Lung recruitment and positive airway pressure before extubation does not improve oxygenation in the post-anaesthesia care unit: a randomized clinical trial

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Background. Atelectasis is known to develop during anaesthesia and after operation atelectasis leads to impaired oxygenation. Lung recruitment manoeuvres, positive end-expiratory pressure (PEEP), and continuous positive airway pressure (CPAP) have been proposed for reduction of atelectasis but their benefits have not been shown to persist after operation. We proposed that a combination of these techniques before extubation would improve oxygenation after operation.

Methods. Adult patients undergoing elective surgery requiring tracheal intubation and an arterial catheter were randomized to receive either: a lung recruitment manoeuvre of 40 cm H2O for 15 s, 30 min before the end of anaesthesia, followed by 10 cm H2O of PEEP and then 10 cm H2O of CPAP from return of spontaneous breathing until extubation; or no lung recruitment manoeuvre, ∆c mH 2O PEEP, and no CPAP. Arterial blood gases were taken at randomization and 1 h after extubation. The primary endpoint of the study was the change in (A–a)DO2 between these times. Statistical analysis of the two groups was done by χ² or unpaired t-test as appropriate.

Results. Twenty-two patients were recruited to each group. There were no significant differences between the groups before randomization. There was no significant difference in the change in (A–a)DO2 between the groups (P=0.82).

Conclusions. Postoperative oxygenation is not improved by a combination of a lung recruitment manoeuvre and maintenance of a positive airway pressure until extubation. Further research is needed to elucidate the mechanism of atelectasis on emergence from anaesthesia and to evaluate more invasive clinical strategies such as post-extubation CPAP.

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Atelectasis in dependent lung units is an almost inevitable consequence of general anaesthesia with IPPV and, along with altered ventilation perfusion relationships, causes an increase in alveolar to arterial oxygen partial pressure difference (A–a)DO2. Atelectasis, which can persist into the postoperative period, is resistant to simple techniques normally used to improve lung function such as patient posture and may contribute to postoperative pulmonary complications. Studies using computerized tomography (CT) during anaesthesia have shown that atelectasis formation can be attenuated by the use of positive end-expiratory pressure (PEEP) and the avoidance of high inspired oxygen fractions (FIO2). Once formed, atelectatic lung regions may be effectively recruited by maintaining a sustained airway pressure of 40 cmH2O for 15 s. This is the only lung recruitment manoeuvre that
has been shown to be effective in CT studies, is safe in clinical use, and improves intraoperative oxygenation. When performed in isolation, this improvement in oxygenation does not persist into the postoperative period.

If an intraoperative recruitment manoeuvre is followed by the use of 10 cm H₂O of PEEP, atelectasis is prevented from recurring even in the presence of a high Fl₁₀. We hypothesized that if positive airway pressure were maintained after a recruitment manoeuvre and throughout emergence from anaesthesia, there would be a reduction in atelectasis even in the presence of 100% oxygen and so an improvement in oxygenation in the postoperative period. The key period for developing atelectasis at the end of anaesthesia is likely to be the period after discontinuation of IPPV and PEEP but before extubation. During this period, patients often cough and zero end-expiratory pressure (ZEEP) is commonly used and will accentuate flow- and volume-related airway collapse. ZEEP may be avoided in the spontaneously breathing, intubated patient by preventing disconnection of the breathing system from the trachea to maintain the positive airway pressure. Circuit disconnection was avoided at all times before extubation of the trachea to maintain the positive airway pressure.

We tested the hypothesis that by performing a recruitment manoeuvre near the end of the anaesthetic followed by PEEP and then CPAP until extubation, the (A–a)DO₂ 1 h after extubation would be improved compared with patients in whom these interventions were not performed.

### Methods

The study was approved by the Leeds Local Research Ethics Committee (Ref: 06/Q1205/17 COREC Locked Code: AB/66109/2) and registered at [http://www.controlled-trials.com](http://www.controlled-trials.com) (Identification number: ISRCTN32464251). Written informed consent was obtained from all the study subjects.

Patients were recruited from elective surgical lists at the two large teaching hospitals in the Leeds Teaching Hospitals NHS Trust. Patients deemed eligible for inclusion were adults undergoing elective operative procedures that were expected to last >45 min, and whose anaesthesia technique was to include tracheal intubation, muscle relaxation, IPPV, and the insertion of an arterial catheter for clinical care. Patients were excluded if there was any contraindication to the use of 10 cm H₂O of PEEP or the recruitment manoeuvre or the clinical requirement for the use of >5 cm H₂O of intraoperative PEEP. Patients were also excluded if any perioperative cardiovascular or respiratory event occurred which the anaesthetist with clinical responsibility for the patient thought would make the study intervention clinically unacceptable.

Patient factors recorded before operation were age, sex, BMI, and smoking status. Apart from the requirements for inclusion in the study described above, the anaesthetic technique was left to the discretion of the anaesthetist. Intraoperative IPPV settings were an Fl₁₀ of 0.3–0.8, PEEP ≤5 cm H₂O, tidal volume 7–10 ml kg⁻¹, and ventilatory frequency as appropriate to maintain end-tidal carbon dioxide partial pressure of 4–5.5 kPa. Thirty minutes before the anticipated end of the anaesthetic, an arterial blood sample was obtained for measurement of arterial Po₂ and Pco₂, and the Fl₁₀ was recorded. The patients’ allocation was concealed from the investigators until this point when an envelope prepared at the start of the trial (by a non-investigator) bearing only a number on the outside and the allocation group on the inside was opened.

Fifteen minutes before the anticipated end of anaesthesia, patients in the intervention group received a lung recruitment manoeuvre consisting of lung inflation to an airway pressure of 40 cm H₂O sustained for 15 s. Immediately after this recruitment manoeuvre, ventilation continued as previously but with PEEP increased to 10 cm H₂O.IPPV continued at the same rate until evidence of return of spontaneous ventilation. At this point, CPAP was maintained at 10 cm H₂O by adjusting the APL valve and providing a fresh gas flow of >10 litre min⁻¹ to maintain the positive airway pressure during inspiration. Circuit disconnection was avoided at all times before extubation of the trachea to maintain the positive airway pressure.

The control group received no lung recruitment manoeuvre and ventilation continued with the current settings including any intraoperative PEEP (≤5 cm H₂O). No CPAP was applied on return of spontaneous ventilation, with the APL valve left fully open. Circuit disconnection was permitted, for example, when moving the patient.

In both groups, the Fl₁₀ was set to 1.0 with fresh gas flow exceeding 10 litre min⁻¹ before extubation of the trachea. This occurred when deemed appropriate by the anaesthetist with clinical responsibility for the patient.

In the post-anaesthesia care unit (PACU), both groups received standard post-anaesthetic care. A Venturi mask (Lifecare Hospital Supplies, Edinburgh, UK) providing an Fl₁₀ of 0.4 was used for all patients from 45 min post-extubation until an arterial blood sample was obtained at 1 h post-extubation for measurement of arterial Po₂ and Pco₂.

The (A–a)DO₂ gradient at 1 h post-extubation was used as the endpoint of the study. Arterial oxygen partial pressure was measured from the arterial blood samples as described above. Alveolar Po₂ was calculated using the following version of the alveolar gas equation:

\[
\text{alveolar } P_{o₂} = P_{l_{o₂}} - \frac{P_{a_{co₂}}}{RQ} \left(1 - Fl_{o₂}(1 - RQ)\right)
\]

RQ assumed to be 0.8.

The standard deviation (SD) of (A–a)DO₂ for the purposes of the power calculation was derived from preinduction values in a previous study of a similar population.
A significance level for the study of 5% (P=0.05) and a power of 80% were used for the calculation. Two groups of 22 subjects were calculated to be needed to detect a difference of 3.33 kPa (0.8 times the previously published SD).

Statistical analysis of the two groups was done by χ² or unpaired t-test as appropriate with significance assumed with P<0.05 using SPSS 15.0 (SPSS Inc., Chicago, IL, USA).

Results
A total of 52 patients were enrolled between June 2006 and February 2009 (Fig. 1). There were no significant differences between the groups with regard to preoperative characteristics, duration, and nature of operation, and arterial gases at randomization and in PACU (Table 1).

The mean (SD) (A–a)DO₂ during anaesthesia was 12.3 (8.4) kPa and this increased slightly in PACU to 14.1 (5.2) kPa. The change in individual (A–a)DO₂ between randomization and PACU displayed no particular pattern (Fig. 2).

Using each patient as their own control, the mean change in (A–a)DO₂ was 1.98 (6.52) in the standard group and 1.52 (6.71) in the intervention group (P=0.82). This showed that there was no statistical difference or clinical benefit accruing from the intervention.

Discussion
This study has shown that patients undergoing general anaesthesia develop abnormal (A–a)DO₂ and that this persists into the postoperative period. We have also shown that a lung recruitment manoeuvre followed by positive airway pressure until extubation does not improve the (A–a)DO₂ in PACU.

In the usual absence of a significant alveolar to pulmonary capillary diffusion barrier, abnormalities of (A–a)DO₂ result from intrapulmonary shunting and areas of lung with low ventilation–perfusion ratios. During anaesthesia, intrapulmonary shunting through areas of atelectasis is the most significant contributor; so, in the absence of acute lung pathology before operation, it may be assumed that changes in (A–a)DO₂ are mostly secondary to the development of atelectasis.

Our results are comparable with a previous study which also used (A–a)DO₂ as a marker of atelectasis, and demonstrate high (A–a)DO₂ values at the end of the
anaesthetic that become even higher at 1 h after operation. As demonstrated by the large standard deviations in our data, there is a wide variation between patients, particularly during anaesthesia, results which are again comparable with the previous study, which also studied the effects of ‘manual inflations’ every 30 min throughout surgery and before extubation. Although details of the manual inflation used and how long before extubation it was applied are not clear, they, like us, failed to demonstrate any beneficial effect on (A –a)DO2 in PACU.

It is known that CPAP at induction prevents the formation of atelectasis and that PEEP applied after a recruitment manoeuvre during anaesthesia prolongs the time taken for atelectasis to reform. We hoped that by applying the same principle at the end of anaesthesia and up until the moment of extubation, we could reduce the effects of atelectasis upon oxygenation in the early recovery period. Our results show that our management strategy, used at the end of anaesthesia, has not provided a significant improvement in patient oxygenation in PACU. There are a number of possible explanations for this. First, (A –a)DO2 may be affected by the FiO2 or the arterial PCO2, but this is unlikely to have affected our results as the values for these factors were the same in each group (Table 1). Secondly, despite using high fresh gas flow rates, our method of applying CPAP during spontaneous respiration may have been ineffective during inspiration, particularly at high inspiratory flow rates. Thirdly, the positive airway pressures used (PEEP and CPAP) after the lung recruitment manoeuvre may have been too low; 10 cm H2O is greater than or equal to that shown to be effective during previous studies at other periods of anesthesia and was the maximum level we felt we could use without risking clinically unacceptable cardiovascular depression. Fourthly, we did not apply CPAP by a facemask after extubation. It was hoped that after extubation, normal vocal fold function would quickly return and provide auto-CPAP by the normal physiological mechanism of vocal fold adduction during expiration, which retards expiration to reduce pulmonary collapse. It seems that this aspect of respiratory muscle activity may not return immediately after extubation. Finally, the mechanism of atelectasis at the end of anaesthesia may differ from that so widely studied at other times. When a patient attempts to cough with a tracheal tube in situ, an unphysiological phenomenon occurs. A normal cough has three phases: an inspiratory phase when lung volume is increased, a compressive phase when there is a forced expiration against a closed glottis, and an expulsive phase when the glottis is quickly opened and extremely high expiratory flows generated. This differs from the expiration reflex which occurs after direct stimulation of the upper airway and involves only the last two phases of the cough reflex. Both these protective reflexes are likely to be active immediately before extubation, and both will be ineffective due to the inability of the glottis to close for the compressive phase. Instead, the expiratory muscles will be contracting strongly against an open airway, so actively reducing lung volume to very low levels, encouraging pulmonary collapse. Although only occurring for a short time, the positive intrathoracic pressure generated during the compressive phase of a cough is very high, and therefore, our strategy of 10 cm H2O positive airway pressure will have had no impact on ameliorating the effects of the cough reflex on lung collapse.

Our study shows that lung recruitment manoeuvres that have been shown to improve atelectasis temporarily may not provide a benefit that persists into the postoperative period.

The patients we studied may have influenced the poor oxygenation observed. In order to be included in the study, all patients were required to have an arterial line inserted on clinical grounds and so were mostly ASA II or III having major and prolonged surgery, and therefore care should be taken when applying our results to other patient populations.

CT is the best technique for quantifying atelectasis but this is impossible during surgery, and is difficult in the early postoperative period when close monitoring is required. We therefore used (A –a)DO2 as a surrogate marker of the severity of atelectasis, allowing an assessment to be made at both the time points in which we were interested.

We did not document whether individual patients coughed, or to what extent, so are unable to comment...
further on whether this mechanism could explain our results. However, it is interesting to speculate whether the trend in recent years to extubate patients when awake enough to protect their airway from aspiration may be causing widespread impairment of lung function in the postoperative period. 

In order to prevent the significantly impaired oxygenation that occurs in PACU, further research is needed both to elucidate the mechanism of atelectasis on emergence from anaesthesia and to evaluate more invasive clinical strategies. 

Future studies using ventilators specifically designed to offer CPAP while spontaneously breathing, or iatrogenic CPAP immediately after extubation, may prove effective in reducing atelectasis. CPAP has been shown to be an effective treatment once postoperative hypoxia has developed, but a functional seal between the mask and patient may be difficult to achieve immediately post-extubation. 

In conclusion, the use of a recruitment manoeuvre and positive airway pressure strategy, which is effective in preventing atelectasis on induction and during anaesthesia, has not been shown to improve oxygenation after operation when used on emergence from anaesthesia and therefore cannot be recommended.

Conflict of interest 
None declared.

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References