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Independent lung ventilation in patients with unilateral pulmonary contusion. Monitoring with compliance and $EtCO_2$

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The last paper from Prof. Antonio Brienza, who recently died, is published herewith. The real value of one man may only be completely appreciated in the final balance. Starting from nothing, Antonio created Intensive Care in the South of Italy and from there his group had a deep impact on our scientific community. It is difficult to realize, nowadays, the tremendous amount of effort and personal time Antonio had to spend to reach these targets. Remembering gives value to our lives. Antonio is still with us. Thanks, Professor Luciano Gattinoni, Istituto di Anestesia e Rianimazione, Via F. Sforza 33, 20122 Milano, Italy,
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Abstract *Objective*: a) to describe a non-barotraumatic ventilatory setting for independent lung ventilation (ILV); b) to determine the utility of single lung end-tidal CO_2 ($EtCO_2$) monitoring to evaluate the ventilation to perfusion (V/Q) matching in each lung during ILV and for ILV weaning.
Design: prospective study.
Setting: general intensive care unit in a university teaching hospital.
Patients: twelve patients with unilateral thoracic trauma needing ILV.
Interventions and results: ILV was started with each lung ventilated with the same tidal volume (Vt): plateau airway pressure (Pplat) was 34.2 ± 3.2 cmH2O in diseased lungs (DL) and 18.1 ± 1.9 cmH2O in normal lungs (NL) ($P < 0.01$). Static compliance (Cst) was 9.9 ± 1.1 ml/cmH_2O in DL and 19.3 ± 1.7 ml/cmH_2O in NL ($P < 0.01$). EtCO2 was 22.5 ± 2.2 mmHg in DL and 36.6 ± 1.9 mmHg in NL ($P < 0.01$). PaO_2/FiO_2 was at 151 ± 20. PEEP was applied on the DL and each lung was ventilated with a Vt that developed Pplat ≤26 cmH2O. With this setting, Vt given to the NL was unchanged, whereas it was reduced in the DL (238 ± 30 ml vs 350 ± 31 ml; $P < 0.01$). Cst and EtCO2 were still significantly lower in the DL ($P < 0.01$, respectively), while the PaO_2/FiO_2 ratio remained unchanged. Vt was then progressively increased in the DL as Pplat decreased, but remained unchanged in the NL. ILV was discontinued when Vt, Cst and $EtCO_2$ were the same in each lung. PaO_2/FiO_2 ratio had then increased to 295 ± 18.
Conclusions: a) during ILV, adequate oxygenation and a reduction in V/Q mismatch can be obtained by setting Vt and PEEP to keep Pplat below a safe threshold for barotrauma; b) measurement of single lung EtCO2 can be useful to evaluate progressive V/Q matching.

Keywords ILV · Vt · $EtCO_2$ · Pplat · Compliance · Barotrauma

Introduction

Independent lung ventilation (ILV) has been applied in severe asymmetric or unilateral lung injury to improve hypoxemia refractory to conventional mechanical ventilation (MV) and positive end-expiratory pressure (PEEP) application [1, 2, 3, 4, 5]. The rationale for ILV is based on the presence of asymmetry in lung pathology producing differences in compliance (Cst) [4, 6]. In this case, during MV, tidal volume (Vt) is mostly diverted towards the less diseased lung (NL), possibly resulting in overinflation and parenchymal damage due to barotrauma [2], while a share of pulmonary perfusion is diverted to the diseased lung (DL), so increasing blood shunting [1, 2, 5, 6, 7]. Under such conditions, ILV has been proposed to ventilate the DL, while avoiding hyperinflation in the NL, thus improving the ventilation/perfusion (V/Q) matching of each lung [1, 2, 3, 5, 8, 9, 10, 11].

Usually ILV is set with the same Vt in both lungs (with an accepted difference < 100 ml), [1, 3, 9, 10, 12, 13, 14]. However, this setting produces higher plateau airway pressure (Pplat), [2, 3, 12, 13] and lower end-tidal CO_2 ($EtCO_2$) in the DL.

Recent studies have demonstrated that in acute respiratory distress syndrome (ARDS), high levels of airways pressure and/or Vt can worsen previous lung damage [18, 19]. Vt reduction with PEEP application to promote alveolar recruitment and improve arterial oxygenation has been suggested in the management of patients with ARDS to limit the risk of ventilation-induced lung injury [18, 19, 20, 21, 23] and a level of Pplat ≤30 cmH2O is considered a safe threshold [20, 21]. If these ventilatory strategies are transposed to the DL in unilateral lung injury, the ventilatory setting in the DL should be chosen to avoid high Pplat.

In two recently published cases [22], we hypothesized that just separating the ventilation of each lung is not enough, because at the beginning of ILV the two lungs are still asymmetric. Accordingly, we set Vt on each lung at a value generating a Pplat ≤26cmH_2O, and obtained a good and stable PaO_2/FiO_2 ratio. As reported in literature, $EtCO_2$ in our patients was lower in the DL than in the NL.

Since pulmonary injury is usually inhomogeneous [23], the finding of different $EtCO_2$ and Pplat between the two lungs could be explained by the presence in the DL, even during ILV, of relatively overinflated and underperfused "normal areas", contributing to the dead-space effect, together with the contused areas responsible for the blood shunting. Therefore, in these patients it is mandatory to optimize the ventilatory therapy and to continuously monitor the ventilation/perfusion matching.

Accordingly, this study was launched to test our hypothesis on a larger number of patients with unilateral lung injury, and to: a) describe a ventilatory setting for ILV in patients with unilateral pulmonary contusion based on Vt reduction and selective PEEP to the diseased lung; b) determine the utility of single lung end-tidal CO_2 monitoring to evaluate the V/Q matching in each lung during ILV and for ILV weaning.

Material and methods

Patients

The study was performed prospectively in twelve thoracic trauma patients (nine males, three females), without preexisting lung disease, admitted to the Intensive Care Unit of the University of Bari from January 1993 to July 1998. The patients presented with asymmetric lung contusion and acute respiratory failure that necessitated mechanical ventilation. The study protocol was approved by the local Ethics Committee and each patient or next of kin had given informed consent. Patient data are summarized in Table 1.

Three patients (#2, 5, 7) presented with thoracic trauma, while the other nine had polytrauma with abdominal and/or limb fractures. Patient #8 had head trauma.

Lung contusion was diagnosed in each patient on the basis of clinical history, chest X-ray, and thoracic computed tomography (CT) scan [24]. All patients except three (#3, 6, 8), had thoracic drains: no patient presented pneumothorax or air leakage from thoracic drains at the moment of the study. The severity of injury was assessed on admission to the ICU by the Injury Severity Score (ISS) [25] and by the Simplified Acute Physiology Score (SAPS) [26].

Measurements

During ILV, the patients were sedated (Propofol 2–3 mg/kg per hour and Fentanyl 0.5 μ/kg per hour) to avoid displacement of the double lumen tube, reduce the risk of upper airways lesions due to coughing, and ensure steady state conditions during measurements. Muscle paralysis (Vecuronium 0.08 mg/kg per hour) was necessary only in patients #2, 3, 4, and 8 during the first 24 h of ILV. The absence of muscular activity was verified by checking the Paw curve and capnogram shapes.

Airway pressure and flow (V) were measured respectively from the pressure transducers and pneumotachograph incorporated in the Servo 300 and by reading the digital values on display. The ventilator transducers were calibrated in vitro by comparison with Validyne MP 45 ± 100 cmH2O transducers and with a heated pneumotachograph (No2, Fleisch, Lausanne, Switzerland), connected to the Validyne transducer. Pplat and end-expiratory pressure (EEP) were measured holding, respectively, the end-inspiratory and end-expiratory knobs for 2 s. Vt and RR were also read on the ventilator digital display; static compliance (Cst) was computed for each lung as the Vt/(Pplat-EEP) ratio. In patients with thoracic drains in place, respiratory mechanics was measured when inspired and expired Vt were equivalent. PEEP on the contused lungs was titrated using the "constant-flow" technique [27], i.e., the level of PEEP that induced an upward displacement of the dynamic volume-pressure (V/P) curve on the ventilator display at different PEEP levels, from 3 cmH2O to 10 cmH2O.

Capnograms were continuously measured for each lung, via two mainstream capnographs (Hewlett Packard, Los Angeles, Calif., USA) located between each lumen tube and the respiratory

Table 1 Patients' characteristics. (*SAPS* simplified acute physiology score, *ISS* injury severity score, *S* survived,; *NS* did not survive)

Pts	Sex	Age (years)	Diagnosis	SAPS	ISS	Time laps Before ILV	PaO_2/FiO_2 At study entry	Days of ILV	PEEP (cmH_2O)	Outcome
1	F	58	Multiple left rib fractures. Left arm fracture	10	25	6 h	84	30 h	7	S
2	M	45	Massive right hemothorax. Multiple right rib fractures	10	9	48 h	85	4	8	S
3	M	59	Multiple right rib fractures. Right arm fracture	10	26	30 h	70	7	5	S
4	M	20	Thoraco-abdominal trauma. Bilateral legs fracture	14	34	2 h	91	5	8	NS
5	M	19	Massive right hemothorax	5	9	60 h	87	30 h	10	S
6	M	24	Multiple right rib fractures. Right arm fracture	11	18	24 h	99	2	8	S
7	M	50	Multiple right rib fractures	9	9	5 h	100	3	10	S
8	F	25	Left lung contusion	14	9	10 h	95	1	5	S
9	M	35	Multiple left rib fractures. Left arm fracture	9	26	4 h	98	2	5	S
10	F	40	Thoraco abdominal trauma	10	25	12 h	87	4	7	S
11	M	30	Multiple left rib fractures	8	9	36 h	100	3	8	S
12	M	22	Multiple right rib fractures. Right arm and leg fracture	9	9	24 h	80	4	7	NS

circuits and recorded on a strip chart. The highest CO_2 concentration point at end-expiration was taken as $EtCO_2$. The values of $EtCO_2$ presented are the mean of 10–12 breaths (1 min).

In patients #1, 2, 3 hemodynamic monitoring was performed with a 7F pulmonary artery Swan-Ganz catheter (Edwards Lab., Irvine, Calif., USA, Model 93A-431H 7.5F) inserted through the right internal jugular or the left subclavian vein. The pulmonary arterial catheters were connected to quartz pressure transducers (Hewlett-Packard P1290A Cupertino, Calif., USA) to measure the pulmonary artery pressure (PAP), right atrial pressure (RAP), and pulmonary artery occlusion pressure (PAOP). The midaxillary line was taken as zero reference level, with the patient supine and horizontal, and all pressures were read at end-expiration. All signals were recorded with an eight-channel strip-chart recorder (Hewlett-Packard 7719A). Cardiac output (CO) was measured by the thermodilution technique (3300 CO Computer, Abbott, North Chicago, Ill., USA), using an injection of 5 ml of cold (< 5 °C) 5% dextrose solution.

Study protocol

On admission to the ICU, all patients were ventilated with intermittent positive-pressure ventilation (IPPV) with square-wave flow, tidal volume (Vt) 10–12 ml/kg, respiratory rate (RR) 12–15/breaths, I/E ratio 0.33, without PEEP. The indication for ILV was based on the conventional criteria: a) chest X-ray and thoracic CT scan showing asymmetric lung contusion; b) failure to improve or even worsening oxygenation with IPPV and PEEP progressively increased from 5 cmH2O to 10 cmH2O [1, 3, 9, 10, 16]. ILV was applied after 22 ± 19 h (range 2–60), of IPPV. Three patients had a tracheostomy tube already in place at the moment of the study, six underwent tracheostomy before starting ILV, and three were ventilated by an orotracheal tube during their entire stay in the ICU. ILV was performed via a left double-lumen tracheostomy tube (Rushelit Rush, Waiblingen, Germany) in patients #1–5 and 9–12, and via a left double-lumen oral tube (Rushelit Rush AG lab, Waiblingen, Germany) in patients #6–8.

ILV was performed using two non-synchronized Servo 300 ventilators, each equipped with a monitor display (Siemens Elema, Solna Sweden). Respiratory rate (13–15/min), I/E ratio (0.33), FiO_2, and control mode ventilation with square-wave flow were identical in both lungs. The study times were as follows:

1. Before ILV. One hour before ILV, Pplat, Cst, $EtCO_2$, PaO_2/FiO_2, and $PaCO_2$ were measured.
2. ILV with the same VT. When starting ILV, each lung received a Vt of 4–5 ml/kg body weight; Pplat, Cst, $EtCO_2$, PaO_2/FiO_2, and $PaCO_2$ were measured after a stabilization time of 30 min.
3. T_1. On the contused lung, PEEP was applied. After setting the PEEP level, Pplat was measured; if it exceeded 26 cmH_2O, Vt was reduced until Pplat was ≤26 cmH2O. With this ventilatory schema, Vt was always reduced in DLs and never in NLs.

During the course of ILV, Vt did not change in the NL. In the DL, when Pplat decreased by ≥5 cmH2O, Vt was increased to a value still resulting in a Pplat ≤26 cmH2O, while the PEEP level was not changed. Pplat, Cst, $EtCO_2$, PaO_2/FiO_2, and $PaCO_2$ were measured after 30 min.

4. The discontinuation of ILV was programmed when the Vt and Cst of the two lungs differed by < 100 ml and 20% [10], respectively. Thirty minutes before stopping ILV Pplat, Cst, $EtCO_2$, PaO_2/FiO_2, and $PaCO_2$ were measured.
5. After ILV. One hour after reinstitution of IPPV with a monolumen endotracheal tube, Pplat, Cst, $EtCO_2$, PaO_2/FiO_2 and $PaCO_2$ were measured.

Table 2 Patients' respiratory mechanics and oxygenation data. (*NL* normal lungs, *DL* diseased lungs, *T1* 30 min after reducing Vt in DL, *T2* 30 min before stopping ILV, *Vt* tidal volume, *Pplat* plateau airways pressure, *Cst* static compliance, $EtCO_2$ end-tidal carbon dioxide tension, $PaCO_2$ arterial carbon dioxide tension, PaO_2 arterial oxygen tension, FiO_2 fraction of inspired oxygen, *mBP* mean blood pressure)

	Vt (ml)		Pplat (cmH_2O)		Cst (ml/cmH_2O)		$EtCO_2$ (mmHg)		PaO_2/FiO_2	$PaCO_2$ (mmHg)	mBP (mmHg)
	NL	DL	NL	DL	NL	DL	NL	DL			
Before ILV	676 ± 56		36 ± 2		18.8 ± 2.2		22.1 ± 1.3		99 ± 9	39.1 ± 2.3	85.8 ± 26.2
ILV with same Vt	353 ± 33[a]	339 ± 31[a]	18 ± 2[a]	34 ± 3[a,b]	19.3 ± 1.8	9.9 ± 1.1[a,b]	36.7 ± 1.9[a]	22.5 ± 2.2 [b]	150.7 ± 20[a]	39.5 ± 2.1	86.2 ± 15.9
T1	351 ± 31[a]	238 ± 30[a, b, c]	18 ± 2[a]	26 ± 0[a, b, c]	19.8 ± 1.9	12.9 ± 2.0[a,b]	36.8 ± 2.6[a]	25.4 ± 2.2[a,b,c]	150.2 ± 18[a]	47.7 ± 1.9[a,c]	91.2 ± 10.8
T2	352 ± 35[a]	345 ± 31[a,d]	18 ± 2[a]	24 ± 1[a,b,d]	19.5 ± 3.5	20.5 ± 2.4 [c,d]	36.5 ± 1.8[a]	36.2 ± 2.3[a,d]	295.7 ± 26[a,c,d]	40.1 ± 0.9 [d]	91.3 ± 6.7
After ILV	685 ± 52		17 ± 1[a,e]		39.8 ± 4.2[a]		36.5 ± 1.5[a]		317 ± 43[a,c]	40.1 ± 0.9	90.3 ± 5.7
Friedman's Anova	[f]		[f]		[f]		[f]		[f]	[f]	NS

[a] Wilcoxon matched pair: $P < 0.001$ vs before
[b] Mann Whitney U-test: $P < 0.001$ NL vs PL
[c] $P < 0.001$ vs ILV with same Vt
[d] $P < 0.001$ vs T1
[e] H $P < 0.001$ vs T2
[f] Friedman's Anova: $P < 0.001$

All patients underwent fiberoptic bronchoscopy both before the positioning of the double lumen tube and after its removal, to check whether any airways injury was present.

During the study period all patients were hemodynamically stable.

Data analysis

All data are presented as mean ± standard deviation (SD). Friedman's analysis of variance and Wilcoxon matched-pairs tests were used to assess differences between conditions, and Mann Whitney U-test was used to assess differences between the two lungs at each study step. A *P*-value < 0.05 was taken as significant.

Results

ILV lasted for a mean of 3.1 ± 1.8 days (range 30 h–7 days) (Table 1). Patients #4 and #12 developed sepsis and died, respectively, 30 and 25 days after complete recovery from the asymmetric lung disease. All the other patients were discharged from the ICU after a mean stay of 29.1 ± 26.6 days (range 60 h–79 days).

Gas exchange

PaO_2/FiO_2 was at 99 ± 9 mmHg before ILV (Table 2, Fig. 1): this ratio increased significantly when ILV was started with the same Vt ($P < 0.001$), remained stable on T_1, and improved gradually during treatment on ILV: on T_2 it reached 296 ± 26 mmHg ($P < 0.01$ vs Before ILV with the same Vt and T_1), and remained stable after ILV (NS vs T_2). $PaCO_2$ was 39 ± 2 mmHg before ILV, and it remained stable during ILV with the same Vt. On T_1, it increased to 48 ± 2 mmHg ($P < 0.001$), while on T_2 it returned to the normocapnic range ($P < 0.001$ vs T_1; NS vs ILV with the same Vt), and remained stable after ILV.

Lung mechanics

Before ILV Pplat was 36.2 ± 2 cmH2O, Cst was 18.9 ± 2.2 ml/cmH_2O, and $EtCO_2$ 22 ± 1 mmHg. During ILV with *the same Vt in both lungs*, there was a significant difference in Pplat ($P < 0.001$), $EtCO_2$ ($P < 0.001$), and Cst ($P < 0.001$) between the NL and the DL (Table 2, Fig. 2).

On T_1 and T_2, *in the NL*, Vt, Pplat, Cst, and $EtCO_2$ remained unchanged compared to ILV with the same Vt. *In the DL*, on T_1, Vt was 238 ± 30 ml ($P < 0.001$ vs NL), and it was raised from T_1 to T_2 ($P < 0.001$ vs T_1; NS vs NL); Pplat was significantly lower and Cst significantly higher on T_1 than during ILV with the same Vt ($P < 0.001$). On T_2, Pplat remained unchanged while Cst significantly increased ($P < 0.001$). On T_1, $EtCO_2$ was significantly increased, but still lower than $EtCO_2$ of the NL ($P < 0.001$). On T_2, there was no difference in $EtCO_2$ between the NL and the DL (Table 2, Fig. 2).

After ILV, Vt was 685 ± 52 ml (NS vs before), Pplat was significantly lower than before ILV, ($P < 0.001$), while Cst and $EtCO_2$ were significantly higher ($P < 0.001$ vs before). PEEP to the DL was 7.3 ± 1.7 cmH2O, and remained stable throughout the study period.

Haemodynamics

Patients #1, 2, 3 hemodynamic data are reported on Table 3. Qs/Qt decreased on T_2, while none of the other hemodynamic variables changed significantly during ILV (Table 3).

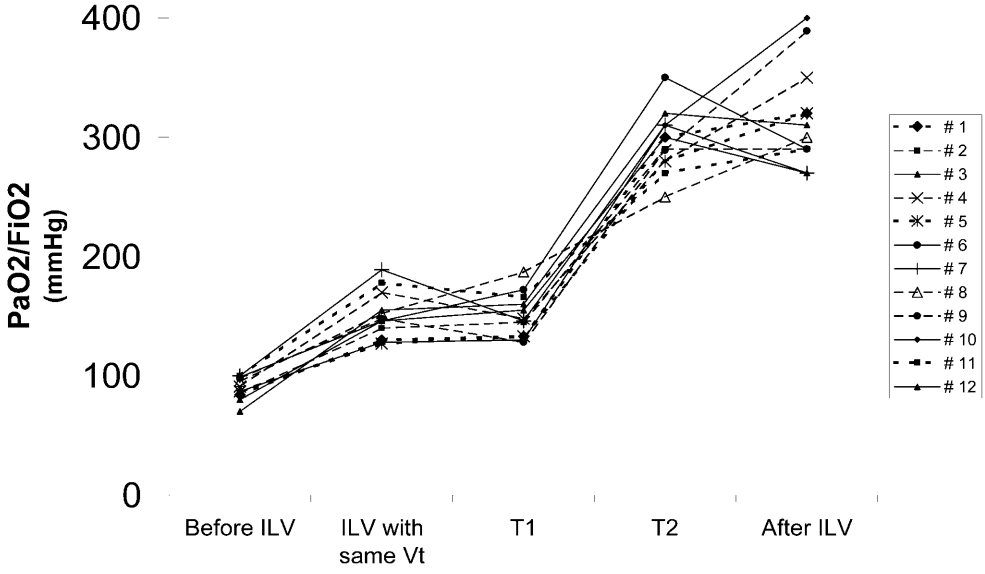

Fig. 1 The PaO2/FiO2 ratio of each patient before, during, and after ILV. (*Before ILV* 1 h before starting ILV, *ILV with same Vt* 30 min after starting ILV with same Vt in both lungs, *T1* 30 min after reducing Vt in DL, *T2* 30 min before stopping ILV, *after ILV* 1 h after reinstitution of intermittent positive-pressure ventilation)

Discussion

The main findings of the present study are: a) during ILV, adequate oxygenation can be obtained by setting ILV so as to keep Pplat below a safe threshold value for barotrauma; b) a significant difference in $EtCO_2$ exists between the two lungs, due mainly to lung inhomogeneity, and only slightly affected by the ventilatory setting. Actually, in our patients, $EtCO_2$ was significantly lower in the DL than in the NL, both with equal Vt and after lowering Vt on the DL (T_1); c) weaning from ILV was performed when there was no longer any difference in Vt between the two lungs (T_2); at the same time there was the equalization of $EtCO_2$.

Up to now, no clear guidelines have yet been established for initiating and qualifying the need for ILV: the spectrum of indications for which ILV has been applied is broad, [1, 4, 5, 7, 8, 9, 10, 12, 14]; however, in almost

Table 3 Hemodynamic data. (*Pra* right atrial pressure, *mPpa* mean pulmonary arterial pressure, *PAOP* pulmonary arterial occlusion pressure, *CI* cardiac index, *Qs/Qt* percent of mixed blood shunt, PvO_2 mixed venous oxygen pressure)

Time	Patients	Pra (mmHg)	mPpa (mmHg)	PAOP (mmHg)	CI (l·min·m^2)	Qs/Qt (%)	PvO_2 (mmHg)
Before ILV	1	6	17	8	3.5	28	28
	2	9	28	11	5.1	32	30
	3	6	18	10	3.8	26	31
	Mean ± SD	7 ± 1.7	21 ± 6.1	9.6 ± 1.5	4.1 ± 0.8	28.7 ± 3.1	29.6 ± 1.5
ILV same Vt	1	6	19	7	3.67	24	35
	2	10	27	12	5.1	27	32
	3	6	18	19	3.7	22	37
	Mean ± SD	7.3 ± 2.3	21.3 ± 4.9	9.3 ± 2.5	4.1 ± 0.8	24.3 ± 2.5	34.7 ± 2.5
T1	1	7	19	8	3.7	24	33
	2	10	27	12	4.8	20	29
	3	8	17	9	3.7	18	34
	Mean ± SD	8.3 ± 1.5	21 ± 5.3	9.7 ± 2.1	4.1 ± 0.6	20.7 ± 3.1	32 ± 2.6
T2	1	8	18	8	3.7	16	34
	2	13	25	11	4.8	15	32
	3	7	16	8	3.6	11	35
	Mean ± SD	9.3 ± 3.2	19.7 ± 4.7	9 ± 1.7	4.1 ± 0.7	14 ± 2.6	33.7 ± 1.7
After ILV	1	7	16	7	3.82	12	37
	2	11	23	11	4.7	14	35
	3	8	16	9	3.9	8	32
	Mean ± SD	8.6 ± 2.1	18.3 ± 4.1	9 ± 2	4.1 ± 0.5	11.3 ± 3.1	34.7 ± 2.5
Friedman's	test	NS	NS	NS	NS	[a]	NS

[a] Friedman's test: $P < 0.05$

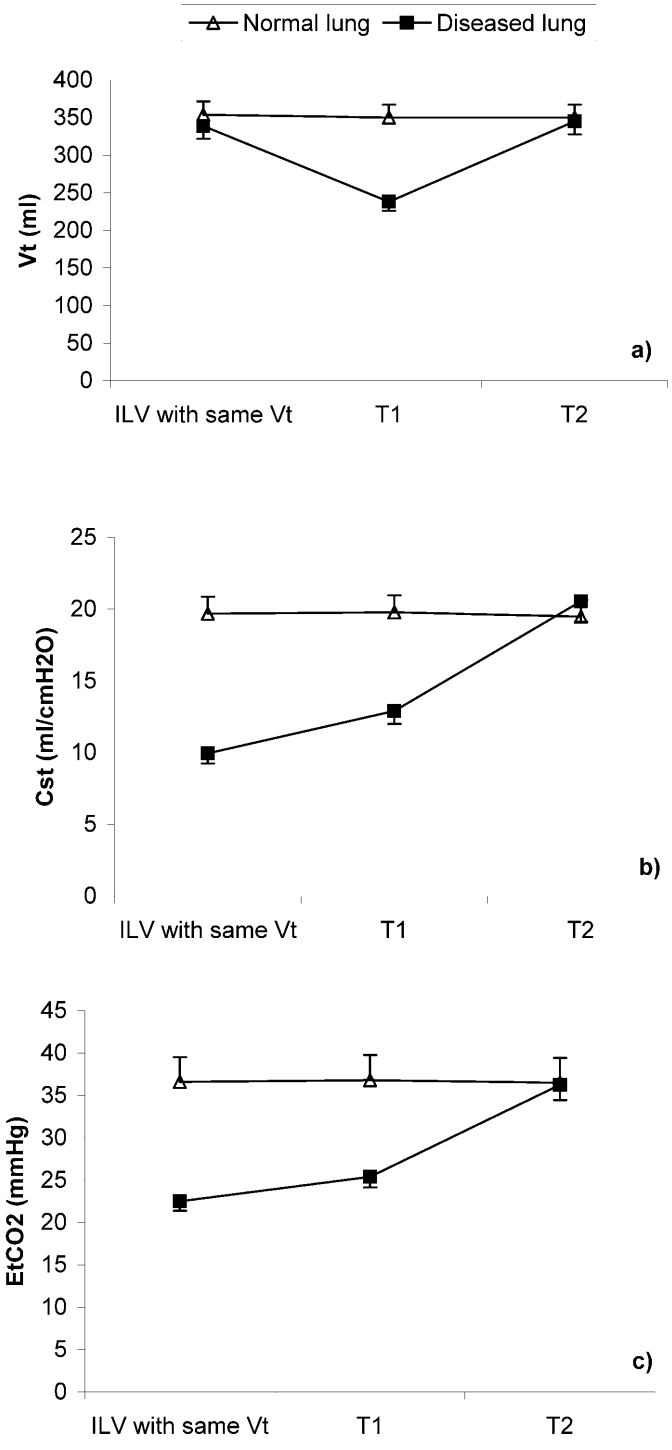

Fig. 2 Mean values ± SD of **a** tidal volume, **b** static compliance, and **c** end-tidal carbon dioxide, of normal *(open triangles)* and diseased *(closed squares)* lungs. (*ILV with same Vt* 30 min after starting ILV with same Vt in both lungs, *T1* 30 min after reducing Vt in DL, *T2* 30 min before stopping ILV)

50% of cases ILV is used in thoracic trauma patients with unilateral lung contusion [9]. The rationale for ILV is the presence of asymmetry in lung pathology that produces differences in compliance between the two lungs with the consequent diversion of Vt towards the less diseased, more compliant lung which is disproportionately distended by the applied PEEP. At the same time PEEP is transmitted to alveolar capillaries, acting as a Starling resistance, and reducing perfusion of the less diseased lung: the net effect of PEEP is the increasing of V/Q mismatching and the worsening of gas exchange [1, 4, 5, 6, 7, 9, 10]. Therefore, a firm criterion for ILV is the demonstration of a paradoxical PEEP effect, with a fall in PaO_2 and an increase in shunt due to redistribution of pulmonary blood flow [1, 3, 4, 5, 9, 10, 16]: this criterion was satisfied in all patients included in the present study.

To improve V/Q, it has also been suggested that the patients could be placed in a lateral position with the less diseased lung in the inferior position [5, 6, 14]. However, the lateral decubitus is not always possible in traumatized patients, or it cannot be maintained for long periods, because there can be serious problems with nursing care. Actually, ILV itself requires highly skilled nursing care and presents some technical difficulty mostly related to the double lumen tube positioning, risk of tube displacement or obstruction, and many techniques have been developed to check tube position [28, 29]. Moreover, each tube's lumen has an internal diameter as little as 5 mm, and lumen obstruction due to the deposit of viscous secretions is a serious, potentially life-threatening risk: adequate airways humidification and frequent bronchial suctioning are mandatory to reduce the incidence of such complication [30]. However, to our knowledge, no major complications related to ILV have been described in the ICU setting.

Rationale for setting a different Vt in each lung: inhomogeneity between the two lungs

Until a few years ago, the conventional ventilatory treatment for ARDS was large Vt with PEEP application to improve alveolar recruitment and arterial oxygenation [18, 19, 20, 21, 23, 31], and the same ventilatory strategy was applied to treat patients with unilateral lung disease. Henceforth, up to now, equal Vt in the two lungs and selective PEEP on DL has been the most widely used setting for ILV in such patients [1, 3, 9, 10, 12, 13, 14]: in an experimental study, East et al. [3], comparing different Vt delivering patterns found that the highest PaO_2/FiO_2 ratio was obtained when equal Vt and selective PEEP were used. However, this pattern led to levels of Pplat higher than 30 cmH2O in the DL [2, 3, 12, 13], as also reported in clinical studies [9, 12], because the DL is less compliant than the NL.

Several studies have questioned the ability of conventional ventilatory treatment to improve respiratory mechanics and gas exchange in ARDS patients [18, 19, 31], and pointed out its potential for harm [18, 21,23]: recent reports [18, 21, 31, 32, 33, 34, 35] showed that mechanical ventilation with high airway pressure and/or high Vt can cause diffuse alveolar damage. Although it is not clear whether a threshold exists below which lung distension does not aggravate previous lung alterations [18, 19], a level of Pplat ≤30cmH_2O, has been suggested as quite safe [20].

If the same ventilatory strategies are transposed from ARDS to the diseased lung in unilateral lung injury, the ventilatory setting in the diseased lung should be chosen in order to keep Pplat below the potentially dangerous threshold. In two recently published cases [22], we set Vt on each lung at a value generating a Pplat ≤26 cmH2O, in order to ventilate each lung with a Vt not resulting in overinflation. The two patients had a good and stable PaO_2/FiO_2 ratio and were weaned from ILV after 48 h and 84 h, respectively. Hence, this study protocol was launched to test our hypothesis and avoid high levels of Paw in the diseased lung. This goal was obtained by setting Vt in both lungs at Pplat < 26 cmH2O and led, on T_1, to a Vt significantly lower in the more diseased lung (Table 2). During the course of ILV, in the DL, Vt was progressively increased to keep Pplat stable. This ventilatory schema did not affect oxygenation: the PaO_2/FiO_2 ratio remained stable after Vt reduction in the DL (Table 2).

Rationale for monitoring the EtCO2 during ILV: inhomogeneity in the DL

In the previously mentioned paper [22], $EtCO_2$ was found to be lower in the DL when using the same Vt, as already demonstrated by experimental and clinical studies [3, 15, 16, 22]. Furthermore, a difference between normal and diseased lung $EtCO_2$ was still observed after the reduction of Vt in DL: these data were confirmed in the present study (Table 2). The difference in $EtCO_2$ value is explained by the inhomogeneity proper to lung injury, characterized by coexistence in the same lung of less diseased, more compliant areas and more diseased, less compliant areas presenting in a different ratio from patient to patient [5, 23, 36].

Pulmonary pathophysiology during ILV can thus be considered as a "dynamic condition" changing over time, and the rationale of ILV should be to keep adjusting the setting to the "best" ventilation for each lung. When ILV is started, in the contused lung more compliant areas are better ventilated or even overinflated, less compliant areas being underventilated. The V/Q ratio of the whole lung results from the coexisting variable ratio of high and low V/Q parenchymal areas. Since the less contused, overinflated areas are those responsible for gas exchange, this explains the finding of a lower $EtCO_2$ in the DL with the same Vt on both lungs, confirming data already reported in literature [3, 12, 16]. On T_1, the lowering of Vt, although producing mild hypercapnia (Table 2), reduced overinflation of the "normal areas". Hence, $EtCO_2$ of the DL increased, although still significantly different from the NL. Nevertheless, blood shunting due to contused lung areas was still present (Table 3). During the course of ILV, in the DL, together with improvement of the lung damage, the Pplat developed by a given Vt was reduced, while blood shunting was reduced and $EtCO_2$ increased (Fig. 2). In our patients, every time Pplat changed, the Vt given to the DL was increased step-wise until the Pplat was equal on both lungs (T_2). By this time, there was no longer any difference in $EtCO_2$ between the two lungs (Fig. 2).

Limitation of the study

This study has two methodological limitations: a) the evaluation of lung mechanics is not especially refined; and b) there is no control group of patients.

The evaluation of lung mechanics is the object of an enormous amount of medical literature, and many alternative methods for evaluating lung compliance have been validated. However, the authors decided to limit mechanical evaluation to the "classical" static compliance measurement, feeling that most of these methods are too complex and cannot be performed many times a day, as would be necessary in these patients for monitoring of the lung injury evolution, especially bearing in mind the complex, cumbersome equipment required for ILV.

The ventilatory schema used in this study should be compared to that most largely used in the literature, i.e., "the same Vt on both lungs". However, this ventilatory pattern, as discussed before, produces high Pplat, suggested to be detrimental in acute respiratory failure. Since our patients were ventilated with the same Vt on both lungs at the beginning of ILV, each patient was therefore his/her own control.

Conclusions

Although more investigations are required, our data seem to confirm that: a) ILV can be performed by setting individual lung Vt to a value not exceeding the safe Pplat threshold of 26 cmH2O, and that this ventilatory pattern improves oxygenation; b) together with the standard lung mechanics parameters, single lung $EtCO_2$ variations can be used as a guideline for monitoring the ventilation to perfusion matching of each lung.

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