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# Accurate measurement of intrinsic positive end-expiratory pressure: how to detect and correct for expiratory muscle activity

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*Accurate measurement of intrinsic positive end-expiratory pressure: how to detect and correct for expiratory muscle activity. S.G. Zakynthinos, T. Vassilakopoulos, E. Zakynthinos, C. Roussos. ©ERS Journals Ltd 1997.*

**ABSTRACT: It has been shown that expiratory muscle contraction leads to an overestimation of intrinsic positive end-expiratory pressure ($PEEP_i$). To quantify this overestimation, we compared $PEEP_i$, measured during spontaneous breathing (SB) by the end-expiratory airway occlusion technique ($PEEP_{i,occl}$) with static $PEEP_i$ ($PEEP_{i,st}$). $PEEP_{i,st}$ was measured using end-expiratory airway occlusion during simulation of SB by the ventilator with the patient relaxed, and was considered to represent the "gold standard" for $PEEP_{i,occl}$.**

**Twelve ventilator-dependent patients were studied during SB (pressure support 5–7 $cmH_2O$). Full mechanical ventilation was resumed when they were unable to sustain SB. Subsequently, by manipulating the variables of the ventilator, we simulated the pattern of SB and measured $PEEP_{i,st}$, corresponding to $PEEP_{i,occl}$. On the basis of the presence or absence of expiratory rise in gastric pressure ($P_{ga}$) (rapid drop of end-expiratory $P_{ga}$ at the beginning of inspiration, $P_{ga,exp,rise}$), and abdominal muscle electromyographic (EMG) activity, patients were subdivided into those either actively (Group 1) or passively expiring (Group 2).**

**In Group 1 (8 patients), $PEEP_{i,occl}$ was higher than $PEEP_{i,st}$ (13.3±2.0 *vs* 6.8±1.1 $cmH_2O$; $p<0.01$). $PEEP_{i,occl}$ - $P_{ga,exp,rise}$ (6.9±1.1 $cmH_2O$) was quite similar to $PEEP_{i,st}$; their mean difference was 0.03 $cmH_2O$ with limits of agreement -0.48 to +0.53 $cmH_2O$. In Group 2, $PEEP_{i,occl}$ was similar to $PEEP_{i,st}$.**

**We conclude that, in actively expiring patients, an accurate estimation of the actual $PEEP_{i,st}$ can be obtained by subtracting $P_{ga,exp,rise}$ from $PEEP_{i,occl}$.**

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In actively breathing patients, either during spontaneous or assisted ventilation, intrinsic positive end-expiratory pressure ($PEEP_i$) can be measured by two methods. In the first method, the airway occlusion method, $PEEP_i$ is measured as the plateau in airway pressure between occluded inspiratory efforts and expresses the elastic recoil pressure of the total respiratory system ($PEEP_{i,occl}$) [1]. In the second method, requiring the placement of an oesophageal balloon, the decrease in oesophageal pressure needed to abruptly bring expiratory flow to the point of zero during unoccluded breathing represents the dynamic $PEEP_i$ ($PEEP_{i,dyn}$) [1]. $PEEP_i$ measurements obtained by these methods have been considered to indicate the presence of dynamic hyperinflation and have been used, especially the second method, for its indiscriminate quantification. Recently, however, it has been shown that expiratory muscle activity during expiration increases the end-expiratory alveolar pressure independently of dynamic hyperinflation, leading to an overestimation of $PEEP_i$. Indeed, during airway occlusion, spontaneously breathing patients, using their expiratory muscles, react by increasing the end-expiratory airway pressure between inspiratory efforts, thus preventing the appearance of a plateau to measure $PEEP_i$ [2, 3]. In this instance, even if an apparent plateau in airway pressure is present, $PEEP_i$ is overestimated [4]. During unoccluded breathing, in turn, either in patients with stable chronic obstructive pulmonary disease (COPD) [2] or in mechanically-ventilated patients [3, 4], expiratory muscles can contract during expiration so that part of the decrease in oesophageal pressure preceding inspiration measured as $PEEP_{i,dyn}$ is due to relaxation of the expiratory muscles rather than to contraction of the inspiratory muscles to counterbalance $PEEP_i$.

The amount of $PEEP_i$ overestimation due to the use of expiratory muscles that should be subtracted from measured $PEEP_i$ to calculate the actual ("true") $PEEP_i$ produced by dynamic hyperinflation, remains unclear. Thus, the purpose of this study was to quantify the influence of expiratory muscle activity on $PEEP_i$ measurement in spontaneously breathing patients. Towards this goal, we measured the static $PEEP_i$ ($PEEP_{i,st}$) caused by dynamic hyperinflation and compared this value with $PEEP_{i,occl}$ measured in actively expiring patients. We used a method described recently [5] to measure $PEEP_i$ during spontaneous breathing under real static conditions, *i.e.* by simulating the exact pattern of spontaneous breathing with the ventilator during controlled mechanical ventilation and occluding the airway at the end of a tidal expiration [6]. $PEEP_{i,st}$ values obtained in this way were considered to represent the "gold standard" for $PEEP_{i,occl}$. The study was performed on patients with

acute respiratory failure, spontaneously breathing after discontinuation from mechanical ventilation and, subsequently, needing resumption of mechanical assistance. Since this condition may be considered as a good model of acute respiratory failure with the patients being in severe respiratory distress, we expected that at least some of them would actively expire during the spontaneous breathing trial. In addition to the actively expiring patients, the passively expiring patients were also studied to compare the results and confirm the accuracy of $PEEP_{i,occl}$ measurement in this latter condition.

## Methods

### *Patients*

Twelve patients (7 males, and 5 females) admitted to the intensive care unit for management of acute respiratory failure of different aetiologies (table 1), participated in the study. Diagnosis in four patients with COPD was based upon previous clinical history and routine lung function tests. Three patients with adult respiratory distress syndrome (ARDS) met the conventional criteria for the diagnosis of the syndrome; their lung injury scores on the day of the study were 1.8, 2.0 and 2.1. Four patients had severe sepsis according to the currently accepted criteria for the diagnosis of septic syndrome; three of them had positive blood cultures associated with a localized site of infection. The investigative protocol was approved by the institutional Ethics Committee and informed consent was obtained from the family of each patient.

All patients were intubated with a Portex cuffed endotracheal tube (Portex Ltd, Kent, UK), 8–9 mm inner diameter, cut to a length of 25 cm. The patients had been mechanically-ventilated for a period of 4–10 days prior to the present investigation and were clinically stable (systolic blood pressure >100 mmHg; heart rate <120 beats·min$^{-1}$; and with no significant fluctuations in blood pressure, heart rate, arterial blood gas values, urine output, or mental status) for the preceding 12 h. All sedative and paralysing medication was discontinued at least 12 h prior to the study. Other drug and fluid therapy continued throughout the study. All patients were studied early in the course of acute respiratory failure, at a time when discontinuation from full ventilatory assistance was felt to be impossible. Mechanical ventilation was delivered by a Siemens 300 servo ventilator (Siemens-Elema, Solna, Sweden) in the assist-control (A/C) mode, with the ventilator settings prescribed by the primary physicians. Tidal volume ($V_T$) was set on the ventilator at 0.47–0.78 L and respiratory frequency ($f_R$) ranged 16–28 breaths·min$^{-1}$. The time of mechanical inflation ($t_I$) ranged 0.49–0.95 s, whereas the duration of expiration ($t_E$) ranged 1.30–2.35 s. Mean inspiratory flow ($V_T/t_I$) ranged 0.69–1.20 L·s$^{-1}$, and PEEP was 0–6 $cmH_2O$. Thirty minutes before the beginning of the study, PEEP was removed and fraction of inspired oxygen ($F_{I,O_2}$) increased to 100% in all patients. Baseline blood gas values were obtained (ABL 300) 25 min later. Arterial oxygen tension ($P_{a,O_2}$) (mean±SD) was 20.1±6.4 kPa

Table 1. – Patient clinical characteristics, pertinent respiratory mechanics, and behaviour during spontaneous breathing after discontinuation from mechanical ventilation

| Pt No. | Sex | Age yrs | Diagnosis | $C_{st,rs}$* L·cm$H_2O^{-1}$ | $R_{rs,max}$* cm$H_2O$·L$^{-1}$·s$^{-1}$ | $R_{rs,min}$* cm$H_2O$·L$^{-1}$·s$^{-1}$ | Duration of discontinuation min | $P_{a,CO_2}$ increase+ mmHg | Expiratory muscle activity‡ |
|---|---|---|---|---|---|---|---|---|---|
| **Group 1** | | | | | | | | | |
| 1 | F | 54 | COPD | 0.068 | 29.5 | 21.7 | 30 | 37 | + |
| 2 | F | 60 | COPD | 0.066 | 31.1 | 22.4 | 35 | 30 | + |
| 3 | M | 56 | COPD | 0.071 | 17.5 | 11.2 | 50 | 28 | + |
| 4 | M | 64 | COPD | 0.060 | 15.3 | 10.8 | 42 | 20 | + |
| 5 | M | 59 | ARDS, flail chest | 0.036 | 13.5 | 8.1 | 15 | 15 | + |
| 6 | F | 42 | ARDS | 0.030 | 12.1 | 7.8 | 20 | 16 | + |
| 7 | M | 70 | ARDS | 0.026 | 13.0 | 6.5 | 26 | 14 | + |
| 8 | M | 70 | Coronary bypass, CPE | 0.048 | 14.7 | 10.6 | 29 | 41 | + |
| **Group 2** | | | | | | | | | |
| 9 | F | 76 | Sepsis | 0.070 | 12.0 | 9.1 | 60 | 29 | - |
| 10 | F | 75 | Atelectasis, sepsis | 0.059 | 13.1 | 8.4 | 23 | 30 | - |
| 11 | M | 50 | Sepsis | 0.065 | 12.4 | 9.2 | 30 | 32 | - |
| 12 | M | 48 | Pneumonia, sepsis | 0.049 | 14.5 | 8.7 | 34 | 18 | - |

Pt: patient; M: male; F: male; COPD: exacerbated chronic obstructive pulmonary disease; ARDS: adult respiratory distress syndrome; CPE: cardiogenic pulmonary oedema; $C_{st,rs}$: static compliance of the total respiratory system; $R_{rs,max}$ and $R_{rs,min}$: total and minimal (airway) respiratory system resistance (after subtraction of the endotracheal tube resistance [7]). $P_{a,CO_2}$: arterial carbon dioxide tension (1 mmHg=0.133 kPa). *: assessed during control mechanical ventilation with the patient sedated and/or paralysed, using the constant flow end-inspiratory occlusion method [7]; +: increase in $P_{a,CO_2}$ at the end of spontaneous breathing trial, compared to its value at the beginning of this trial; ‡: Based on the presence or absence of expiratory muscle activity during expiration, patients were divided into two groups. +: denotes the presence of expiratory muscle activity at expiration during spontaneous breathing; -: denotes the absence of expiratory muscle activity.

(151±48 mmHg), and arterial carbon dioxide tension ($P_{a,CO_2}$) was 6.3±1.2 kPa (47±9 mmHg). During the study, a physician not involved in the procedure was always present to provide for patient care as needed. The electrocardiogram, the heart rate, the systemic arterial blood pressure and the arterial oxygen saturation ($S_{a,O_2}$) (Nellcor, CA, USA) were monitored continuously.

*Experimental set-up*

All measurements were made with the patients in a semirecumbent position. Airflow ($V'$) was measured with a heated pneumotachograph (Fleisch No. 2; Lausanne, Switzerland) inserted between the endotracheal tube and the Y-piece of the ventilator, and a differential pressure transducer (Validyne MP-45, ±2 $cmH_2O$; Northridge, CA, USA). $V_T$ was obtained by integrating the flow signal. Oesophageal ($P_{oes}$) and gastric ($P_{ga}$) pressures were measured with conventional balloon-catheter systems placed in the midoesophagus and the stomach, respectively. The oesophageal balloon was filled with 0.5 mL of air, and the gastric balloon contained 1.0 mL of air. Both balloons were connected to separate differential pressure transducers (Validyne MP-45, ±100 $cmH_2O$). Appropriate placement of the oesophageal balloon was verified by an occlusion test [8]. Airway pressure ($P_{aw}$) was recorded at the distal end of the endotracheal tube with a differential pressure transducer (Validyne MP-45, ±100 $cmH_2O$). Rib cage (RC) and abdominal (AB) displacements were measured with a respiratory inductive plethysmograph (Respitrace Ambulatory Monitoring, Ardsley, NY, USA). The bands were placed circumferentially around the RC and AB, in such a way that they were at the level of the nipples and umbilicus, respectively. Care was taken to avoid overlap of the AB band with the lower RC. The electrical activity of the abdominal muscles (EMGab) was recorded with surface electrodes placed in the right anterior axillary line, midway between the costal margin and the iliac crest, and conditioned with a Nihon-Kohden electromyograph amplifier (band-pass between 20 Hz and 1 kHz). All signals, except EMGab, were continuously recorded on an eight-channel electrostatic recorder (Gould ES 1000; Gould Instruments, Cleveland, OH, USA) at a paper speed of 10 or 25 $mm \cdot s^{-1}$, and taped on a videorecorder (including EMGab) *via* an analogue-to-digital converter. The data were played back to a personal computer (Wyse 486) by the same analogue-to-digital converter, at a sampling rate of 1,500 Hz (EMGab) or 200 Hz (other variables), for subsequent data analysis.

*Protocol*

Patients were initially allowed to breathe spontaneously through the ventilator with a small inspiratory assistance (pressure support 5–7 $cmH_2O$) to compensate for the additional work due to the endotracheal tube and inspiratory circuit [9]. Fifteen to sixty minutes (33±13 min) after the beginning of spontaneous breathing, $P_{a,CO_2}$ increased ≥1.9 kPa (14 mmHg) in all patients (3.5±1.2 kPa) (26±9 mmHg)) (table 1), and reinstitution of mechanical ventilation on A/C was required. In every patient, $S_{a,O_2}$ remained higher than 90% throughout the spontaneous breathing trial. $P_{a,O_2}$ was 17.2±7.3 kPa (129±55 mmHg) at the end of this trial. During spontaneous breathing, the airway was occluded with the end-expiratory hold knob of the ventilator. Each occlusion lasted 8–12 s, and was repeated 2–3 times in every patient. As soon as mechanical ventilation on the A/C mode had been reinstituted, the patients were sedated (midazolam) and some were also paralysed (pancuronium bromide). After an initial bolus dose of these drugs (15 mg midazolam, 0.06 $mg \cdot kg^{-1}$ pancuronium bromide) had been given, the patients continued to receive the same sedative and paralysing medication in order to maintain undetectable respiratory muscle activity (judged by the lack of inspiratory swings of $P_{oes}$, airway pressure wave contour representative of passive inflation and clinical assessment).

Recordings of $V'$ and volume during spontaneous breathing were analysed in terms of $V_T$, $f_R$ and duty cycle ($t_I/t_{tot}$) to obtain the breathing pattern. The period of time selected for analysis in every patient was just before the initiation of an airway occlusion (randomly selected among the 2–3 performed in each patient). The value obtained was the mean of three consecutive breaths. With the patient ventilated with control mechanical ventilation and constant inspiratory flow, we subsequently tried to simulate this pattern of spontaneous breathing in each patient by regulating the appropriate buttons of the ventilator [5]. Inclusion criteria for accepting simulated breaths as representative of the breathing pattern during spontaneous ventilation were: 1) $V_T$ ±0.02 L; 2) $f_R$ ±0.1 breaths; and 3) $t_I/t_{tot}$ ±0.02. For the patients as a group, the breathing pattern before the initiation of the airway occlusion was (mean±SD): $V_T$ 0.42±0.06 L; $f_R$ 28±6 $breaths \cdot min^{-1}$; and $t_I/t_{tot}$ 0.33± 0.03. Breaths simulated by the ventilator were almost identical, *i.e*: $V_T$ 0.42±0.07 L; $f_R$ 28±6 $breaths \cdot min^{-1}$; and $t_I/t_{tot}$ 0.33± 0.02.

*Measurements and data analysis*

During spontaneous breathing, expiratory muscle activity was assessed as follows. Firstly, the rise of $P_{ga}$ was measured during the expiration from its end-inspiratory level to the maximum at end-expiration, and its subsequent abrupt decrease at the beginning of next inspiration ($P_{ga,exp,rise}$) (fig. 1). This pattern, associated with a decrease in abdominal cross-sectional area during the expiration and a subsequent increase during the inspiration, gives characteristic $P_{ga}$-AB displacement loops [2, 3] and clearly indicates expiratory contraction of the abdominal muscles. The increase in $P_{ga}$ occurring during the expiratory phase of the breathing cycle can be considered as a reflection of the direct mechanical effect of this contraction [2]. Secondly, the increase in end-expiratory $P_{oes}$ ($\Delta P_{oes}$) was measured, relative to its level at the onset of spontaneous breathing trial, when expiratory muscle activity was nil (fig. 1). This increase in end-expiratory oesophageal pressure indicates recruitment of the expiratory muscles during expiration which, at least in normal subjects, leads to an end-expiratory lung volume below the subject's normal functional residual capacity (FRC) [10]. Thirdly, the EMG activity of the abdominal muscle (EMGab) was measured. Fourthly, palpation of the abdominal wall was performed.

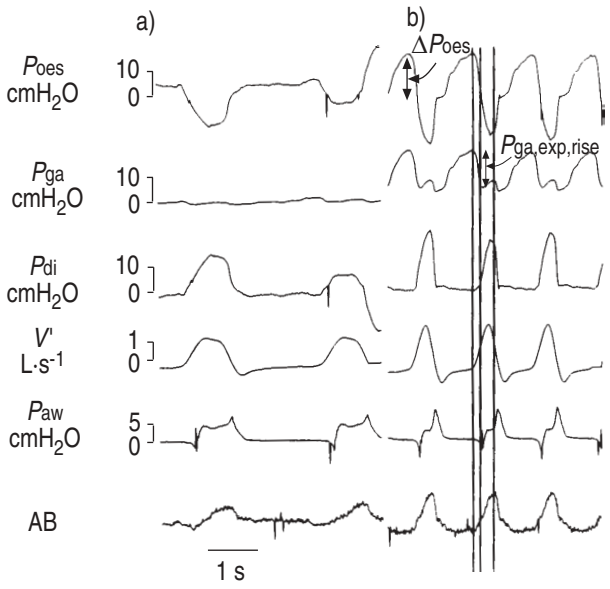


Fig. 1. – Tracings of oesophageal pressure ($P_{oes}$), gastric pressure ($P_{ga}$), transdiaphragmatic pressure ($P_{di}$), flow ($V'$), airway pressure ($P_{aw}$), and cross-sectional area of the abdomen (AB) in a representative patient (patient No. 1) actively expiring during spontaneous breathing through the ventilator (pressure support 6 $cmH_2O$). After discontinuation from full mechanical assistance, the patient was unable to sustain spontaneous breathing and resumption of mechanical ventilation was needed. a) corresponds to the beginning of spontaneous breathing (SB). b) corresponds to the end of spontaneous breathing, 1 min before the reinstitution of control mechanical ventilation. The three vertical lines are passed through the onset of inspiratory muscle activity (*i.e.* beginning of $P_{oes}$ decay) and the beginning and the end of inspiratory flow, respectively. In contrast to the beginning of spontaneous breathing (a), note the large increase of $P_{ga}$ and $P_{oes}$ during expiration due to expiratory muscle recruitment at the end of spontaneous breathing trial (b). For further explanation, see text.

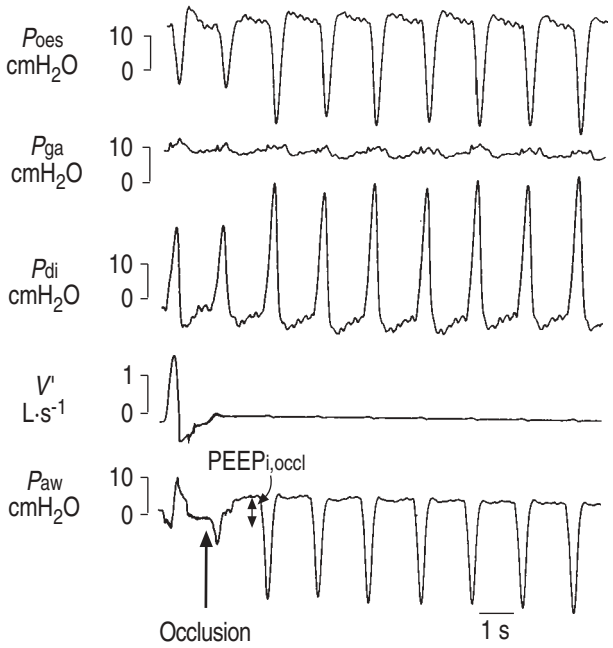


Fig. 2. – Recordings of oesophageal pressure ($P_{oes}$), gastric pressure ($P_{ga}$), transdiaphragmatic pressure ($P_{di}$), flow ($V'$) and airway pressure ($P_{aw}$) during end-expiratory airway occlusion in a representative patient (patient No. 9) passively expiring during spontaneous breathing through the ventilator (pressure support 5 $cmH_2O$). Airway occlusion was performed with the expiratory hold knob of the Siemens 300 ventilator. An apparent end-expiratory plateau in $P_{aw}$ is present between occluded inspiratory efforts representing the elastic recoil of the total respiratory system. Note that expiration remains passive in all consecutive inspiratory efforts post-occlusion.

PEEPi during spontaneous breathing was measured by two methods described previously [1, 5]. In the first method, the airway was occluded at end-expiration using the end-expiratory hold button of the ventilator, and the occlusion was maintained for several consecutive inspiratory efforts. The value of end-expiratory plateau in $P_{aw}$ between occluded inspiratory efforts represents the elastic recoil pressure of the total respiratory system [1], and is called "$PEEP_{i,occl}$" in the present study. An expiratory plateau in $P_{aw}$ postocclusion was apparent in all patients who did not use their expiratory muscles during spontaneous breathing (fig. 2). In contrast, this was not the case in the actively expiring patients (fig. 3). For the sake of consistency, in these patients the value of $P_{aw}$ at the end of expiration of the first postocclusion inspiratory effort was also referred to here as "$PEEP_{i,occl}$". However, it is important to emphasize that, in this circumstance, the term $PEEP_{i,occl}$ was only used to indicate the presence of a positive alveolar pressure at end-expiration produced through whatever mechanism. This pressure does not reflect the elastic recoil of the total respiratory system, since expiratory muscle contraction obviously contributes to its value. The second method was performed under static conditions [5]. With the patient sedated and sometimes paralysed during the simulation of spontaneous breathing by the ventilator, the airway was occluded at the end of a tidal expiration using the end-expiratory hold button of the ventilator (fig. 3). The end-expiratory plateau of $P_{aw}$ directly reflects intrinsic PEEP ($PEEP_{i,st}$) [6]. With the ventilator, we simulated one pattern of spontaneous breathing in each patient, *i.e.* that corresponding to the period just prior to an airway occlusion.

When the patient was ventilated with controlled mechanical ventilation during the simulation of spontaneous breathing, respiratory mechanics (table 1) were assessed by the constant flow end-inspiratory occlusion method described in detail previously [7].

Results are expressed as mean±SE, unless otherwise specified. Statistical analysis was performed using Student's paired t-test and linear regression analysis. A p-value of 0.05 was considered significant. The agreement between $PEEP_{i,occl}$ (corrected for expiratory muscle activity) and $PEEP_{i,st}$ which measure the same physiological variable, *i.e.* the static PEEPi, was evaluated by the method of BLAND and ALTMAN [11]. The degree of agreement was summarized by calculating the mean difference and the standard deviation of the differences (SD). If differences within the limits of agreement, *i.e.* mean difference ±2SD, are not clinically important, the two methods can be used interchangeably.

## Results

Patients were divided in two groups on the basis of expiratory muscle activity during the spontaneous breathing trial (table 1). Group 1 consisted of patients who were using their expiratory muscles at expiration (mostly COPD and ARDS). In every Group 1 patient, active expiration was detected by the coexistence of a typical

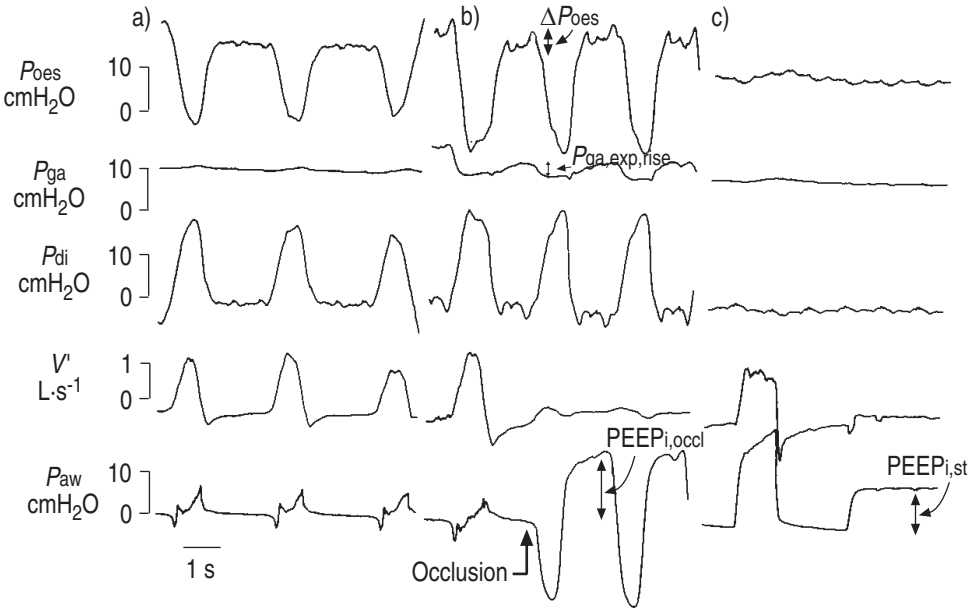


Fig. 3. – Representative recordings of oesophageal pressure ($P_{oes}$), gastric pressure ($P_{ga}$), transdiaphragmatic pressure ($P_{di}$), flow ($V'$) and airway pressure ($P_{aw}$) during airway occlusion in a patient (patient No. 3) actively expiring during spontaneous breathing (SB) through the ventilator (pressure support 5 $cmH_2O$). a) corresponds to the beginning of spontaneous breathing, after discontinuation from full mechanical ventilation. b) Thirty minutes later the airway was occluded with the end-expiratory hold knob of the ventilator. Twenty minutes after airway occlusion (50 minutes from the beginning of spontaneous breathing), the patient was unable to sustain spontaneous breathing and reinstitution of control mechanical ventilation was needed. c) With the patient relaxed, the pattern of breathing just prior to the airway occlusion was subsequently simulated by the ventilator. $PEEP_{i,st}$ was measured during the simulation under static conditions by the end-expiratory occlusion technique [6]. For further explanation, see text. $\Delta P_{oes}$: increase in end-expiratory $P_{oes}$ relative to its level at the onset of the spontaneous breathing trial (a); $P_{ga,exp,rise}$: gastric pressure decay from its maximal value to its minimal value at the beginning of the next inspiration; $PEEP_{i,occl}$: end-expiratory value of airway pressure of the first inspiratory effort after airway occlusion at the end of expiration during spontaneous breathing, using the end-expiratory hold button of the ventilator; $PEEP_{i,st}$: $PEEP_i$ measured as the value of end-expiratory plateau of airway pressure during simulation of the pattern of spontaneous breathing with the ventilator, occluding the airway by the end-expiratory hold button.

Table 2. – Respiratory mechanics at the airway occlusion during spontaneous breathing in actively (Group 1) and passively expiring patients (Group 2)

| Pt. No. | $PEEP_{i,st}$ $cmH_2O$ | $PEEP_{i,occl}$ $cmH_2O$ | $P_{ga,exp,rise}$ $cmH_2O$ | $\Delta P_{oes}$ $cmH_2O$ | $PEEP_{i,occl}$-$PEEP_{i,st}$ $cmH_2O$ | $PEEP_{i,occl}$-$P_{ga,exp,rise}$ $cmH_2O$ |
|---|---|---|---|---|---|---|
| **Group 1** | | | | | | |
| 1 | 6.6 | 18.7 | 12.0 | 17.6 | 12.1 | 6.7 |
| 2 | 7.2 | 18.6 | 11.0 | 8.3 | 11.4 | 7.6 |
| 3 | 12.3 | 19.5 | 7.5 | 5.4 | 7.2 | 12.0 |
| 4 | 10.1 | 15.8 | 6.0 | 5.9 | 5.7 | 9.8 |
| 5 | 2.5 | 5.5 | 3.1 | 3.4 | 3.0 | 2.4 |
| 6 | 4.1 | 7.2 | 3.0 | 2.8 | 3.1 | 4.2 |
| 7 | 4.8 | 7.5 | 2.7 | 2.0 | 2.7 | 4.8 |
| 8 | 7.1 | 13.4 | 6.0 | 7.4 | 6.3 | 7.4 |
| Mean±SE | 6.8±1.1** | 13.3±2.0 | 6.4±1.3 | 6.6±1.8 | 6.4±1.3 | 6.9±1.1 |
| **Group 2** | | | | | | |
| 9 | 6.5 | 6.6 | 0 | 0 | 0.1 | 6.6 |
| 10 | 2.3 | 2.3 | 0 | 0 | 0 | 2.3 |
| 11 | 5.0 | 5.2 | 0 | 0 | 0.2 | 5.2 |
| 12 | 4.0 | 4.1 | 0 | 0 | 0.1 | 4.1 |
| Mean±SE | 4.5±0.9 | 4.6±0.9 | 0 | 0 | 0.1±0.0 | 4.6±0.9 |

Pt: patient. **: $p<0.01$, significantly different from $PEEP_{i,occl}$. For further definitions see legend to figure 3.

$P_{ga}$ - AB displacement loop produced by the increasingly positive $P_{ga}$ at the same time that abdominal cross-sectional area decreased during expiration, and a positive $\Delta P_{oes}$ and $EMG_{ab}$. It is notable that palpation of the abdominal wall also revealed the abdominal muscle contraction during expiration in all Group 1 patients. In Group 2, the patients did not use their expiratory muscles at expiration; all were septic. In the $P_{ga}$ - AB displacement loops, $P_{ga}$ increased from the beginning to the end of inspiration and decreased during expiration, indicating passive expiration. Moreover, $\Delta P_{oes}$ and $EMG_{ab}$ were zero. Palpation of the abdominal wall did not detect abdominal muscle contraction in any patient of this group.

The results of measurements performed during airway occlusion are summarized in table 2. During the

airway occlusion (fig. 3) in Group 1 patients, measurements showed that $PEEP_{i,occl}$ was significantly higher than $PEEP_{i,st}$ (13.3±2.0 *vs* 6.8±1.1 $cmH_2O$; $p<0.01$; n=8). The difference, $PEEP_{i,occl}$ - $P_{ga,exp,rise}$ (6.9±1.1 $cmH_2O$), was very close to $PEEP_{i,st}$ (6.8±1.1 $cmH_2O$) (fig. 4a). Accordingly, the difference, $PEEP_{i,occl}$ - $PEEP_{i,st}$, was quite similar to $P_{ga,exp,rise}$ (and $\Delta P_{oes}$) (table 2). Good correlations were detected between $PEEP_{i,occl}$ - $PEEP_{i,st}$ and $\Delta P_{oes}$ ($r=0.869$; $p<0.01$), and $PEEP_{i,occl}$ - $P_{ga,exp,rise}$ and $PEEP_{i,st}$ ($r=0.997$; $p<0.001$) (fig. 5). The values of both the above regressions were very close to the line of identity. The mean difference between $PEEP_{i,occl}$ - $P_{ga,exp,rise}$ and $PEEP_{i,st}$ was 0.03 $cmH_2O$. The limits of agreement (*i.e.* mean difference -2SD to mean difference ±2SD) were -0.48 to +0.53 $cmH_2O$. These data convincingly indicate that, in spontaneously breathing and actively expiring patients, the so-called $PEEP_{i,occl}$, overestimates the "true" static $PEEP_i$ by an amount equal to $P_{ga,exp,rise}$ (or $\Delta P_{oes}$).

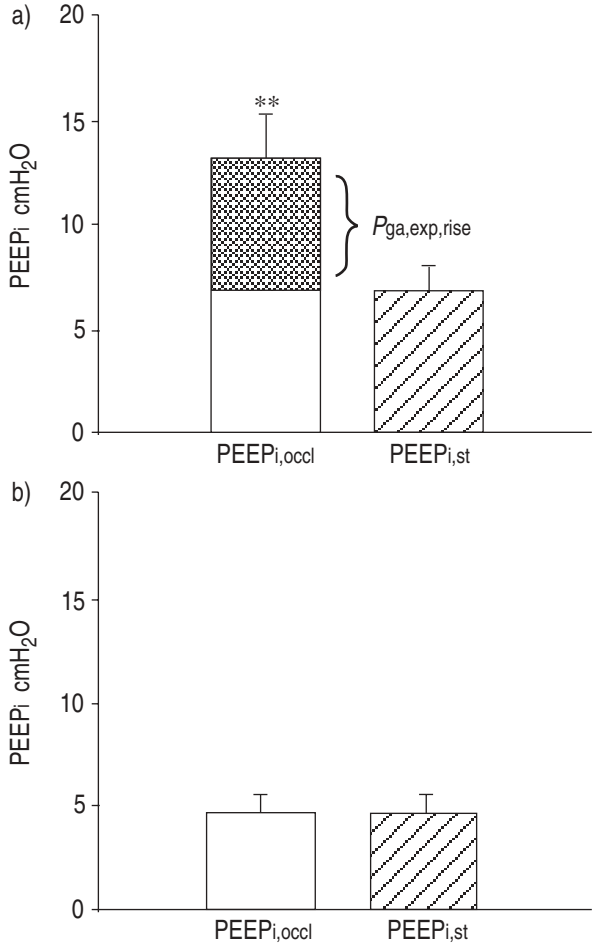


Fig. 4. – Values of $PEEP_{i,occl}$ and $PEEP_{i,st}$ in: a) actively expiring patients (Group 1) (n=8) and b) passively expiring patients (Group 2) (n=4). $PEEP_{i,occl}$ was measured from the end-expiratory plateau in airway pressure between occluded inspiratory efforts. Although an apparent plateau was not present in actively expiring patients, for the sake of consistency, in these patients the value of airway pressure at the end-expiration of the first postocclusion inspiratory effort was also referred to here as $PEEP_{i,occl}$. $PEEP_{i,st}$ was considered to represent the actual static $PEEP_i$ and was measured from end-expiratory plateau of airway pressure during simulation by the ventilator of the pattern of spontaneous breathing just before airway occlusion. $PEEP_{i,occl}$ was higher than $PEEP_{i,st}$ in actively expiring patients (a). Subtracting the mean value of $P_{ga,exp,rise}$ from that of $PEEP_{i,occl}$, the difference obtained was almost equal to $PEEP_{i,st}$. In contrast to actively expiring patients, in passively expiring patients $PEEP_{i,occl}$ was similar to $PEEP_{i,st}$ (b), indicating that $PEEP_{i,occl}$ reliably measures the actual static $PEEP_i$. **: $p<0.01$, compared to $PEEP_{i,st}$. For definitions see legend to figure 3.

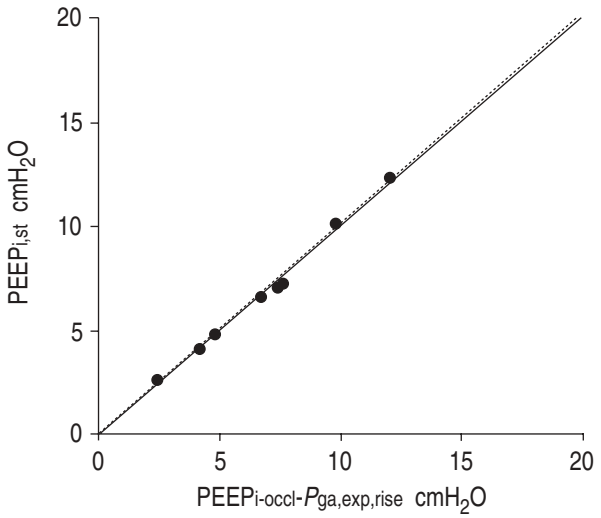


Fig. 5. – Identity plot for values of the difference $PEEP_{i,occl}$ - $P_{ga,exp,rise}$ and $PEEP_{i,st}$ in actively expiring patients (n=8). Regression equation (dotted line) is shown ($p<0.001$); all values are very close to the line of identity (solid line). This finding indicates that $PEEP_{i,occl}$ overestimates the actual static $PEEP_i$ (represented by $PEEP_{i,st}$) by an amount equal to $P_{ga,exp,rise}$. For definitions see legend to figure 3.

In contrast to Group 1, in Group 2 during the airway occlusion (fig. 2), $PEEP_{i,occl}$, and $PEEP_{i,st}$ had quite similar mean values (4.6±0.9 and 4.5±0.9 $cmH_2O$, respectively; n=4), (table 2, and fig. 4b). Individual values of $PEEP_{i,occl}$ and $PEEP_{i,st}$ were well-correlated ($r=0.999$; $p<0.001$) and were positioned close to the identity line. Their mean difference was 0.1 $cmH_2O$, and the limits of agreement were -0.06 to +0.26 $cmH_2O$. Therefore, in those patients who did not use their expiratory muscles at expiration, $PEEP_{i,occl}$ accurately represented the $PEEP_{i,st}$.

## Discussion

This study yielded two principal findings. Firstly, in spontaneously breathing and actively expiring patients, $PEEP_i$ measured with the airway occlusion ($PEEP_{i,occl}$) technique overestimates the actual $PEEP_i$. $P_{ga,exp,rise}$ can accurately express the amount of this overestimation. Thus, by subtracting $P_{ga,exp,rise}$ from $PEEP_{i,occl}$, an accurate estimation of the actual $PEEP_{i,st}$ can be made. Secondly, in passively expiring patients, the airway occlusion technique measures the actual $PEEP_{i,st}$ accurately.

### *$PEEP_{i,occl}$ measurement in the presence of expiratory effort*

The measurement of $PEEP_i$ during spontaneous breathing by the end-expiratory airway occlusion technique ($PEEP_{i,occl}$) is of particular interest for two reasons. Firstly, the value of $PEEP_i$ is obtained under static conditions, and is considered to represent an average level

of regional $PEEP_i$ reflecting the alveolar pressure after readjustment of dynamic regional volume and pressure differences [1]. In contrast, the value of $PEEP_{i,dyn}$ indicates the pressure required to initiate inspiratory flow into those lung units with the lowest levels of end-expiratory alveolar pressure [1], thus underestimating the $PEEP_i$ in the presence of regional time constant inequalities, as in patients with COPD. Secondly, and probably more important in clinical practice, $PEEP_{i,occl}$ is measured by monitoring the $P_{aw}$ or simply by observing the airway pressure manometer of the ventilator noninvasively, that is, without the need to position an oesophageal balloon. In the presence of expiratory muscle activity during expiration, however, an accurate estimation of alveolar pressure reflecting the elastic recoil of the total respiratory system by the measurement of $PEEP_{i,occl}$, cannot be obtained. In this instance, what is measured as $PEEP_{i,occl}$ simply indicates the presence of positive alveolar pressure at the end of expiration postocclusion; besides the elastic recoil of the respiratory system, expiratory muscle contraction apparently contributes to its amount.

The results of this study clearly indicate that, by subtracting $P_{ga,exp,rise}$ from $PEEP_{i,occl}$ measured at the end-expiration of the first inspiratory effort after the airway occlusion, an accurate value of the actual $PEEP_{i,st}$ can be obtained. The values of $P_{ga,exp,rise}$ used were the average of three consecutive breaths prior to the airway occlusion. These values were almost the same as the $P_{ga,exp,rise}$ of the first inspiratory effort against the occluded airway (6.4±1.3 *vs* 6.5±1.3 $cmH_2O$), indicating that expiratory muscle activity during expiration of the first inspiratory effort postocclusion was nearly the same as that during unoccluded spontaneous breathing just prior to the airway occlusion. It stands to reason, therefore, that the positive alveolar pressure measured as $PEEP_{i,occl}$ at the end of expiration of the first inspiratory effort after the airway occlusion is the sum of the actual $PEEP_{i,st}$ and $P_{ga,exp,rise}$.

### *Clinical detection of expiratory muscle contraction*

To interpret the rise in $P_{ga}$ as being a consequence of expiratory contraction of the abdominal muscles, it is necessary to check that it occurs together with a decrease in abdominal dimensions [2, 4]. In every patient included in the present study, as in the four cases of LESSARD *et al*. [4], the rise in $P_{ga}$ during active expiration occurred simultaneously with a decrease in abdominal dimensions. This finding suggests that $P_{ga}$ recording may be adequate to detect abdominal muscle contraction during expiration, without necessitating measurement of the AB displacement.

In the present study, palpation of the abdominal wall had 100% accuracy in revealing abdominal muscle activity during expiration. Therefore, when palpation of the abdominal wall can not demonstrate any abdominal muscle activity during expiration, the $PEEP_{i,occl}$ measurements obtained may be considered as accurate, without the need for $P_{ga}$ recording. On the contrary, when abdominal muscle contraction exists, the positioning of a gastric balloon is necessary to correct $PEEP_{i,occl}$ for the contribution of expiratory muscle activity. It must be stressed, however, that the patients in the present study belonged to a special category; they had acute respiratory failure and were allowed to breathe spontaneously, being in severe respiratory distress when the palpation of the abdominal wall was performed. Hence, in these actively expiring patients, the powerful abdominal muscle contraction producing a $P_{ga,exp,rise}$ of 6.4± 1.3 $cmH_2O$ (range 2.7–12 $cmH_2O$) (table 2) was easy to detect clinically; the absence of such an abdominal muscle contraction was indicative of passive expiration. This may not be the case when active expiration is associated with a weak contraction of the abdominal muscles. For example, in stable COPD [2] or during exacerbations of COPD not needing intubation [3], $P_{ga,exp,rise}$ may be only a few $cmH_2O$. In this instance, palpation of the abdominal wall may not be accurate in detecting abdominal muscle contraction at expiration.

Nevertheless, we have observed in this study that actively expiring patients after airway occlusion react by further increasing their expiratory muscle activity in an unpredictable manner. In contrast, passively expiring patients after airway occlusion do not react by recruiting their expiratory muscles and expiration remains passive throughout the airway occlusion manoeuvre (fig. 2). Therefore, an airway occlusion manoeuvre may increase the ability of palpation of the abdominal wall to detect the presence or absence of expiratory muscle contraction at expiration during unoccluded spontaneous breathing. Abdominal muscle activity during expiration that is small and undetectable by palpation during unoccluded breathing, may become apparent during airway occlusion. On the contrary, absence of abdominal muscle contraction by palpation of the abdominal wall during the airway occlusion indicates that expiration was passive during the preceding unoccluded breathing. In this instance it is not necessary to position a gastric balloon to detect expiratory muscle contraction.

### *Clinical implication*

The possible clinical implication of these findings is related to the accurate and easy measurement of $PEEP_{i,st}$ during spontaneous breathing in order to apply the appropriate level of external continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP) to decrease inspiratory muscle effort due to $PEEP_i$. Airway occlusion appears to be the method of choice for measuring $PEEP_i$ in this instance, since the $P_{aw}$ recording that is needed to obtain $PEEP_{i,occl}$ may be routinely performed in almost any clinical setting. When $P_{aw}$ tracing and/or palpation of the abdominal wall during airway occlusion indicates expiratory muscle contraction during expiration, the placement of a gastric balloon will provide $P_{ga}$ fluctuations required to correct $P_{aw}$ tracing for the contribution of expiratory muscle activity and obtain the actual static $PEEP_i$. Assessment of $PEEP_{i,dyn}$ carries the disadvantage of necessitating an additional invasive measurement, that is, $P_{oes}$. In addition, the clinical implications of $PEEP_{i,dyn}$ have not yet been fully defined [12].

In conclusion, the present study demonstrates that in spontaneously breathing and passively expiring patients the airway occlusion technique ($PEEP_{i,occl}$) accurately measures $PEEP_{i,st}$. On the contrary, in actively

expiring patients, this technique overestimates the actual $PEEP_{i,st}$ by an amount equal to the mechanical effect produced by expiratory muscle contraction at end-expiration. This effect is accurately expressed by $P_{ga,exp,rise}$ during $PEEP_{i,occl}$ measurement. Thus, to correct $PEEP_{i,occl}$ for the contribution of expiratory muscle activity, we should subtract $P_{ga,exp,rise}$ measured either exactly before the airway occlusion or at the end-expiration of the first inspiratory effort postocclusion. Palpation of the abdominal wall may help in the detection of abdominal muscle activity. When this activity is absent by palpation even during the airway occlusion manoeuvre, it is not necessary to position a gastric balloon to detect expiratory muscle contraction, and $PEEP_{i,occl}$ measurements accurately reflect the actual $PEEP_{i,st}$. When it is present, $P_{ga}$ recording is needed to correct $PEEP_i$ measurements for the contribution of expiratory muscle contraction.

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