Alveolar recruitment improves ventilatory efficiency of the lungs during anesthesia

[Le recrutement alvéolaire améliore l'efficacité ventilatoire des poumons pendant l'anesthésie]

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Purpose: The goal of this study was to analyze the effect of positive end-expiratory pressure (PEEP), with and without a lung recruitment maneuver, on dead space.

Methods: 16 anesthetized patients were sequentially studied in three steps: 1) without PEEP (ZEEP), 2) with 5 cm H_2O of PEEP and 3) with 5 cm H_2O of PEEP after an alveolar recruitment strategy (ARS). Ventilation was maintained constant. The single breath test of CO_2 (SBT-CO_2), arterial oxygenation, end-expiratory lung volume (EELV) and respiratory compliance were recorded every 30 min.

Results: Physiological dead space to tidal volume decreased after ARS (0.45 ± 0.01) compared with ZEEP (0.50 ± 0.07, $P < 0.05$) and PEEP (0.51 ± 0.06, $P < 0.05$). The elimination of CO_2 per breath increased during PEEP (25 ± 3.3 mL·min^{-1}) and ARS (27 ± 3.2 mL·min^{-1}) compared to ZEEP (23 ± 2.6 mL·min^{-1}, $P < 0.05$), although ARS showed larger values than PEEP ($P < 0.05$). Pa-etCO_2 difference was lower after recruitment (0.9 ± 0.5 kPa, $P < 0.05$) compared to ZEEP (1.1 ± 0.5 kPa) and PEEP (1.2 ± 0.5 kPa).

Slope II increased after ARS (63 ± 11%/L, $P < 0.05$) compared with ZEEP (46 ± 7.7%/L) and PEEP (56 ± 10%/L). Slope III decreased significantly after recruitment (0.13 ± 0.07 l/L) compared with ZEEP (0.21 ± 0.11 l/L) and PEEP (0.18 ± 0.10 l/L). The angle between slope II and III decreased only after ARS.

After lung recruitment, PaO_2, EELV, and compliance increased significantly compared with ZEEP and PEEP.

Conclusion: Lung recruitment improved the efficiency of ventilation in anesthetized patients.

Objectif : *Analyser l'effet de la pression télé-expiratoire positive (PEEP) sur l'espace mort, avec et sans recrutement pulmonaire.*

Méthode : *Nous avons réalisé une étude séquentielle en trois étapes auprès de 16 patients anesthésiés : 1) sans PEEP (ZEEP), 2) avec 5 cm H_2O de PEEP et 3) avec 5 cm H_2O de PEEP à la suite d'une stratégie de recrutement alvéolaire (SRA). La ventilation a été maintenue constante. L'épreuve de l'apnée inspiratoire du CO_2, l'oxygénation artérielle, le volume pulmonaire télé-expiratoire (VPTE) et la compliance respiratoire ont été enregistrées toutes les 30 min.*

Résultats : *Le rapport espace mort/volume courant a été réduit après la SRA (0,45 ± 0,01) comparée à la ZEEP (0,50 ± 0,07, $P < 0,05$) et à la PEEP (0,51 ± 0,06, $P < 0,05$). L'élimination du CO_2 pour chaque respiration a augmenté pendant la PEEP (25 ± 3,3 mL·min^{-1}) et la SRA (27 ± 3,2 mL·min^{-1}) comparées à la ZEEP (23 ± 2,6 mL·min^{-1}, $P < 0,05$), même si la SRA a présenté des valeurs plus élevées que la PEEP ($P < 0,05$). La différence Pa-etCO_2 a été plus faible après le recrutement (0,9 ± 0,5 kPa, $P < 0,05$) comparé à la ZEEP (1,1 ± 0,5 kPa) et à la PEEP (1,2 ± 0,5 kPa). La pente II s'est accentuée après la SRA (63 ± 11 %/L, $P < 0,05$) comparée à la ZEEP (46 ± 7,7 %/L) et à la PEEP (56 ± 10 %/L). La pente III s'est abaissée significativement après le recrutement (0,13 ± 0,07 l/L) comparé à la ZEEP (0,21 ± 0,11 l/L) et à la PEEP (0,18 ± 0,10 l/L). L'angle entre les pentes II et III a diminué seulement après la SRA. Après le recrutement alvéolaire, comparé à la ZEEP et à la PEEP, la PaO_2, le VPTE et la compliance ont augmenté significativement.*

Conclusion : *Le recrutement alvéolaire améliore l'efficacité de la ventilation chez les patients anesthésiés.*

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ATELECTASIS observed during general anesthesia causes a decrease in arterial oxygenation, functional residual capacity and respiratory compliance.[1–3] Lung recruitment maneuvers are defined as ventilatory strategies used for treating these negative effects of lung collapse.[4–6] The goal of these maneuvers is to open up the collapsed lung areas and keep them open over time. This "open lung condition", i.e. a lung without collapse, represents the best ventilation/perfusion relationship (V/Q) in a particular lung.[7]

Dead space is defined as "wasted" ventilation and can be studied with the single breath test of CO_2 (SBT-CO_2), the graphic of exhaled CO_2 against tidal volume. The SBT-CO_2 is closely related to the matching of pulmonary ventilation and perfusion giving information on both the efficiency of ventilation and CO_2 exchange.[8–10]

We hypothesized that a lung recruitment maneuver would reduce dead space. The aim of this work was to study the effect of PEEP, with and without a lung recruitment maneuver, on dead space analyzed with the SBT-CO_2.

Methods

After approval by the local Ethics Committee and after obtaining written informed consent, we prospectively studied sixteen patients undergoing open lower abdominal surgery. We enrolled patients ASA II–III, without smoking history or cardiopulmonary uncompensated diseases.

Anesthesia induction was performed with fentanyl 4 $\mu g \cdot kg^{-1}$, thiopental 3 $mg \cdot kg^{-1}$ and vecuronium 0.08 $mg \cdot kg^{-1}$ and maintained with isoflurane and bupivacaine 0.5% through an epidural catheter inserted at L2–3.

After tracheal intubation with a cuffed endotracheal tube, we ventilated the lungs with a Siemens 900 C ventilator (Siemens-Elema, Solna, Sweden). Air leak around the endotracheal tube was detected by comparing inspired-expired tidal volume (VT) measured proximally in the airway. A volume controlled mode was used with a VT of 8 $mL \cdot kg^{-1}$, respiratory rate (RR) between 10 to 15 $beats \cdot min^{-1}$, FIO_2 of 0.5, inspiratory time of 0.3 without pause and, initially, without positive end-expiratory pressure (ZEEP). We increased or decreased alveolar ventilation by adjusting RR to reach an end-tidal CO_2 value of 34 mmHg while maintaining VT constant.

Static respiratory compliance was measured dividing VT by the pressure differences between plateau and total PEEP.

End-expiratory lung volume (EELV) was measured by pushing the expiratory pause button of the Servo 900C for six seconds during the inspiratory pause while releasing PEEP from 5 cm H_2O to ZEEP. Thus, a volume of gas is expelled until functional residual capacity at ambient pressure is reached. The EELV was then determined by subtracting the average value of the latest three normal expiratory tidal volumes before the maneuver from the volume of gas measured. We recorded this volume continuously in a computer and analyzed it off-line. The return of the expiratory flow curve to baseline at the EELV-maneuver was used for checking air trapping.

Carbon dioxide elimination (VCO_2) was calculated by multiplying alveolar ventilation and mean alveolar fraction of CO_2. Oxygen consumption (VO_2) was calculated as the product of alveolar ventilation and inspiratory-expiratory O_2 difference. The respiratory quotient was calculated dividing VCO_2 by VO_2.

The SBT-CO_2 and its variables are explained in Appendix I, available as Additional Material at www.cja-jca.org.

Protocol

We maintained the ventilatory, hemodynamic and metabolic states constant during the study. In each patient we studied three periods sequentially:

1. ZEEP: ventilation with zero PEEP.
2. PEEP: ventilation with 5 cm H_2O of PEEP.
3. ARS: between point 2 and 3, we ventilated the lungs for 20 min without PEEP to reach baseline conditions once again. The ARS is a maneuver assigned to treat pulmonary collapse by reaching the alveolar opening pressure for ten breaths and keeping the lung open with a PEEP level above the lung's closing pressure.[5] In our patients we assume that the lung opening pressure was 40 cm H_2O of peak inspiratory pressure (PIP) and the closing pressure lower than 5 cm H_2O.[1,4,5]

The maneuver was performed in pressure control ventilation following sequential steps (Figure 1):

(a) Ventilatory frequency was set to 15 $breaths \cdot min^{-1}$.
(b) Inspiration/expiration ratio was set at 1:1.
(c) Delta pressure or the pressure difference between PIP and PEEP (PIP/PEEP) was maintained at 20 cm H_2O.
(d) Airway pressures were increased in steps: 25/5 to 30/10 and then to 35/15 cm H_2O. Each step of pressure was maintained for five breaths.
(e) A final PIP/PEEP step of 40/20 cm H_2O was reached and maintained for ten breaths.
(f) After the ten breaths, airway pressures were

TABLE I Dead space data

Variable	*ZEEP*	*PEEP*	*ARS*
VD/VT	0.50 ± 0.07	0.51 ± 0.06	0.45 ± 0.01*†
VDAW (mL)	160 ± 48	161 ± 38	137 ± 32
VDALV (mL)	110 ± 35	113 ± 30	108 ± 32
VDPHYS (mL)	270 ± 54	274 ± 56	246 ± 50
VDALV/VTALV	0.29 ± 0.05	0.28 ± 0.06	0.26 ± 0.04
VDAW/VT	0.30 ± 0.08	0.29 ± 0.04	0.25 ± 0.04†‡
$VTCO_{2,br}$ (mL)	23 ± 2.6	25 ± 3.3*	27 ± 3.2 †‡
VTALV (mL)	340 ± 72	355 ± 71	373 ± 68†‡
Vol I/VT	0.22 ± 0.09	0.21 ± 0.06	0.18 ± 0.06
Vol II/VT	0.35 ± 0.05	0.28 ± 0.05*	0.26 ± 0.05†
Vol III/VT	0.45 ± 0.08	0.51 ± 0.1*	0.57 ± 0.09†‡
Slope II (%/L)	46 ± 7.7	56 ± 10*	63 ± 11†‡
Slope III/N (L^{-1})	0.21 ± 0.11	0.18 ± 0.10*	0.13 ± 0.07†‡
Angle II/III (°)	127 ± 2.1	125 ± 7.7	113 ± 4†‡

PEEP = positive end-expiratory pressure; ZEEP = without positive end-expiratory pressure; ARS = alveolar recruitment strategy; VD/VT = physiologic dead space to tidal volume; VDAW = airway dead space (mL); VDALV = alveolar dead space (mL); VDPHYS = physiologic dead space (mL); VDAW/VTALV = alveolar dead space to tidal volume; VDAW/VT = airway dead space to tidal volume; $VTCO_{2,br}$ = CO_2 elimination per breath (mL); VTALV = alveolar tidal volume (mL); Vol I/VT = volume of phase I to tidal volume; Vol II/VT = volume of phase II to tidal volume; Vol III/VT = volume of phase III to tidal volume; slope II = phase II slope; slope III/N = normalized phase II slope divided by the mean alveolar concentration of CO_2; angle II/III = angle formed between phases II and III slopes (°).

Values are presented as mean ± SD. A P value lower than 0.05 was considered significant. *PEEP against ZEEP, $P < 0.05$; †ARS against ZEEP, $P < 0.05$; ‡ARS against PEEP, $P < 0.05$.

TABLE II Alveolar ventilation and partial pressures of CO_2

Variable	*ZEEP*	*PEEP*	*ARS*
$PaCO_2$ (kPa)	5.1 ± 0.4	5.2 ± 0.6	4.9 ± 0.5
$etCO_2$ (kPa)	4.0 ± 0.3	4.0 ± 0.4	3.9 ± 0.2
Pa-$etCO_2$ (kPa)	1.1 ± 0.5	1.2 ± 0.5	0.9 ± 0.5*†
VA ($L \cdot min^{-1}$)	3.3 ± 0.8	3.4 ± 0.6	3.6 ± 0.8*†

PEEP = positive end-expiratory pressure; ZEEP = without positive end-expiratory pressure; ARS = alveolar recruitment strategy; $PaCO_2$ = arterial partial pressure of CO_2 (kPa); $etCO_2$ = end-tidal partial pressure of CO_2 (kPa); Pa-$etCO_2$ = arterial to end-tidal differences of CO_2 (kPa); and VA = alveolar minute ventilation ($L \cdot min^{-1}$). *ARS against ZEEP, $P < 0.05$; †ARS against PEEP, $P < 0.05$.

gradually decreased returning to the previous setting at 5 cm H_2O of PEEP reassuming a volume controlled ventilation mode.

At the end of each period (30 min), we recorded SBT-CO_2 curves and took blood samples for dead space analysis. Blood specimens were processed and corrected for body temperature within five minutes of extraction by a gas analyzer ABL 510 (Radiometer, Copenhagen, Denmark). Body temperature was measured with an esophageal thermometer.

Comparison of variables among periods was carried out using analysis of variance performed by INSTAT 2.0 (GraphPad, San Diego, CA, USA). If the variance F-statistic was significant the Student-Newman-Keuls post-test detected significant differences. EELV between PEEP and ARS was evaluated by the Student's t test. Values are reported as mean ± SD and a $P < 0.05$ was considered significant.

Results

Nine females and seven males, aged 65 to 80 yr (71.2 ± 4.5), with body mass indices between 24 to 30 (26.8 ± 2.1) undergoing hysterectomies ($n = 3$) and hemicolectomies ($n = 13$) were studied.

Lung recruitment increased dead space variables related to lung efficiency and decreased variables related to inefficiency. PEEP alone did not have same effect on dead space (Table I).

Phase II slopes showed a significant increase with PEEP and ARS although lung recruitment showed the highest values. These steeper slopes were associated with a corresponding decreases in Vol II/VT (Table I).

Normalized phase III slope decreased with PEEP ventilation and showed an additional diminution after ARS. Volume of phase III increased with ARS and PEEP compared with ZEEP. The angle between II-III showed significant differences only after the recruitment maneuver (Table I).

Table II shows partial pressures of CO_2 and the alveolar ventilation at constant minute ventilation. Pa-$etCO_2$ was significantly lower and alveolar ventilation larger after ARS compared with ZEEP and PEEP.

Arterial oxygenation, EELV and respiratory compliance showed a significant increase after lung recruitment compared with ZEEP and PEEP. PEEP without lung recruitment showed compliance values significantly higher than ZEEP but without changes in PaO_2 (Figure 2).

Hemodynamic and metabolic variables stayed constant at all times (Table III, available as Additional Material at www.cja-jca.org).

Discussion

When compared with ZEEP or PEEP, lung recruitment decreased those SBT-CO_2 variables which are related to pulmonary inefficiency and increased the ones related to efficiency. The increased efficiency of ventilation was associated with an increase in arterial oxygenation, expiratory lung volume and respiratory compliance, all parameters commonly used as markers of an "open lung condition".[1,5–7]

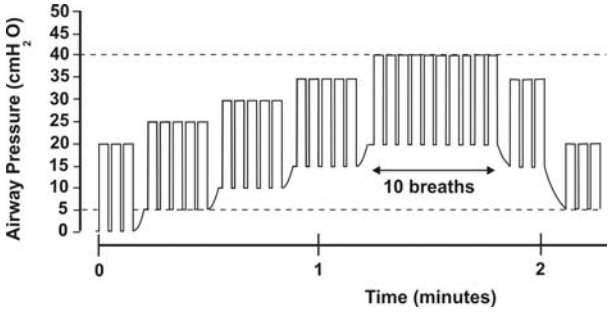

FIGURE 1 Schematic representation of the alveolar recruitment strategy using pressure control ventilation. Rectangles represents tidal volumes set at a pressure difference between peak inspiratory and end-expiratory pressure of 20 cm H_2O. Airway pressures are increased sequentially in steps from 25/5 to 30/10 and then to 35/15 cm H_2O every five breaths. A final step of 40/20 cm H_2O is reached and maintained for ten breaths. After these ten breaths, a progressive decrease to baseline settings is done maintaining a PEEP level of 5 cm H_2O.

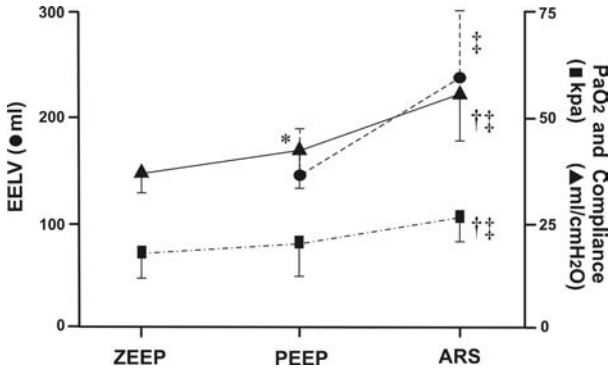

FIGURE 2 PaO_2 (kPa), Respiratory compliance (mL·cm^{-1} H_2O), and end-expiratory lung volume (EELV in mL). ZEEP = no PEEP, PEEP = 5 cm H_2O, and ARS = alveolar recruitment strategy. *PEEP against ZEEP, $P < 0.05$; †ARS against ZEEP, $P < 0.05$; ‡ARS against PEEP, $P < 0.05$.

PEEP without recruitment showed an intermediate effect between ZEEP and ARS in all variables studied. In anesthetized patients low levels of PEEP has a contradictory effect on arterial oxygenation[5] and atelectasis.[11] Studies results agree in that the recruitment of collapsed airways is the main effect of PEEP without a recruitment maneuver.[12,13] Atelectasis treatment requires higher airway pressures than the amount of PEEP commonly used during anesthesia to pop open collapsed alveoli.[1,4,5,11] The incomplete lung recruitment observed with the use of PEEP alone must be the main explanation for our findings.

In contrast to PEEP alone, lung recruitment maneuver increase both, the cross-sectional area of small airways and the alveolar-capillary area, by reversing airway and acinar collapse respectively.[4,11–13] This total recruitment or open lung condition[7] improves the diffusive CO_2 transport at the acinar level and could explain the changes observed in the SBT-CO_2.

Increasing CO_2 diffusion after the ARS moves the interface between convective-diffusive transport mouthward, thus decreasing the VDAW measured by Fowler's method[8] (Appendix I, available as Additional Material at www.cja-jca.org).

Lung recruitment was also associated with an improved efficiency in CO_2 elimination as expressed by a larger $VTCO_{2,br}$ and a lower Pa-etCO_2 at constant VCO_2 and ventilator settings. These results indicate that the area of gas exchange increased and V/Q improved.

Differences between PEEP and ARS in the distribution of gas volumes within the lung may have an impact on gas exchange and respiratory compliance. Analyzing EELV and the volumes of phases I–II–III, we observed that the recruitment maneuver re-distributed the VT away from phases I–II towards the volume of phase III (alveolar gas). Compared with ZEEP, PEEP without a recruitment maneuver increased volume of phase III but at the same time, retained some volume within the inefficient parts of the VT (phases I and II).

Changes in the slope of phases II and III at ZEEP could be explained by the co-existence of acini with different time constants due to aging[14] and partial collapse. Our results resemble those found in asthma and emphysema where both, small airway narrowing and tissue degeneration cause a diffusional resistance to CO_2 transport.[15,16]

Total lung recruitment has a positive effect on CO_2 diffusion as reflected by the changes observed in volumes and slopes of phases II–III after ARS. On the one hand, we think that an increase in the cross-sectional area caused by airway recruitment could improve the CO_2 diffusive transport from alveoli to bronchioli. On the other hand, an increase in the area of gas exchange due to a recruitment of atelectasis improved the diffusive transport from the capillaries to the alveoli (Appendix II, available as Additional Material at www.cja-jca.org).

Some clinical data support our explanation: in children, the multiplication of alveolated airways and pul-

monary capillaries increases the airway's cross-sectional and gas exchange area with a corresponding decrease in phase III slope.[17] In contrast, Schwardt *et al.*[18] showed increased phase III slope in emphysema patients with a known reduction in functional zones of the lung.

The design of the study is linear making possible lung volume deteriorations over time. For this reason, we chose to evaluate the recruitment effect in the last term reflecting possibly the worse condition. Future randomized studies are needed to extrapolate our findings into routine clinical practice.

In summary, the ARS improved the efficiency of ventilation in anesthetized patients. Differences observed in the SBT-CO_2 between PEEP with and without an lung recruitment maneuver, can be explained by the effectiveness of the treatment of pulmonary collapse.

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Lung recruitment during general anesthesia

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INCREASED alveolar to arterial oxygen gradients occur in most patients after the induction of anesthesia. Mild hypoxemia (saturation not below 85%) is noted in up to half of patients under general anesthesia and more severe hypoxemia (saturation below 85%) in up to 20%. The cause of this hypoxemia is an increase in low ventilation to perfusion areas which can become absolute shunts (i.e., no ventilation but persistent perfusion to collapsed alveoli as airways close). The loss of aerated alveoli (atelectasis) occurs as functional residual capacity is reduced below closing capacity and is a consequence of alveolar oxygen re-absorption, dependent lung compression, and loss of surfactant. Atelectasis under general anesthesia routinely involves 5 to 6% of the lung predominantly in dependent areas, can increase up to 50% of the lung in cardiothoracic operations, and can persist for multiple postoperative days.[1] Atelectasis and airway closure explain as much as 74% of gas exchange impairment noted during routine general anesthesia as these poorly ventilated regions are relatively overperfused.[2] Atelectasis may be associated with longer length of hospitals stays,[3,4] pneumonia, initiation of the inflammatory cascade by activating macrophages, and decrease in surfactant production.

Anesthesiologists can minimize atelectasis using four strategies: 1) ventilation with positive end-expiratory pressure (PEEP); 2) lung volume recruitment; 3) maintenance of muscular tone; 4) minimization of pulmonary gas re-absorption. The later technique has led to suggestions that the use of high inspired oxygen prior to induction of anesthesia should be re-evaluated. Preoxygenation with 100% oxygen has not been uniformly adopted by all practioners.[5] The use of 100% oxygen for preoxygenation is based upon the rationale of increasing the safety margin if important hypoxemia develops during the induction of general anesthesia. For example, difficult and prolonged intubations without the ability to hand ventilate may be unexpectedly encountered. Even with preoxygenation, significant hypoxemia can occur in healthy patients prior to resumption of spontaneous ventilation after the use of short-acting muscle relaxants.[6] Rather than eliminate preoxygentation, the use of lower inspired oxygen (60%) may limit the formation of atelectasis.[7] The risk for significant hypoxemia and consequent need of higher inspired oxygen during induction of anesthesia may have a physiological rationale (decreased functional residual capacity with pregnancy or increase metabolic rate in newborns). In the presence of lung pathology and need for high-inspired oxygen even prior to anesthesia, the use of 100% oxygen during preoxygenation seems more defensible.

More recently the use of lung recruitment maneuvers during routine anesthesia has been suggested in order to minimize atelectasis and the need for high inspired oxygen. The Canadian Journal of Anesthesia has added to this discussion by publishing such an article by Tusman *et al.*[8] The rationale of using recruitment maneuvres early after induction of anesthesia is to forestall the atelectasis that can occur after induction. An atelectatic lung may be re-expanded during anesthesia by applying a vital capacity maneuver or by applying an inflation pressure of 40 cm H_2O to the lungs.[9] Rothen *et al.*[10] showed that atelectasis was reduced mostly during the first seven to eight seconds of a vital capacity maneuver with virtually no change after this time. A brief lung recruitment maneuver potentially avoids cardiovascular collapse associated with more prolonged high inspiratory pressure.[11] The composition of inspiratory gas used in the recruitment maneuver plays an important role in the duration of its effects. Lung re-expansion is more sustained if 40% oxygen in nitrogen is used. If 100% oxygen is used after re-expansion, lung collapse recurs within a few minutes. After a recruitment maneuver, the lack of oxygenation improvement during ventilation using low-oxygen gas mixtures suggests that low ventilation-perfusion lung areas are still present.[10]

Most information about lung recruitment maneuvers relates to pressure-limited lung-protective ventilatory strategies in order to prevent atelectasis when

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small tidal volumes and low to moderate PEEP levels are used.[12] Bond *et al.*[13] showed that oxygenation improved after performing recruitment maneuvers during volume-controlled ventilation using low tidal volume ventilation (7 $mL \cdot kg^{-1}$) and zero end-expiratory pressure. Rismensberger *et al.*[14] showed that a single recruitment maneuver resulted in better oxygenation at a PEEP level less than the lower inflection point of the pressure-volume curve compared with a PEEP level higher than the lower inflection point of the pressure-volume curve and no recruitment maneuver. The lower inflection "point" of the lung pressure-volume curve is noted when airway opening occurs most frequently - not the inspiratory pressure at which lung unit closure no longer occurs. To maximally open the lung and prevent collapse of unstable lung units, it is necessary to apply inspiratory pressures that produce total lung capacity.[15] Opening of lung units occurs along the entire length of the inspiratory limb of the pressure-volume curve so that tidal opening and closing of some lung units occur at virtually any tidal volume unless the lungs were fully recruited before initiating ventilatory support. Refractory but potentially recruitable lung units tend to be located in dependent zones and require high and sustained airway pressures to open. The inspired pressures required may substantially exceed those sufficient to achieve total lung capacity in a normal lung.[16] This specific type of recruitment maneuver - the stepwise application of moderately high airway pressure - is less likely to help either patients with primary (pulmonary, as opposed to extrapulmonary) causes of the acute respiratory distress syndrome, those who are receiving ventilatory support in the prone position, or those with atelectasis due to mechanical forces.[17] Laboratory studies demonstrate that surfactant depletion and oleic models of lung injury are highly recruitable, whereas lung injury inflicted by pneumonia and purely mechanical forces are not.[14] Moreover, any improved oxygenation resulting from the recruitment maneuver will be transient unless sufficient PEEP is applied to sustain the benefit.

Recruitment maneuvers are not entirely benign. They may do harm by redirecting blood flow due to high alveolar pressure subsequently interfering with hypoxic pulmonary vasoconstriction. The result is hypoxemia during and after the recruitment maneuver. Hypotension is a risk in patients who are hypovolemic, in those with unusually high "transmission" ratios of lung to chest wall compliance, and in those whose adaptive cardiac reserves are blunted by intrinsic disease or medications. Barotrauma should also be considered whenever high and sustained alveolar pressures are used.

Clinical studies employing recruitment maneuvers have shown atelectasis may be improved in healthy patients.[18] Routine use has not shown to improve outcomes in at least one patient group[19] with a high incidence of postoperative atelectasis. A number of technical questions regarding recruitment maneuvers remain unanswered. These include what is the ideal inspiratory pressure to be used, what is the time length for each recruitment maneuver, what is the repetition frequency for multiple recruitment maneuvers, and what preoperative, operative, or postoperative times are most critical in the application of recruitment maneuvers.

The more difficult, unanswered question is demonstrating a causal relationship between preventing atelectasis and improved patient outcome (such as the incidence of postoperative pneumonia, postoperative length of hospital stay, need or duration of mechanical ventilation). Thus, at this time, the risk *vs* benefit of routine recruitment maneuvers during general anesthesia must be questioned. Similar questions should be asked about more technologically sophisticated techniques of ventilation during anesthesia such as airway pressure release ventilation[20] which may also prevent atelectasis by avoiding the use of supplemental oxygen and maintenance of spontaneous ventilatory effort. The use of therapy that prevents or improves atelectasis but has not been shown to improve patient outcomes is not restricted to anesthesiologists. These therapies include physiotherapy, intermittent positive pressure breathing, and bronchoscopy. It would seem that, yet again, the therapy has been applied before knowing that the problem is being solved. Until anesthesiologists insist upon patient outcome trials before adopting new techniques and therapies, even when the rationale of these therapies would seem most reasonable, we will continue to be limited to expert opinion.

Le recrutement alvéolaire pendant l'anesthésie générale

Des gradients alvéolo-artériels d'oxygène accrus sont observés chez la plupart des patients après l'induction de l'anesthésie. Une hypoxémie légère (saturation non inférieure à 85 %) atteint près de la moitié des patients sous anesthésie générale et une hypoxémie plus sévère

(saturation inférieure à 85 %), près de 20 %. Cette hypoxémie est causée par une augmentation de l'hypoventilation vers les aires de perfusion, ce qui peut évoluer vers des shunts absolus, c'est-à-dire aucune ventilation mais une perfusion persistante vers les alvéoles collabées au moment de la fermeture des voies aériennes. La perte d'alvéoles aérées (atélectasie) survient avec la réduction de la capacité résiduelle fonctionnelle sous le volume de fermeture et est une conséquence de la réabsorption d'oxygène alvéolaire, de la compression du poumon déclive et de la perte de surfactant. L'atélectasie sous anesthésie générale touche habituellement de 5 à 6 % du poumon, surtout dans les régions déclives, peut affecter jusqu'à 50 % du poumon pendant la chirurgie cardio-thoracique et peut persister pour plusieurs jours postopératoires.[1] L'atélectasie et la fermeture des voies aériennes peuvent expliquer jusqu'à 74 % de l'altération de l'échange gazeux pendant l'anesthésie générale régulière, car ces régions hypoventilées sont relativement surperfusées.[2] L'atélectasie peut être associée avec des séjours hospitaliers prolongés,[3,4] la pneumonie, le début d'une cascade inflammatoire par l'activation des macrophages, et la baisse de production de surfactant.

Les anesthésiologistes peuvent réduire l'atélectasie de quatre façons : 1) la ventilation à pression télé-expiratoire positive (PEEP) ; 2) le recrutement du volume pulmonaire ; 3) le maintien du tonus musculaire ; 4) la réduction de la réabsorption pulmonaire des gaz. Selon cette dernière technique nous pourrions avoir à réévaluer l'administration de fortes concentrations d'oxygène avant l'induction de l'anesthésie. La préoxygénation avec 100 % d'oxygène n'a pas été uniformément adoptée par les praticiens.[5] Elle est fondée sur l'idée d'accroître la marge de sécurité au cas où une importante hypoxémie se développerait pendant l'induction de l'anesthésie générale. Par exemple, il est possible que nous soyons subitement confrontés à une intubation difficile sans possibilité de ventiler manuellement. Même avec la préoxygénation, une importante hypoxémie peut survenir chez des patients sains avant le retour de la ventilation spontanée à la suite de l'utilisation de myorelaxants à action brève.[6] Plutôt que d'éliminer la préoxygénation, l'administration d'oxygène à 60 % pourrait limiter la formation d'atélectasie.[7] Le risque d'une importante hypoxémie et de la nécessité subséquente d'administrer de l'oxygène à forte concentration pendant l'induction de l'anesthésie peut avoir un fondement physiologique. En effet, pensons à la réduction de la capacité résiduelle fonctionnelle pendant la grossesse ou à l'augmentation du rythme métabolique chez les nouveau-nés. En présence d'une pathologie pulmonaire et de la nécessité de forte concentration d'oxygène inspirée juste avant l'anesthésie, l'usage d'oxygène à 100 % pendant la préoxygénation semble plus justifiable.

Plus récemment, l'usage régulier de manœuvres de recrutement alvéolaire pendant l'anesthésie a été suggéré pour réduire l'atélectasie et la nécessité d'oxygène inspiré à forte concentration. Le Journal canadien d'anesthésie contribue à cette discussion en publiant l'article de Tusman *et coll.*[8] L'idée d'utiliser le recrutement peu après l'induction de l'anesthésie est de prévenir l'atélectasie qui pourrait survenir après l'induction. Un poumon atélectasique peut être redilaté pendant l'anesthésie par l'application d'une manœuvre d'inspiration à capacité vitale ou d'une pression d'inspiration à 40 cm H_2O.[9] Rothen *et coll.*[10] ont montré que l'atélectasie a été réduite surtout pendant les sept ou huit premières secondes d'une manœuvre d'inspiration à capacité vitale sans changement significatif après ce temps. Un bref recrutement alvéolaire peut éviter un collapsus cardiovasculaire associé à une pression inspiratoire élevée prolongée.[11] La composition des gaz inspirés, utilisés pour le recrutement, joue un rôle important dans la durée de leurs effets. La redilatation des poumons dure plus longtemps avec 40 % d'oxygène dans de l'azote. Si on administre 100 % d'oxygène après la réexpansion, l'affaissement du poumon revient en quelques minutes. Après le recrutement, l'absence d'amélioration de l'oxygénation pendant la ventilation avec des mélanges gazeux à faible concentration d'oxygène suppose qu'il y a encore des régions du poumon avec des bas ratios de ventilation/perfusion.[10]

La plupart des informations sur les manœuvres de recrutement alvéolaire ont trait aux stratégies de protection pulmonaire limitées par la pression et visent à prévenir l'atélectasie quand on utilise des petits volumes courants et des niveaux de PEEP de faibles à modérés.[12] Bond *et coll.*[13] ont montré que l'oxygénation s'améliore après le recrutement pendant la ventilation limitée par le volume qui utilisent une ventilation à faible volume courant (7 $mL \cdot kg^{-1}$) et aucune pression télé-expiratoire. Rismensberger *et coll.*[14] ont montré qu'une seule manœuvre de recrutement permet une meilleure oxygénation à un niveau de PEEP plus bas que le point d'inflexion inférieur de la courbe pression-volume, comparé à un niveau de PEEP plus élevé que ce point de la courbe et sans manœuvre de recrutement. Le «point» d'inflexion inférieur de la courbe de pression-volume pulmonaire est identifié quand l'ouverture des voies aériennes se fait le plus fréquemment – et non lorsque la pression inspiratoire à laquelle la fermeture des unités alvéo-

laires n'est plus manifeste. Pour ouvrir les poumons au maximum et prévenir l'affaissement d'unités alvéolaires instables, il est nécessaire d'appliquer des pressions inspiratoires qui puissent atteindre une capacité pulmonaire totale.[15] L'ouverture des unités alvéolaires survient tout au long de la branche inspiratoire de la courbe pression-volume de sorte que l'ouverture et la fermeture cycliques de certaines unités alvéolaires surviennent pour presque tout volume courant à moins que les poumons soient complètement recrutés avant le début de l'assistance ventilatoire. Les unités alvéolaires réfractaires, mais possiblement recrutables, surtout localisées dans les zones déclives, nécessitent des pressions élevées et soutenues pour s'ouvrir. Les pressions inspirées nécessaires peuvent dépasser considérablement celles qui suffisent à atteindre la capacité pulmonaire totale d'un poumon normal.[16] Ce type de recrutement spécifique - l'application progressive d'une pression modérément élevée dans les voies respiratoires - n'aidera probablement pas les patients atteints du syndrome de détresse respiratoire aiguë de causes primaires (pulmonaires, opposées à extrapulmonaires), ceux qui reçoivent une assistance respiratoire en décubitus ventral ou ceux qui présentent de l'atélectasie d'origine mécanique.[17] Des études en laboratoire démontrent que la déplétion du surfactant et les modèles oléiques de lésions pulmonaires sont hautement recrutables, tandis que les lésions pulmonaires causées par une pneumonie et des forces purement mécaniques ne le sont pas.[14] Qui plus est, toute amélioration de l'oxygénation résultant d'une manœuvre de recrutement sera transitoire à moins qu'une PEEP suffisante soit appliquée pour maintenir les bienfaits.

Le recrutement n'est pas tout à fait inoffensif. Il peut nuire en redirigeant le débit sanguin à cause de la pression alvéolaire élevée qui, subséquemment, interfère avec la vasoconstriction pulmonaire hypoxique. Il en résulte une hypoxémie pendant et après la manœuvre de recrutement. L'hypotension est un risque chez les patients hypovolémiques, chez ceux qui présentent des rapports de «transmission» exceptionnellement élevés de compliance entre le poumon et la paroi thoracique et chez ceux dont les réserves cardiaques adaptatives sont émoussées par une affection intrinsèque ou des médicaments. Un barotrauma doit aussi être envisagé quand on utilise des pressions alvéolaires élevées et soutenues.

Des études cliniques sur le recrutement ont montré que l'atélectasie peut être corrigée chez des patients sains.[18] On a montré, auprès d'au moins un groupe de patients[19] qui présentaient une incidence élevée d'atélectasie, que son utilisation régulière n'améliorait pas l'évolution. Un certain nombre de questions techniques demeurent sans réponse. Quelle est, par exemple, la pression inspiratoire idéale à utiliser, combien de temps doit durer une manœuvre de recrutement, quelle doit être la fréquence de répétition de manœuvres multiples et quels moments préopératoires, opératoires ou postopératoires sont les plus critiques dans l'application de manœuvres de recrutement.

La question la plus difficile, toujours sans réponse, concerne la démonstration d'une relation causale entre la prévention de l'atélectasie et l'amélioration de l'état du patient (comme l'incidence de pneumonie postopératoire, la longueur du séjour hospitalier postopératoire, la nécessité et la durée de la ventilation mécanique). À présent il faut donc revoir le rapport risque-avantage des manœuvres régulières de recrutement pendant l'anesthésie générale. Il faut aussi s'interroger sur la ventilation de haute technicité utilisée pendant l'anesthésie comme la ventilation avec dépressurisation expiratoire intermittente[20] qui peut aussi prévenir l'atélectasie en évitant l'usage d'oxygène supplémentaire et s'interroger sur le maintien d'une ventilation spontanée. Les anesthésiologistes ne sont pas les seuls à utiliser un traitement qui prévient ou réduit l'atélectasie sans améliorer nécessairement l'évolution du patient. Ces traitements comprennent la physiothérapie, la respiration au moyen de respirateurs à pression positive intermittente et la bronchoscopie. Il semble, encore une fois, que la thérapie ait été appliquée avant de savoir si le problème se règle. Aussi longtemps que nous ne mettrons pas l'accent sur des d'essais cliniques avant d'adopter de nouvelles techniques et thérapies, même si la logique de ces thérapies semble raisonnable, nous continuerons d'être limités à une opinion d'expert.

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