Reports of Investigation

Haemodynamic changes induced by two I:E ratios: a transoesophageal echocardiographic study

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Purpose: To assess the effects of controlled ventilation with two I:E ratios on haemodynamic and left ventricular function in mechanically ventilated patients with moderate to severe respiratory disease, using fluctuation of the arterial pressure waveform and the changes in left ventricular areas obtained by transoesophageal echocardiography.

Methods: Nine patients had their lungs ventilated using volume controlled ventilation with two I:E ratios (1:3 and 1:1). Respiratory rate was adjusted so that six cardiac beats occurred during a respiratory cycle. Systolic blood pressure variation (SBPV), left ventricular area variations measured by TEE and haemodynamic variables measured by PA catheter were compared.

Results: When compared with I:E (1:3), I:E (1:1) decreased end diastolic area (EDA) throughout the respiratory cycle from 3% to 8% ($P < 0.01$) and increased SBPV from 6 ± 1 to 11 ± 1 mmHg ($P < 0.01$). In four patients, SBPV was > 12 mmHg with I:E 1:1. Conversely, SBPV was < 10 mmHg in all patients with I:E 1:3. With I:E (1:1), EDA decreased up to 7% during expiration ($P < 0.01$). The ejection fraction area remained stable for both ventilatory patterns and throughout the ventilatory cycle for a given I:E. The usual invasive haemodynamic variables were unchanged throughout the study, as was $\text{PaO}_2/\text{FiO}_2$.

Conclusion: In this setting, EDA and SBPV allow beat-to-beat evaluation of left ventricular preload during change of I:E ratio. Switch from I:E 1:3 to 1:1 may be used as a rapid, safe and reversible test to estimate intravascular volume status assessed by changes in SBPV or EDA.

Objectif : Évaluer les effets de la ventilation contrôlée à rapport I:E variable sur les paramètres hémodynamiques et la fonction ventriculaire gauche de patients ventilés pour insuffisance respiratoire modérée à sévère. Les deux principales variables étudiées étaient l'amplitude de la variation de la pression artérielle systolique (VPAS) et la modification de la surface télédiastolique ventriculaire gauche (STD) obtenue par échographie transoesophagienne (ETO).

Méthodes : Neuf patients ont été ventilés en volume contrôlé avec un I:E soit de 1:1 soit de 1:3. La fréquence respiratoire a été fixée à un cycle respiratoire pour six cycles cardiaques. La VPAS, la STD, et la fraction de raccourcissement de surface (FRS) obtenues par ETO ainsi que les autres variables hémodynamiques usuelles obtenues par cathéter pulmonaire ont été comparées.

Résultats : L'allongement du I:E entraîne une diminution de la STD de 3% à 8% durant le cycle respiratoire ($P < 0.01$) et une augmentation de la VPAS de 6 ± 1 à 11 ± 1 mmHg ($P < 0.01$). Pour un I:E de 1:1, quatre patients ont une VPAS > 12 mmHg. Avec un I:E de 1:3, tous les patients ont une VPAS < 10 mmHg. Avec un I:E de 1:1, la STD diminue de 7% pendant l'expiration comparée à l'inspiration ($P < 0.01$). La FRS ne varie ni au cours du cycle respiratoire ni lorsque que le I:E change. Les variables hémodynamiques invasives ainsi que le rapport $\text{PaO}_2/\text{FiO}_2$ demeurent inchangés tout au long de l'étude.

Conclusion : La STD et la VPAS permettent une évaluation précise de la précharge ventriculaire gauche induite par un allongement du I:E. L'augmentation du I:E de 1:3 à 1:1 peut être utilisée comme un test rapide, sûr et réversible pour estimer le niveau du remplissage ventriculaire apprécié par la STD ou la VPAS.

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MECHANICAL ventilation with intermittent or positive end expiratory pressure is used to restore arterial oxygenation and alveolar ventilation in severe respiratory failure. Left ventricular function is modified by intrathoracic pressure changes with either negative¹ or positive² pressure. Different I:E ratios have been evaluated with volume-controlled and pressure-controlled ventilation in patients with acute respiratory distress syndrome to reduce ventilation-related lung injury and to yield better oxygenation. The haemodynamic consequences of such ventilation is debatable, depending on the pathophysiology and characteristics of the patients. Cardiac output, measured by thermodilution, may decrease,^{3,4} remain stable^{5,6} or increase,⁷ and mean arterial pressure may be stable^{3,4} or decrease.^{5,8} These discrepancies are related to the complex cardiopulmonary interactions depending on many variables such as heart and lung compliance, level of PEEP, intrinsic PEEP, tidal volume and heart rate. This cardiopulmonary interaction leads to cyclical changes in the arterial pulse wave during respiratory support.⁹ The cyclical changes in arterial pulse during respiratory support have been reported to be a sensitive index of hypovolaemia during ventilatory support.^{10,11} Moreover, changes in ventricular size^{9,12} have been reported with mechanical ventilation.

The accurate estimation of preload is difficult in a given patient and invasive haemodynamic techniques are sometimes questionable.^{13,14} The use of left ventricular end-diastolic area (EDA) as a surrogate for left ventricular end-diastolic volume has been validated^{15,16} and provides an accurate estimation of preload.^{10,17} To test the intravascular volume status of the patient, rapid increase in preload may be obtained with a fluid challenge. Conversely, a rapid and safe decrease in preload is more difficult to achieve (blood-letting, vasodilator drugs). This study was designed to assess the effects of controlled ventilation with two I:E ratios on haemodynamic and ventricular function in patients with moderate to severe respiratory disease, using fluctuation of the arterial pressure waveform and the changes in left ventricular area obtained by transoesophageal echocardiography.

Methods

Patients

The study was approved by the local ethics committee, and informed written consent was obtained from the family of each patient. A prospective study was conducted to compare the effects of volume controlled ventilation with various inspiratory to expiratory times (I:E) on haemodynamic variables assessed either by right heart catheterisation or transoesophageal echocar-

diography (TEE) and gas exchange in nine patients with moderate to severe respiratory failure. The following criteria were accepted for inclusion: (1) arterial hypoxaemia with $\text{PaO}_2/\text{FIO}_2 < 300$ (hypoxaemia score 1 in the lung injury score),¹⁸ (2) bilateral diffuse infiltrates on the chest-X-ray, (3) volume controlled ventilation with PEEP $< 10 \text{ cmH}_2\text{O}$. Patients were excluded if they had haemodynamic instability, dysrhythmias or heart rate $> 120 \text{ bpm}$. Patients with intracardiac shunt, severe tricuspid insufficiency or left ventricular wall motion abnormalities, as observed with TEE were also excluded from the study, as were those exhibiting any contraindication to TEE.

All patients received sedation with 1 to $2 \mu\text{g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ fentanyl and 2 to $5 \text{ mg}\cdot\text{hr}^{-1}$ midazolam *iv*. Muscle relaxation was achieved with $0.1 \text{ mg}\cdot\text{kg}^{-1}$ pancuronium. Patients were studied in the recumbent position.

Ventilation protocol

The lungs were ventilated with a Siemens Servo 900C ventilator at a respiratory rate adjusted so that six cardiac beats occurred during a respiratory cycle, as previously described.¹² Tidal volume ($8\text{--}12 \text{ ml}\cdot\text{kg}^{-1}$ body weight), inspiratory flow and FIO_2 were kept constant throughout the study period. The patients were then randomized to receive volume controlled ventilation with I:E ratios of 1:3 or 1:1 without pause time, so that three cardiac beats occurred during inspiration with I:E 1:1 and less than two with I:E 1:3. There was a 30 min stabilization period before measurements. Peak and mean inspiratory airway pressure were measured. Arterial blood gases were sampled for measurements of PaO_2 and PaCO_2 .

Haemodynamic protocol

Each patient had a radial artery catheter and a pulmonary artery catheter in place. Heart rate, systemic arterial pressure, systolic blood pressure variation (SBPV) defined as the difference between the maximum and minimum values of systolic blood pressure following a single positive pressure breath, pulmonary artery pressure (PAP), right atrial pressure (RAP) and pulmonary capillary wedge pressure (PCWP) were recorded on a multi-channel recorder (Kone 565 A) and averaged over the ventilatory cycle. The zero references were levelled at mid-chest for all vascular pressures. Cardiac output was measured by the thermodilution technique in triplicate.

Transoesophageal Echocardiography protocol

Connected to an ultrasound system (UM7, Advanced Technology Laboratories, les Ulis France), a 5-MHZ probe was used to perform TEE. After comprehensive study of the heart from the oesophagus, the probe was

advanced in the stomach with the tip anteflexed. A transgastric short axis view of the left ventricle at mid-papillary muscle was performed. Echocardiographic data were recorded on videotape (Panasonic S-VHS 7330E) together with an ECG lead and a respiratory curve displayed on the videoscreen. Precise timing of each cardiac beat within the respiratory cycle was thus obtained. Echocardiographic recordings were reviewed in real time and stop-frame analysis of six successive cardiac beats occurring during an entire respiratory cycle was evaluated. The end diastolic area (EDA) was selected at the peak of the R-wave on the ECG and the end systolic area (ESA) was defined as the smallest left ventricular dimension during the last half of the T wave. The ejection fraction area (Efa) of each beat was calculated as follows:

$$\text{Efa} = (\text{EDA} - \text{ESA}) \cdot 100 / \text{EDA}.$$

The EDA was divided by the body surface area of the patient to obtain indexed EDA (EDAi). Three successive respiratory cycles were analyzed and averaged. Haemodynamic and echocardiographic measurements were obtained simultaneously.

Statistical analysis

Data were expressed as mean $\pm$ SEM. Continuous, non-normal data were analyzed with the Wilcoxon W test for paired data. A *P* of 0.05 was considered as the threshold of significance. A Friedman ANOVA test was used in each ventilatory mode throughout the ventilatory cycle.

Results

Nine patients with acute respiratory failure were studied. Primary pathological findings at the time of admission to the ICU are shown in Table I. The respiratory rate and the tidal volume remained identical for each patient throughout the study. Mean respiratory rate was 18 ± 2 bpm and mean tidal volume was 10.9 ± 0.2

ml·kg⁻¹. Peak airway pressure decreased from 30 ± 3 to 26 ± 2 cmH₂O (*P* < 0.015), and mean airway pressure increased from 10 ± 1 to 12 ± 1 cmH₂O (*P* < 0.015) when I:E ratio was increased from 1:3 to 1:1 respectively. The PaO₂/FIO₂ was not different between 1:3 I:E ratio (183 ± 22) and 1:1 I:E ratio (190 ± 23) and PaCO₂ remained unchanged (32 ± 2 mmHg).

A weak correlation was found between the decrease in EDAi and the SBPV observed for 1:3 and 1:1 I:E ratios (*r* = 0.59 and 0.42 respectively). When the I:E ratio was increased from 1:3 to 1:1, the SBPV increased in all patients. In four, the SBPV was >12 mmHg with the 1:1 I:E ratio, whereas in all patients, the SBPV did not exceed 10 mmHg with an I:E ratio of 1:3 (Table II). No difference was observed in haemodynamic variables between the two ventilatory patterns except for the SBPV which was increased with 1:1 I:E ratio (*P* < 0.005) (Table III).

Echocardiographic results are reported in Figures 1, 2 and 3. The first beat was chosen in the early inspiratory phase. Three beats occurred during the inspiratory phase when the I:E ratio was 1:1 and less than two when the I:E ratio was 1:3. Indexed EDA was reduced

TABLE II Systolic Blood Pressure Variation (SBPV) and indexed End Diastolic Area (EDAi) during I:E increase.

Patient	SBPV (mmHg) 1:3 I:E ratio	SBPV (mmHg) 1:1 I:E ratio	Decrease in EDAi* (%)
1	4	8	-1.8
2	10	14	-6.6
3	6	12	-5.7
4	0	10	-0.4
5	8	15	-11.8
6	4	6	-2.9
7	2	7	-9.2
8	10	13	-10.3
9	7	15	-4.0

*Mean decrease in indexed EDA throughout a ventilatory cycle between 1:3 and 1:1 I:E ratios.

TABLE I Patient demographic data

Patient	Sex (M/F)	Age (yr)	Diagnosis	SAPS	PEEP (cmH ₂ O)	PaO ₂ /F _I O ₂	Respiratory rate (bpm)	Outcome
1	M	65	Head trauma	19	0	269	18	Survived
2	F	62	Intra-abdominal sepsis	13	6	251	18	Survived
3	M	52	Pulmonary oedema	11	6	245	14	Survived
4	F	37	Inhalation pneumonia	9	8	180	20	Survived
5	M	72	Septicaemia	13	8	128	17	Died
6	M	73	Pneumonia	21	4	123	20	Died
7	F	75	Septic shock	25	5	174	17	Died
8	M	34	Pneumonia	8	9	83	16	Survived
9	F	58	Septic shock	10	7	136	19	Survived

SAPS = Simplified Acute Physiologic Score

TABLE III Haemodynamic variables

	I:E = 1:3	I:E = 1:1	P
Heart rate (bpm)	103 ± 4	105 ± 5	NS
MAP (mmHg)	83 ± 3	79 ± 2	NS
MPAP (mmHg)	26 ± 1	24 ± 1	NS
RAP (mmHg)	11 ± 0.5	11 ± 0.5	NS
PCWP (mmHg)	13 ± 2	12 ± 1	NS
CI (L·min ⁻¹ ·m ⁻²)	3.8 ± 0.4	3.6 ± 0.4	NS
LVSWI (g·m ⁻¹ ·m ⁻¹) ²	31 ± 5	35 ± 6	NS
SVI (ml·m ⁻²)	37 ± 4	34 ± 3	NS
SBPV (mmHg)	6 ± 1	11 ± 1	0.005

(MAP = mean arterial pressure, MPAP = mean pulmonary arterial pressure, RAP = right atrial pressure, PCWP = pulmonary capillary wedge pressure, CI = cardiac index, LVSWI = left ventricular stroke work index, SVI = stroke volume index, SBPV = systolic blood pressure variation.)

throughout the respiratory cycle when the I:E ratio was increased. The smallest decrease in EDA was observed for the third beat (3%), and the greatest for the sixth beat (8.3%). With the 1:3 pattern, the indexed EDA did not change throughout the ventilatory cycle. Conversely, with the 1:1 pattern, the indexed EDA decreased (by up to 7%) during the expiratory phase compared with the inspiratory phase ($P < 0.01$). The EFa was not different between the two I:E settings or for a given I:E ratio throughout the ventilatory cycle.

Discussion

The main findings of this study were that: 1. changes occurred in EDA and SBPV when the I:E ratio is increased; 2. with 1:1 I:E ratio only, changes in EDA occurred between the inspiratory and the expiratory phase; 3. the EFa remained unchanged whatever the respiratory phase for a given I:E and between the two I:E ratios; 4. invasive haemodynamic variables were unable to detect such changes.

The haemodynamic consequences of mechanical ventilation have been extensively studied. The major effect of respiratory support is a reduction of the pressure gradient for systemic venous return, which reduces right ventricular preload and, as a result, left ventricular preload.^{2,9} Moreover, right ventricular afterload is increased.¹² In the present study, we used EDA as a reference index for the left ventricular preload, because a correlation has been observed between EDA and preload assessed by left ventricular end diastolic volume.^{15,16,19} We observed a decrease in EDA throughout the ventilatory cycle when the I:E ratio was increased from 1:3 to 1:1. This means that left ventricular preload was decreased throughout the ventilatory cycle when only the inspiratory phase was increased. The decrease was greatest at the end expi-

FIGURE 1 Change in indexed end-diastolic area throughout the ventilatory cycle at two ventilatory patterns. Solid bars I:E = 1:3; hatched bars I:E = 1:1. Mean ± SEM (* $P < 0.05$, ** $P < 0.01$)

FIGURE 2 Change in indexed end-systolic area throughout the ventilatory cycle in two ventilatory patterns. Solid bars I:E = 1:3; hatched bars I:E = 1:1. Mean ± SEM (* $P < 0.05$)

FIGURE 3 Change in Ejection Fraction area throughout the ventilatory cycle in two ventilatory patterns. Solid bars I:E = 1:3; hatched bars I:E = 1:1. Mean ± SEM.

ratory phase where it reached 8.3%. In this study, the change was not sufficient to be detected by usual haemodynamic monitoring such as mean arterial pressure, cardiac output and mean capillary wedge pressure, but the small sample studied decreased the power of the study. Our results regarding usual haemodynamic monitoring support those of Mercat *et al.*,⁴ Lessard *et al.*⁵ and Mang *et al.*⁶ who found no differences among most of the haemodynamic, cardiac output or capillary wedge pressure data between study modes. Nevertheless, a discrepancy was observed in another study, where cardiac output decreased when the I:E ratio was increased.³ In that study, the increase in mean airway pressure when the I:E ratio was increased was greater than in other studies.⁴⁻⁶ This may explain why in the study by Chan *et al.*³ the haemodynamic consequences of mechanical ventilation were clinically relevant.

For a given I:E ratio, EDA variations throughout the ventilatory cycle are different. With an I:E ratio of 1:3, indexed EDA did not change, i.e., no alteration occurred in left ventricular preload during this setting. Conversely, increasing the I:E ratio altered left ventricular filling during the expiratory phase because of changes occurring, during the inspiratory phase, on the right side of the heart. First, venous return to right atrium decreases during mechanical insufflation. In our study, with an I:E ratio of 1:1 and six cardiac beats per ventilatory cycle, venous return was reduced during three beats, compared with an I:E ratio of 1:3 where venous return was reduced during less than two beats. Second, during insufflation, right ventricular afterload increases.¹² These two effects converged to decrease EDA by as much as 7% in the second part (exsufflation) of the respiratory cycle.

The magnitude of the SBPV depends on cardiopulmonary interaction. The fluctuation of the arterial pressure waveform and its down component (defined as the difference between the end expiratory systolic blood pressure and the minimal blood pressure) is a result of intermittent positive pressure ventilation and is enhanced by hypovolaemia.^{10,11} Perel *et al.*¹¹ showed that SBPV was increased in ventilated dogs subjected to graded haemorrhage. In postoperative patients with good left ventricular function, Coriat *et al.*¹⁰ showed that rapid infusion of albumin decreased SBPV, the only haemodynamic variable that correlated with the initial EDA and predicted the response of EDA to volume loading. Moreover, they found that a SBPV >12 mmHg was associated with inadequate intravascular volume. In our study, no haemorrhage nor rapid infusion was performed, so that the SBPV increase can only be due to a change in the I:E ratio; the 1:1 I:E ratio impeding

venous return longer than the 1:3 I:E ratio. An increase in I:E ratio to 1:1 may display undetected hypovolaemia in patients with SBPV >12 mmHg. A rapid switch between I:E 1:3 to 1:1 may, therefore, be used as an easy, reversible method of assessing volume status and/or the haemodynamic tolerance to mechanical ventilation when SBPV exceed 12 mmHg. For example, at the bedside or in the operating room, one can give a prolonged positive pressure inspiration and examine aortic pressure waveform – first increasing (up), then decreasing (down) with regard to the reference pressure (obtained during five second apnea).¹¹ The magnitude of the SBPV may be used as an indication of the volume within the pulmonary capacitor, i.e., related to hyper – vs hypovolaemic total volume status, as an index of hypovolaemia.

In our study, changes in the I:E ratio did not influence EFa. A similar result has been reported for right ejection fraction,²⁰ while left ejection fraction increased slightly during continuous positive pressure ventilation and inverse ratio ventilation compared with intermittent positive pressure ventilation.⁸ These results suggest that, in the setting of our study, a change in preload induced by a change in ventilatory pattern did not influence the ejection fraction. Similarly, EFa remained stable in both the inspiratory and expiratory phases. These results confirm that, although the ejection fraction is preload dependent, it is relatively constant over a wide range of preload conditions.²¹

Invasive haemodynamic monitoring was unable to detect differences between the two ventilatory patterns, even though SBPV changed. This confirms that SBPV and echocardiographic measurements are more sensitive indices of changes in left ventricular function induced by mechanical ventilation than is conventional invasive haemodynamic monitoring.¹⁴

We did not observe any improvement in $\text{PaO}_2/\text{FIO}_2$ with increasing I:E ratio. However, $\text{PaO}_2/\text{FIO}_2$ may have been expected to improve when I:E ratio is increased because of recruitment of alveolar units with slow time constants secondary to the prolonged inspiratory time²² and the increase of mean airway pressure, which should reflect mean alveolar pressure, a major determinant of arterial oxygenation.²³ The absence of any improvement in our study may be related to the slight increase in mean airway pressure that was insufficient to improve oxygenation. Similar results have been observed previously.⁴⁻⁶

We conclude that EDA and SBPV allow precise beat-to-beat evaluation of left ventricular function during change of I:E ratio. An increase in I:E ratio from 1:3 to 1:1 impedes venous return and decreases left ventricu-

lar preload as assessed by EDA and SBPV. Switching the I:E ratio from 1:3 to 1:1 may be used as a rapid, safe and reversible test to estimate intravascular volume status assessed by changes in SBPV or EDA. Conversely, EFa remains unchanged between the two I:E ratios and throughout the ventilatory cycle.

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