objective:** To evaluate clinical practice in Australia and New Zealand ICUs in relation to  $SpO_2$  monitoring, prescription of  $SpO_2$  targets by doctors, and upper and lower limits of tolerance of high and low  $SpO_2$  levels by ICU bedside nurses.

**Method:** Cross-sectional, observational study conducted on 2 days in 2013 involving adult patients in Australia and New Zealand ICUs.

**Results:** Data from 350 adult ICU patients were included.  $SpO_2$  alarms were less likely to be disabled in patients who were invasively ventilated than in patients not receiving supplemental oxygen (4.8% v 15.1%;  $P=0.02$ ). In mechanically ventilated patients and non-ventilated patients receiving supplemental oxygen, the lower prescribed  $SpO_2$  limit and the ICU bedside nurses' stated limits for action for low  $SpO_2$  levels were 92% (interquartile range, 90%–94%). Upper  $SpO_2$  limits were less frequently prescribed than lower  $SpO_2$  limits (4.9% [95% CI, 3.0%–7.7%] v 36.6% [95% CI, 31.7%–41.7%]);  $P<0.01$  and the observed  $SpO_2$  exceeded the prescribed upper limit on 10/17 occasions (59%) when an upper limit was prescribed.

**Conclusion:** Our findings suggest a relatively low level of vigilance in relation to prevention of high  $SpO_2$  compared with low  $SpO_2$  for adult patients in Australian and New Zealand ICUs.

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Clinical Trials Group (ANZICS-CTG) Point Prevalence Program (PPP). Site-based contributors are listed in the Appendix (online at [cicm.org.au/Resources/Publications/Journal](http://cicm.org.au/Resources/Publications/Journal)). The PPP facilitates a 24-hour data-capture period in patients who are occupying a bed in participating ICUs in Australia and New Zealand on one of two PPP days at 10 am. Each

**Table 1. Demographic and intensive care unit admission characteristics of patients**

Characteristic	Invasively vent.		Non-invasively vent.		Not vent., on supp. O <sub>2</sub>		Not vent., not on supp. O <sub>2</sub>
	n = 134	P*	n = 8	P*	n = 110	P*	n = 98
Mean age, years (SD)	55.7 (16.6)	0.93	64.1 (20.3)	0.26	60.4 (16.8)	0.08	55.9 (19.7)
Male, %	60.4%	0.69	50.0%	0.73	60.9%	0.67	57.0%
Mean weight, kg (SD)	80.0 (22.1)	0.83	90.1 (14.7)	0.73	82.9 (29.5)	0.52	80.6 (19.9)
Admission source, %							
Emergency department	32.8%	0.67	0	0.19	21.8%	0.62	25.5%
Ward	20.1%	0.16	75.0%	0.01	25.5%	0.73	28.6%
Other intensive care unit	6.7%	0.79	0	1.0	3.6%	1.0	5.1%
Other hospital	12.7%	0.52	0	1.0	6.4%	0.60	9.2%
OR after emergency surg.	17.9%	0.13	25.0%	0.22	10.0%	1.0	10.2%
OR after elective surg.	9.7%	0.01	0	0.35	32.7%	0.08	21.4%
ICU readmission	5.2%	0.06	12.5%	0.56	9.1%	1.0	9.2%
Reason for admission, %							
Trauma	18.7%	0.06	12.5%	0.56	6.4%	0.60	9.2%
Sepsis	40.3%	<0.01	25.0%	0.56	19.1%	1.0	12.2%
Mean APACHE II score (SD)	21.7 (7.4)	<0.01	20.3 (6.4)	0.28	16.4 (7.6)	0.19	15.8 (5.8)

Vent. = ventilated. Supp. O<sub>2</sub> = supplemental oxygen. OR = operating room. Surg. = surgery. APACHE = Acute Physiology and Chronic Health Evaluation.

\* Comparisons with patients not ventilated or on supplemental oxygen.

participating ICU enrolled patients on 7 November 2013 or 11 December 2013.

All patients aged 16 years or older were eligible for enrolment in this study if they were in a study ICU on one of the PPP days. The study cohort was prospectively divided into four groups: invasively ventilated, non-invasively ventilated, not ventilated but receiving supplemental oxygen, and not ventilated and not receiving supplemental oxygen. Invasive ventilation was defined as any form of positive pressure ventilation administered via an endotracheal tube or tracheostomy tube, including T-pieces, and spontaneous breathing with positive end-expiratory pres-

sure (PEEP) and/or pressure support. Non-invasive ventilation was defined as continuous positive airway pressure (CPAP) or bilevel positive airway pressure (BIPAP) administered via a face mask or nasal mask; it did not include high-flow nasal prongs.

Data collected at the bedside during the PPP day included demographic data, SpO<sub>2</sub> recordings, physiological monitoring data, alarm limits, prescribed limits of SpO<sub>2</sub>, and the stated threshold of each ICU nurse for action in response to low or high SpO<sub>2</sub> recordings. Data obtained from the medical notes (including the ICU flow chart) included patient demographic data, comorbidities, severity of illness

**Table 2. Characteristics of SpO<sub>2</sub> alarms**

SpO <sub>2</sub> alarm characteristic	Invasively vent.		Non-invasively vent.		Not vent., on supp. O <sub>2</sub>		Not vent., not on supp. O <sub>2</sub>
	n = 126	P*	n = 8	P*	n = 103	P*	n = 73
Disabled, n (%)	6 (4.8%)	0.02	1 (12.5%)	1.0	12 (11.7%)	0.51	11 (15.1%)
High SpO <sub>2</sub> alarm							
Median, % (IQR)	100% (100%–100%)	0.80	100% (100%–100%)	0.61	100% (100%–100%)	0.50	100% (100%–100%)
Min %, max %	100%, 125%		92%, 105%		100%, 100%		100%, 105%
Low SpO <sub>2</sub> alarm							
Median, % (IQR)	90% (90%–92%)	0.44	88% (86%–90%)	0.03	90% (90%–92%)	0.39	90% (90%–92%)
Min %, max %	60%, 95%		80%, 92%		65%, 95%		65%, 95%

Vent. = ventilated. Supp. O<sub>2</sub> = supplemental oxygen. IQR = interquartile range. \* Comparisons with patients not ventilated or on supplemental oxygen.

**Table 3. Characteristics of SpO<sub>2</sub> target upper limits**

SpO <sub>2</sub> characteristic	Invasively vent.		Non-invasively vent.		Not vent., on supp. O <sub>2</sub>		Not vent., not on supp. O <sub>2</sub>
	n = 134	P*	n = 8	P*	n = 110	P*	n = 98
Upper limit prescribed by doctor, n (%)	7 (5.2%)	0.76	1 (12.5%)	0.33	5 (4.5%)	1.0	4 (4.1%)
Median prescribed upper limit (IQR); min %, max %	92% (92%–94%); 92%, 94%	0.90	92% (92%–92%); 92%, 92%	NA	95% (94%–95%); 90%, 100%	0.54	94% (92%–95%); 90%, 95%
Current SpO <sub>2</sub> value > doctor-prescribed upper limit	3 (42.9%)	0.23	0	0.20	2/5 (40%)	0.44	4 (100%)
Upper limit set by nurse							
Median nurse-set upper limit (IQR); min %, max %	100% (100%–100%); 80%, <sup>†</sup> 100%	< 0.01	100% (100%–100%); 92%, 100%	0.99	100% (100%–100%); 90%, 105%	0.11	100% (100%–100%); 95%, 105%
Current SpO <sub>2</sub> value > nurse-set upper limit for action	6 (5.5%)	0.26	1 (12.5%)	0.40	3 (3.2%)	0.51	1 (1.5%)

Vent. = ventilated. Supp. O<sub>2</sub> = supplemental oxygen. IQR = interquartile range. NA = not applicable. \* Comparisons with patients not ventilated or on supp. O<sub>2</sub>. † Next lowest SpO<sub>2</sub> value was 90%.

(Acute Physiology and Chronic Health Evaluation [APACHE]-II scores) recorded at ICU admission, and prescribed SpO<sub>2</sub> limits. Current physiological monitoring, alarm limits, and SpO<sub>2</sub> recordings were obtained by research coordinators from direct observation of the bedside monitor. The ICU bedside nurses' thresholds for action were determined from direct questioning by research coordinators.

Study groups were compared using the group of patients who were not ventilated and were receiving no supplemental oxygen as a reference. When relevant, 95% confidence intervals for proportions were calculated using the modified Wald method. Differences between means were tested

using the student *t* test for normally distributed data and the Mann–Whitney test for non-normally distributed data. Differences in proportions were tested using the Fisher exact test. No assumptions were made about missing data. Data were collected prospectively by ICU research coordinators at participating hospitals. Study data were collected and managed using REDCap (Research Electronic Data Capture).<sup>11</sup> Statistical analysis was performed using GraphPad Prism 6.1 (GraphPad Software). Statistical significance was set at *P* < 0.05 with no adjustment for multiple measures. Ethics approval was obtained and included a waiver of consent for the PPP.

**Table 4. Characteristics of SpO<sub>2</sub> target lower limits**

SpO <sub>2</sub> characteristic	Invasively vent.		Non-invasively vent.		Not vent., on supp. O <sub>2</sub>		Not vent., not on supp. O <sub>2</sub>
	n = 134	P	n = 8	P	n = 110	P	n = 98
Lower limit prescribed by doctor, n (%)	53 (39.6%)	0.41	4 (50%)	0.45	38 (34.5%)	1.0	33 (33.7%)
Median prescribed lower limit (IQR); min, max	92% (90%–94%); 80%; 95%	0.28	90% (85%–91%); 70%, 92%	0.02	92% (90%–94%); 85%, 96%	0.46	92% (92%–94%); 85%, 95%
Current SpO <sub>2</sub> value < doctor-prescribed lower limit	7/46; 13.2%	0.14	0	1.0	2/37 (5.4%)	1.0	1/29 (3.4%)
Lower limit set by nurse							
Median nurse-set lower limit (IQR); min, max	92% (90%–94%); 50%,* 100%	0.11	90% (89%–90%); 70%, 95%	0.02	92% (90%–94%); 80%, 96%	0.40	92% (90%–94%); 85%, 96%
Current SpO <sub>2</sub> value < nurse-set lower limit for action	6/121; 5.0%	0.26	0	1.0	4/99 (4.0%)	0.40	1/73 (1.4%)

Vent. = ventilated. Supp. O<sub>2</sub> = supplemental oxygen. IQR = interquartile range. \* Next lowest SpO<sub>2</sub> value was 80%.

## Results

A total of 467 patients were included on the two PPP days. Of these, 350 patients (75%) were 16 years or older and were included in this study. Demographic and ICU admission data for the study cohort are shown in Table 1. At the time the patient assessments were undertaken by the research coordinator, 252 of 350 patients (72%) were ventilated and/or receiving supplemental oxygen. Compared with patients who were not ventilated and were not receiving supplemental oxygen, invasively ventilated patients had a higher APACHE-II illness severity ( $P < 0.01$ ), were less likely to have been admitted following elective surgery, and were more likely to have sepsis ( $P < 0.01$ ). In other respects, the baseline characteristics of the patient groups were similar.

Monitoring data for SpO<sub>2</sub> levels were available for 310 patients (Table 2), representing 94% of all patients ventilated or receiving supplemental oxygen (95% CI, 90.3%–96.4%) and 74% of all patients who were not receiving oxygen (95% CI, 65%–82%) ( $P < 0.01$ ). Alarms were less likely to be disabled in patients who were invasively ventilated than in patients who were not receiving supplemental oxygen (4.8% v 15.1%;  $P = 0.02$ ). The median upper limit SpO<sub>2</sub> alarm set in all groups was 100% (interquartile range [IQR], 100%–100%). Only one patient had a set upper limit SpO<sub>2</sub> alarm of less than 100%. This patient was receiving non-invasive ventilation and the set upper SpO<sub>2</sub> limit was 92%. The lower limit SpO<sub>2</sub> alarms were set at around 90% in all groups (Table 2).

SpO<sub>2</sub> targets were only prescribed in a minority of patients (Table 3 and Table 4). Upper SpO<sub>2</sub> limits were prescribed less frequently than lower SpO<sub>2</sub> limits (4.9% [95% CI, 3.0%–7.7%] v 36.6% [95% CI, 31.7%–41.7%];  $P < 0.01$ ). Patients receiving no supplemental oxygen had similar prescribed SpO<sub>2</sub> limits to invasively ventilated patients, non-invasively ventilated patients, and non-ventilated patients receiving supplemental oxygen. Bedside nurses had similar thresholds for action in relation to low and high SpO<sub>2</sub> in the groups receiving oxygen compared with the group of patients who were not receiving supplemental oxygen.

In the small number of patients for whom upper limits of SpO<sub>2</sub> were prescribed by doctors, limits were generally between 90% and 95%. The observed SpO<sub>2</sub> exceeded the prescribed upper limit on 10/17 occasions (59%) when an upper limit was prescribed (Table 3). The upper limits set by bedside nurses for action in relation to high SpO<sub>2</sub> levels were 100% (IQR, 100%–100%) in all groups.

The lower SpO<sub>2</sub> limits prescribed by clinicians and set by bedside nurses were 92% (IQR, 90%–94%) for all patient groups except for the non-invasively ventilated patients, for whom the limit was generally slightly lower (Table 4).

## Discussion

### Key findings

We conducted a cross-sectional, observational study to evaluate current practice in SpO<sub>2</sub> targets and monitoring. In accordance with our hypothesis, we showed that high SpO<sub>2</sub> levels appear to be less rigorously monitored and avoided than low SpO<sub>2</sub> levels. In particular, we showed that upper-limit SpO<sub>2</sub> alarms are effectively never used because they are always set at or above the maximum physiologically possible value of 100%. We also showed that bedside ICU nurses generally did not specifically state that they would act on a high SpO<sub>2</sub> value. Upper limits for SpO<sub>2</sub> were prescribed by ICU doctors infrequently and, even when they were prescribed, the observed SpO<sub>2</sub> values often exceeded the prescribed limits.

In contrast to upper SpO<sub>2</sub> limit alarms, lower SpO<sub>2</sub> limit alarms were used commonly and acceptable lower SpO<sub>2</sub> limits were prescribed by doctors more often than upper SpO<sub>2</sub> limits. For most patients, lower prescribed SpO<sub>2</sub> limits were about 92% and lower SpO<sub>2</sub> alarm limits were about 90%.

### Relation to previous work

Our study is the first to evaluate clinical ICU practice in SpO<sub>2</sub> alarms, physiological monitoring and prescribed SpO<sub>2</sub> targets in a broad cohort of ventilated and non-ventilated ICU patients. Our findings suggest a relatively low level of vigilance in relation to prevention of high SpO<sub>2</sub> compared with low SpO<sub>2</sub> and are consistent with the existing literature.

Previous studies have shown that hyperoxaemia occurs commonly in critically ill patients who are receiving mechanical ventilation.<sup>3,4,12,13</sup> Survey findings suggest that most ICU nurses and doctors have some concern about oxygen toxicity in mechanically ventilated patients,<sup>9,10</sup> but that there is a clear difference between self-reported practice and actual practice of oxygen therapy.<sup>14</sup>

Our data are similar to data from a cross-sectional, observational study, conducted as part of the ANZICS-CTG PPP in 2013, of 178 non-ventilated patients receiving oxygen.<sup>15</sup> In this study, oxygen saturation targets were prescribed in 28 patients and 98.3% of patients had SpO<sub>2</sub> monitoring.<sup>15</sup> No data were provided on acceptable targets or alarm limits, but the mean highest Pao<sub>2</sub> was in the hyperoxaemic range, at 129 mmHg (range, 58–681 mmHg) and the mean lowest Pao<sub>2</sub> was 88 mmHg (range, 35–383 mmHg).<sup>15</sup>

### Clinical implications and significance

Clinical teaching and current ICU practice generally emphasises that avoidance of hypoxaemia is more important than

concerns about hyperoxaemia or exposure to high  $\text{FiO}_2$ .<sup>16</sup> Investigation of a strategy for precise control of arterial oxygen levels has been identified as a high research priority in critically ill patients<sup>17,18</sup> but there is currently insufficient evidence to guide clinical practice.<sup>1</sup> The acceptable lower  $\text{SpO}_2$  limits observed in our study were less than the minimum  $\text{SpO}_2$  of 94% that is currently recommended for acutely ill medical patients by the British Thoracic Society (BTS).<sup>19</sup> Similarly, the upper  $\text{SpO}_2$  limit of 98% suggested by the BTS does not appear to be adhered to in current Australian and New Zealand ICU practice. However, the BTS guidelines in relation to  $\text{SpO}_2$  targets are not supported by high-level evidence and were not specifically intended for use in mechanically ventilated ICU patients.<sup>19</sup>

### Strengths and limitations

Our study provides contemporary, prospective, multicentre, bi-national, cross-sectional observational data in relation to monitoring of oxygen therapy in a broad cohort of critically ill patients. We directly questioned ICU bedside nurses in order to determine when they would act on high and low  $\text{SpO}_2$  levels. Although our results may reflect what nurses say they would do rather than what they actually do, we verified concurrent monitor settings by direct observation and compared stated responses to concurrent patient  $\text{SpO}_2$  levels. A consistent message emerged that hyperoxaemia is less rigorously avoided than hypoxaemia.

Previous data have shown that tolerance of low  $\text{SpO}_2$  in mechanically ventilated patients tends to increase as the  $\text{FiO}_2$  increases.<sup>12,20</sup> We did not evaluate the relationship between  $\text{FiO}_2$  and upper and lower  $\text{SpO}_2$  limits in this study because our sample size was too small to allow this to be done in a statistically robust manner.

We chose to focus on  $\text{SpO}_2$  levels rather than  $\text{SaO}_2$  or  $\text{Pao}_2$  because  $\text{SpO}_2$  is the variable which is continuously monitored. However, we acknowledge that if liberal oxygen administration is harmful, the  $\text{Pao}_2$  and/or the  $\text{FiO}_2$  may be more important than the  $\text{SpO}_2$ .

We noted that for a small number of patients, the lower  $\text{SpO}_2$  alarm limit was extremely low (eg, 60%). We speculate that these very low limits were chosen to effectively bypass the alarm system rather than because these values were regarded as physiologically acceptable. However, we did not collect information on why particular limits were chosen and cannot be certain of the reasons.

### Conclusion

We found a relatively low level of vigilance in relation to prevention of high  $\text{SpO}_2$  compared with low  $\text{SpO}_2$  for adult ICU patients. A better understanding of current oxygen

therapy practice in the ICU is a fundamental first step in the development of future interventional trials.

### Competing interests

None declared.

### Author details

Paul J Young, Intensivist<sup>1,2</sup>

Richard W Beasley, Director<sup>1</sup>

Gilles Capellier, Intensivist<sup>3,4</sup>

Glenn M Eastwood, Intensivist<sup>5</sup>

Steve A R Webb, Intensivist<sup>6,7</sup>

1 Medical Research Institute of New Zealand, Wellington, New Zealand.

2 Intensive Care Unit, Wellington Regional Hospital, Wellington, New Zealand.

3 CHRU Besançon, Université de Franche-Comté, Besançon, France.

4 Australian and New Zealand Intensive Care Research Centre, Department of Epidemiology and Preventive Medicine, Monash University, Melbourne, VIC, Australia.

5 Department of Intensive Care, Austin Hospital, Melbourne, VIC, Australia.

6 The George Institute for International Health, Sydney, NSW, Australia.

7 School of Medicine and Pharmacology, University of Western Australia, Perth, WA, Australia.

**Correspondence:** Paul.Young@ccdhb.org.nz

### References

- 1 Suzuki S, Eastwood G, Bellomo R. A Re-evaluation of oxygen therapy and hyperoxemia in critical care. In: Vincent J-L, editor. Annual update in intensive care and emergency medicine. Switzerland: Springer International Publishing, 2014: 81-91.
- 2 Webb SA, Young PJ, Bellomo R. The "sweet spot" for physiological targets in critically ill patients. *Crit Care Resusc* 2012; 14: 253-5.
- 3 de Jonge E, Peelen L, Keijzers PJ, et al. Association between administered oxygen, arterial partial oxygen pressure and mortality in mechanically ventilated intensive care unit patients. *Crit Care* 2008; 12: R156.
- 4 Eastwood G, Bellomo R, Bailey M, et al. Arterial oxygen tension and mortality in mechanically ventilated patients. *Intensive Care Med* 2012; 38: 91-8.
- 5 Young P, Beasley R, Bailey M, et al. The association between early arterial oxygenation and mortality in ventilated patients with acute ischaemic stroke. *Crit Care Resusc* 2012; 14: 14-9.
- 6 Miles LF, Bailey M, Young P, Pilcher DV. Differences in mortality based on worsening ratio of partial pressure of oxygen to fraction of inspired oxygen corrected for immune system status and respiratory support. *Crit Care Resusc* 2012; 14: 25-32.
- 7 Bellomo R, Bailey M, Eastwood GM, et al. Arterial hyperoxia and in-hospital mortality after resuscitation from cardiac arrest. *Crit Care* 2011; 15: R90.
- 8 Suzuki S, Eastwood GM, Glassford NJ, et al. Conservative oxygen therapy in mechanically ventilated patients: a pilot before-and-after trial. *Crit Care Med* 2014; 42: 1414-22.

## ORIGINAL ARTICLES

- 9 Eastwood GM, Reade MC, Peck L, et al. Critical care nurses' opinion and self-reported practice of oxygen therapy: a survey. *Aust Crit Care* 2012; 25: 23-30.
- 10 Eastwood GM, Reade MC, Peck L, et al. Intensivists' opinion and self-reported practice of oxygen therapy. *Anaesth Intensive Care* 2011; 39: 122-6.
- 11 Harris PA, Taylor R, Thielke R, et al. Research electronic data capture (REDCap) — a metadata-driven methodology and workflow process for providing translational research informatics support. *J Biomed Inform* 2009; 42: 377-81.
- 12 Panwar R, Capellier G, Schmutz N, et al. Current oxygenation practice in ventilated patients — an observational cohort study. *Anaesth Intensive Care* 2013; 41: 505-14.
- 13 Suzuki S, Eastwood GM, Peck L, et al. Current oxygen management in mechanically ventilated patients: a prospective observational cohort study. *J Crit Care* 2013; 28: 647-54.
- 14 Helmerhorst HJ, Schultz MJ, van der Voort PH, et al. Self-reported attitudes versus actual practice of oxygen therapy by ICU physicians and nurses. *Ann Intensive Care* 2014; 4: 23.
- 15 Parke RL, Eastwood GM, McGuinness SP; George Institute for Global Health; Australian and New Zealand Intensive Care Society Clinical Trials Group. Oxygen therapy in non-intubated adult intensive care patients: a point prevalence study. *Crit Care Resusc* 2013; 15: 287-93.
- 16 Beasley R, McNaughton A, Robinson G. New look at the oxyhaemoglobin dissociation curve. *Lancet* 2006; 367: 1124-6.
- 17 Capellier G, Panwar R. Is it time for permissive hypoxaemia in the intensive care unit? *Crit Care Resusc* 2011; 13: 139-41.
- 18 Martin DS, Grocott MP. Oxygen therapy in critical illness: precise control of arterial oxygenation and permissive hypoxemia. *Crit Care Med* 2013; 41: 423-32.
- 19 O'Driscoll BR, Howard LS, Davison AG; British Thoracic Society. BTS guideline for emergency oxygen use in adult patients. *Thorax* 2008; 63 Suppl 6:vi1-68.
- 20 Graaff AE, Dongelmans DA, Binnekade JM, de Jonge E. Clinicians' response to hyperoxia in ventilated patients in a Dutch ICU depends on the level of FiO<sub>2</sub>. *Intensive Care Med* 2011; 37: 46-51. □

## Appendix 1. Participating Sites and Investigators

This appendix was part of the submitted manuscript and has been peer reviewed. It is posted as supplied by the authors.

<b>Site/Institution</b>	<b>Principal Investigator/s</b>	<b>Research Co-ordinator/s</b>
<b>The George Institute for Global Health</b>	Parisa Glass Naomi Hammond Ashleigh Myburgh John Myburgh Dorrilyn Rajbhandari Ian Seppelt Nicola Watts	
<b>Canberra Hospital</b>	Sean Chan	Helen Rodgers Amy Harney Katie Milburn
<b>Royal Children's Hospital, Brisbane</b>	Anthony Slater	Debbie Long Tara Williams
<b>Starship Hospital</b>	John Beca Dr Liz Segedin	Claire Sherring Miriam Rea Tracey Bushell
<b>Royal Children's Hospital</b>	Warwick Butt	Carmel Delzoppo
<b>Mater Children's Hospital</b>	Andreas Schibler Christian Stocker	Sara Mayfield
<b>Concord Hospital</b>	David Milliss	Helen Wong

<b>Nepean Hospital</b>	Ian Seppelt	Leonie Weisbrodt Anne Ritchie Maria Nikas Rebecca Gresham
<b>North Shore Private Hospital</b>	Anthony Delaney	Dena-Louise Hogben Laura Davies
<b>Prince of Wales Hospital</b>	Prof Yahya Shehabi	Nicola Straiton
<b>Royal North Shore Hospital</b>	Prof Simon Finfer	Frances Bass Naomi Hammond Anne O'Connor Elizabeth Yarad Simon Bird
<b>St George Hospital</b>	Prof John Myburgh Manoj Saxena	Jennene Miller Rebecca Sidoli Deborah Inskip
<b>St Vincent's Hospital, Sydney</b>	Priya Nair	Serena Knowles
<b>Westmead Hospital</b>	Vineet Nayyar	Christina Skelly Jing Kong
<b>Wollongong Hospital</b>	Martin Sterba	Bronwyn Johnson Wenli Geng
<b>Auckland City Hospital</b>	Rachael Parke	Eileen Gilder Lianne McCarthy Rachael Parke
<b>Auckland DCCM</b>	Colin McArthur Lynette Newby	Lynette Newby Yan Chen
<b>Christchurch Hospital</b>	Seton Henderson	Jan Mehrrens



	David Knight	
<b>Middlemore Hospital</b>	Tony Williams	Chantal Hogan Tony Williams
<b>Waikato Hospital</b>	Rob Frengley	Mary La Pine John Durning
<b>Wellington Hospital</b>	Dick Dinsdale	Lynn Andrews Sally Hurford Anna Hunt
<b>North Shore Hospital (Auck)</b>	Janet Liang	Jeanette Bell
<b>Tauranga Hospital</b>	Troy Browne Rachel Atkin	Jennifer Goodson
<b>Flinders Medical Centre</b>	Santosh Verghese	Elisha Matheson Kate Schwartz
<b>Lyell McEwin Hospital</b>	Rajaram Ramadoss	Josette Wood
<b>The Queen Elizabeth Hospital</b>	Sandra Peake	Catherine Kurenda JoAnne McIntrye
<b>Royal Adelaide Hospital</b>	Stephanie O'Connor	Sonya Kloeden Justine Rivett
<b>Austin Hospital</b>	Rinaldo Bellomo	Glenn Eastwood Leah Peck Helen Young
<b>Bendigo Hospital</b>	Jason Fletcher	Julie Smith
<b>Cabrini Hospital</b>	Jonathan Barrett	Gabrielle Hanlon
<b>Geelong Hospital</b>	Claire Cattigan	Tania Salerno Allison Bone Tania Elderkin

<b>The Northern Hospital</b>	Dr Angaj Ghosh John Green Andrew Casamento	Mary Park Olga Burgess
<b>Royal Melbourne Hospital</b>	Christopher Macisaac	Deborah Barge Andrea Jordan
<b>St Vincent's Hospital, Melbourne</b>	John Santamaria	Roger Smith Jennifer Holmes
<b>Western Health</b>	Craig French	Samantha Bates
<b>Albury Hospital</b>	Charles Mashonganyika	Clare Maher