Gas Exchange during Mechanical Ventilation and Spontaneous Breathing

Intermittent Mandatory Ventilation after Open Heart Surgery*

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Pulmonary gas exchange rates in eight patients after open heart surgery were studied during weaning from the ventilator. We investigated continuous positive pressure ventilation (CPPV), intermittent mandatory ventilation (IMV) and spontaneous breathing with continuous positive airway pressure (CPAP). During each mode of ventilation we measured: CO₂ production (VCO₂), O₂ consumption (VO₂), cardiac output (CO), PaO₂, Q̇/Q̇̇ and functional residual capacity (FRC). In addition, we analyzed in each single breath: tidal volume (Vₜ), series dead space volume (Vds), alveolar ventilation, alveolar efficiency for CO₂ elimination (alv eff CO₂) and end-tidal CO₂ concentration (FECO₂). We compared the results of CPPV, IMV and CPAP and the mandatory breaths (MB) with the spontaneous breaths (SB) measured during IMV. CO was low during CPPV, when the patient still deeply sedated; it was increased in IMV and remained constant in the following CPAP period. VCO₂ and VO₂ did not differ significantly when switching from IMV to CPAP, therefore, work due to breathing seemed not to be reduced by the mandatory breath during IMV. Oxygenation (PaO₂, Q̇̇/Q̇̇̇) did not change significantly when switching from one mode to the other. FRC was constant when changing from CPPV to IMV, did not alter within the IMV-cycle and was reduced significantly when switching from IMV to CPAP. Dead space ventilation was reduced in SB (compared to MB). The latter result is discussed on the basis of two mechanisms: Vds was reduced and alv eff CO₂ was increased. We conclude that compared to CPPV, IMV decreases mean alveolar pressure and reduces dead space ventilation at constant FRC and with constant oxygenation. This may explain why, in the weaning process, IMV makes it possible to start spontaneous breathing very early.

Intermittent mandatory ventilation (IMV) is a ventilatory mode with a complex pattern of breaths. An IMV-cycle starts with a mandatory breath (MB) imposed on the patient by the ventilator. A sequence of spontaneous breaths (SB) follows. This mode of breathing was reported to have clinical advantages over other forms of ventilation by reducing the need for sedatives, narcotics and muscle relaxants. While the results of extensive studies are available concerning effects on hemodynamics, very few quantitative data on gas exchange have been published. Some authors have measured physiologic dead space, respiratory rate, CO₂ elimination, functional shunt fraction, cardiac output by thermodilution and functional residual capacity. In other studies, mean values taken over a period of IMV are compared with mean values taken over a period of other modes of ventilation. To our knowledge, there are only two papers reporting single breath data about mandatory breath (MB) and spontaneous breaths (SB), respectively. The lack of data, together with the fact that many IMV systems are inadequately used, appears to have caused some controversy concerning the clinical application of IMV.

The purpose of this study was to investigate patients with quantitative methods appropriate for a breath-by-breath analysis. The comparison of spontaneous breaths and mechanical (mandatory) breaths should give a better understanding of the IMV mode. It is hypothesized that the quantitative measures support our good clinical experience with IMV.

Materials and Methods

We investigated eight patients after open heart surgery (Table I) during the weaning period. Immediately after the operation, the patients received volume-controlled ventilation (IPPV) and sedation with diazepam; analgesia (morphine) and muscular paralysis (pancuronium) therapy were continued. As soon as intravascular volume, cardiac rhythm (atrial pacing), core temperature and peripheral circulation were adequate, PEEP was applied (CPPV) and kept constant throughout the subsequent investigations. Inspired oxygen concentration was constant at 40 percent. After the first set of measurements was taken (CPPV, 6.6 hrs [SD ± 5.7] after the completion of operation), weaning procedure was introduced by discontinuing muscular paralysis therapy and reducing analgesic/sedative treatment. When the patients started to become active, the ventilator was switched into the IMV mode with identical PEEP. Frequency of the mechanical breaths was slowly reduced and the

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patients started to breathe spontaneously. Mandatory breaths during IMV were not triggered by the patient. As soon as the breathing pattern was regular and in phase with the ventilator, and the frequency of mandatory breaths was reduced below five per minute, a second set of measurements was taken (IMV, 10.6±5.3 hrs after completion of operation). As soon as the patients were able to breathe unassisted, the ventilator was switched into CPAP mode. When the breathing pattern remained constant, CPAP was investigated (CPAP, 12.5±4.9 hrs after completion of operation). One patient was not investigated in CPAP. All patients were extubated within 20 hrs after the operation and had a normal postoperative course. The protocol of this study was approved by the ethical committee of our institution.

All measurements were taken in steady state conditions. Steady state was assumed when the end-tidal CO₂ concentration remained constant for more than 20 min. Flow was measured at the endotracheal tube with a heated pneumotachograph (Fleisch No. 2). Airway pressure (Paw) was measured from within the endotracheal tube. A mass spectrometer (Centronic MGA 200 quadrupol) sampled gas through a 3.5 m capillary, also from within the endotracheal tube. Each set of measurements included a continuous recording of flow, pressure and gas composition over a period of 3 min during each mode of ventilation. Analog data was entered into a computer (processing of signals is described in the Appendix) as well as monitored continuously on an eight-channel chart recorder (MFE 1850-00). Simultaneously, an arterial blood sample was drawn from a systemic artery over a period of at least 20 sec and analyzed immediately for PaCO₂, PaO₂ and pH (ABL3, Radiometer, Copenhagen). Mixed venous blood was sampled simultaneously from the pulmonary artery and its oxygen saturation was determined at the bedside (OSM2, Radiometer, Copenhagen). Nitrogen washout with argon was then performed.

During CPPV, IMV and CPAP the patients were connected to a modified Monaghan M-550 ventilator. This system is equipped with a highly compliant pressurized inspiratory reservoir. CPAP was maintained by an overflow system (without demand valve), thus avoiding airway pressure drops during spontaneous breathing. Our ventilator enabled us to perform Nₐ washouts, without producing a change in the ventilatory pattern during CPPV, IMV and CPAP.³⁴

The following pulmonary function indices were calculated breath-by-breath, duration of inspiration and expiration (Ti, Te), respiratory rate (RR) as number of breaths within one minute, inspiratory volume and expiratory volume (Vi, Ve) by numerical integration of flow during inspiration and expiration respectively, tidal volume (V₉) as the mean of Vi and Ve of a breath. (Breath-by-breath estimates of V₉ were summed and divided by the period of measurement to get the minute volume [MV]), peak airway pressure (Pawmax), mean airway pressure (Pawmean) and end-expiratory airway pressure (Pawee, mean value of the last ten samples during expiration) were also calculated in each breath.

End tidal CO₂ (Fco₂-et) was measured as maximal CO₂ concentration. Volume of CO₂, O₂ and N₂ (VCO₂, VO₂, VN₂) was calculated by multiplication and integration of flow and the corresponding concentration throughout inspiration and expiration. Breath-by-breath estimates of VCO₂ and VO₂ were summed and divided by the period of measurement to get CO₂ production and O₂ consumption (VCO₂, VO₂). Physiologic dead space ratio (Vd/Vt) was calculated according to the conventional formula using arterial Pco₂ as the ideal alveolar Pco₂ (Vd/Vt=1–Vco2/MV/Faco2-ideal).

Series dead space volume (Vds) was assessed using the CO₂ diagram (Appendix). Volume reaching the alveoli was calculated as Vt–Vds (Vd). Efficiency of alveolar CO₂ elimination (alv eff-CO₂) is the quotient of measured VCO₂ and expected VCO₂ (Faco2-ideal × Vd) expressed as a percentage.

O₂ content of arterial and mixed venous blood were used together with the ideal capillary O₂ content to calculate the fraction of venous admixture Qs/Qt.² O₂ contents of arterial and mixed venous blood were used, together with Vco₂ to calculate cardiac output (CO) according to the Fick principle. N₂ washout was used to determine FRC.

We present the results of the single breath analysis as mean values.

For CPPV and CPAP, a consecutive series of 15 to 20 breaths was analyzed and the mean values of the breath-by-breath indices are reported. For IMV, a consecutive series of 12 to 20 IMV cycles was analyzed. Mean values were determined separately for mandatory breaths (MB). No difference was made between spontaneous breaths within an IMV cycle; one mean value was calculated (SB). The number of mandatory breaths and the number of spontaneous breaths was counted and related to one minute, yielding RRs and RRˢ. The quotient RRˢ/RRᵇ gives the number of spontaneous breaths within an IMV cycle. For Vr, Vds, alv eff-CO₂ and Fco₂, we calculated the difference between CPPV and the mandatory breaths in IMV as well as the difference between CPAP and the spontaneous breaths in IMV for each patient (paired differences). All spontaneous breaths with tidal volumes of less than 200 ml, of which there were 42, were discarded for the breath-by-breath analysis. Statistical analysis was carried out with the Wilcoxon signed rank test for paired samples (p<0.05).

RESULTS

A summary of results in CPPV, IMV and CPAP is presented in Table 2. Single breath indices during IMV

<table>
<thead>
<tr>
<th>Table 1—Description of Patients</th>
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<tr>
<td>Patient</td>
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<tr>
<td>1</td>
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<td>2</td>
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<td>8</td>
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<tr>
<td>Mean</td>
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<td>SD</td>
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IMV after Open Heart Surgery (Wolff, Brunner, Gradel)
Table 3—Breath-by-breath Indices of Gas Exchange during IMV

<table>
<thead>
<tr>
<th>Patient</th>
<th>Breath</th>
<th>VT (ml)</th>
<th>RR (l/min)</th>
<th>FCO₂et (vol%)</th>
<th>Vds (ml)</th>
<th>alv eff-CO₂ (%)</th>
<th>Va (L/min)</th>
<th>Pao₂ (mm Hg)</th>
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<tr>
<td>1</td>
<td>MB</td>
<td>1203</td>
<td>2.9</td>
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<td>114</td>
<td>82.2</td>
<td>3.16</td>
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<td></td>
<td>SB</td>
<td>460</td>
<td>8.8</td>
<td>6.3</td>
<td>96</td>
<td>94.4</td>
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<td>SB</td>
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<td>6.6</td>
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<td>MB</td>
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<td>88.7</td>
<td>3.40</td>
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<td>445</td>
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<td>6.1</td>
<td>79</td>
<td>91.3</td>
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<td>405</td>
<td>5.1</td>
<td>6.1</td>
<td>87</td>
<td>77.3</td>
<td>1.93</td>
<td>8.6</td>
</tr>
</tbody>
</table>

| mean MB | 21 | 1172 | 3.8 | 5.9 | 197 | 81.2 | 3.7 | 26.8 |
| mean SB | 91 | 149 | 0.6 | 0.5 | 133 | 6.5 | 0.6 | 4.7 |
| mean Δ  | 21 | 623* | 5.1* | 0.2* | 59* | 6.2* | 0.2 | 19.4* |

*Difference is statistically significant at p<0.05.

Breath-by-breath indices were stable in each patient. Mean SEMs are given in Table 4 for CPPV, IMV and CPAP. The largest scatter was observed in the SB during IMV.

Mean values describing CPPV and CPAP compared with the IMV period are given in Figure 1. FRC was decreased in IMV compared to CPPV in some patients. The difference, however, did not reach the level of significance; whereas from IMV to CPAP, the small fall in FRC (mean difference = 210 ml) was statistically significant. There were large and significant changes of CO and mean airway pressure. VCO₂, MV and PaO₂ changed very little and non-uniformly from CPPV to IMV and from IMV to CPAP.

Some breath-by-breath indices of CPPV, IMV and CPAP are given in Figure 2. Tidal volumes of the MB in IMV were similar to those in CPPV and tidal volumes of the SB in IMV were similar to those in CPAP. On the other hand, FCO₂et and PaCO₂ were significantly increased in IMV and CPAP as compared with CPPV.

**DISCUSSION**

Since pleural pressure rises during inspiration in MB and falls in SB, we hypothesized that gas exchange rates in mandatory breathing and spontaneous breathing differ. This hypothesis is difficult to investigate quantitatively by comparing the mean values of a period of IMV with the mean values of a period of CPPV and CPAP, respectively. It is necessary to compare mandatory breaths and spontaneous breaths separately. Consequently, we had to use methods suitable for breath-by-breath analysis. The disadvantage of these methods is the poor resolution with respect to ventilation-perfusion (V/Q) ratios, which means that we were unable to distinguish between alveolar dead space ventilation and ventilation of areas with poor but finite perfusion, with the exception of the series dead space ventilation. Consequently, we are unable to distinguish between a broad unimodal and a multimodal distribution of ventilation. The same applies to low
FIGURE 1. Net effect of gas exchange during periods of CPPV, IMV and CPAP. Mean airway pressure (Pawo), accessible pulmonary gas volume to nitrogen washout (FRC), cardiac output (CO) and VT are plotted as mean values. An asterisk indicates statistically significant changes in respect to IMV.

VA/Q ratios in respect to Qs/Qt. There are methods which give better resolution in terms of VA/Q ratios under the assumption of a steady state. These methods, however, are limited in respect to time-resolution, are not suited to studying breath-by-breath differences, and do not assess series dead space ventilation.

On average, IMV was investigated four hours later than CPPV and, therefore, the level of anesthesia was apparently reduced when IMV was investigated. In contrast, only two hours had passed on average between the investigations of IMV and CPAP, and the level of anesthesia may be considered comparable. Mean body temperature was 37 ± 1.7°C; it increased slightly from CPPV to IMV (mean change in temperature 1.3°C) and remained constant thereafter. Therefore, differences in physiologic measurements between CPPV and IMV, and perhaps also between IMV and CPAP, are not necessarily due to the mode of ventilation alone. Such a systematic difference in sedation and temperature may be reflected in the low CO during CPPV as compared to IMV and CPAP. Mean airway pressure did not fall below PEEP in IMV and in CPAP. Obviously, our IMV system adequately maintained the end-expiratory pressure without loading the patients' breathing system with high inspiratory flow resistance.

CPPV Compared with IMV

No uniform change in CO₂ production was found switching from CPPV to IMV (nor from IMV to CPAP). In a study of other authors in patients similar to ours, but using an IMV system with demand valves, CO₂ production during CPPV was found to be similar but increased by 40 percent during IMV.¹⁸ In that study, the ratio of CO₂ elimination and O₂ uptake (RQ) was found to be 1.03 in CPPV and decreased to 0.68 to 0.77 during IMV, a result which has not been mentioned in the discussion of that paper. In our study, RQ was about 0.95 during CPPV and did not decrease during IMV or CPAP. We are reluctant to attribute these discrepancies to differences between patients. They point, rather, to technical difficulties due to the fact that the sensors in that study were placed at different sites, ie, not all directly at the airway opening. We consider the measurement of gas flow and gas concentrations directly at the airway opening indispensable to obtain accurate results during IMV.

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With the exception of one patient, mean airway pressure was reduced in IMV. This result is plausible since the thorax is no longer passively inflated during IMV. Gas flow into the lungs (at least during SB) is caused by lowering the alveolar pressure, thereby reducing mean pressure at the airway opening.

In IMV, we measured a slight increase in $V_d/V_t$ (Fig. 1), a result which is contradicted by some other authors. Physiologic dead space ventilation ($V_d/V_t$) is composed of series dead space ventilation and alveolar dead space ventilation (measured as the inverse of $alv_{eff-CO_2}$). Alveolar dead space ventilation, as well as $V_d$, are decreased during IMV (Fig 2). But the small spontaneous volumes during IMV show a higher $V_d$ to $V_t$ ratio. The series dead space ventilation (in terms of $ml/min$) is therefore increased in spite of a decreased $V_d$ (in terms of $ml/breaths$). Our conclusion is that the slightly increased $V_d/V_t$ observed in our patients during IMV is caused by an increased series dead space ventilation per minute and not by an increased alveolar dead space ventilation. This result depends not only on alterations of $V_d$ and $alv_{eff-CO_2}$, but also on the ratio $RR^{SB}/RR^{MB}$ and, consequently, not only on the patient but also on the setting of the ventilator.

**IMV$^{SB}$ Compared with IMV$^{MB}$ (Fig 2, Table 3)**

IMV is composed of mandatory breaths and spontaneous breaths. They differ in two major features: the tidal volumes of MB are larger than those of the SB, and pleural pressure rises during mandatory inspiration, whereas it falls during spontaneous inspiration.

Inspired and expired volume of MB did not differ. The same was true for SB. Consequently, end-expiratory lung volume or functional residual capacity was constant throughout the IMV cycle.

Series dead space volume ($V_d$) is much larger in MB than in SB (mean change, $59 \, ml$). $V_d$ develops during inspiration and three mechanisms could contribute to this alteration. It has been known for some time that $V_d$ is increased if end-inspiratory lung volume ($V_L$-$E$I) is increased. The tidal volumes of the spontaneous breaths were only $46\%$ percent (mean) compared to the tidal volumes of the mandatory breaths, resulting in a larger end inspiratory lung volume in the MB. Therefore, one would expect $V_d$ to be larger in the MB. However, in the study mentioned, $\Delta V_d/\Delta V_L$-$E$I is $25 \, ml/L$. In our study, the mean difference in end-inspiratory lung volume after SB compared to after MB is $620 \, ml$, which explains a difference in $V_d$ of only $15 \, ml$, ie, this mechanism cannot fully explain our results. Two further mechanisms have to be discussed, both based on the concept that $V_d$ is contained within anatomic and functional walls. It has been shown that, during positive pressure ventilation, convective airways are distended. We can therefore assume that during IMV convective airways are distended more in MB and less in SB. Since convective airways are the anatomic walls of $V_d$, we conclude that distensibility of the airways is the second mechanism of increase in $V_d$ in MB compared to SB. Stationary interfaces (SI) between fresh gas and alveolar gas are considered functional walls of $V_d$. The position of SI may be moved upwards (towards the mouth) by decreasing the inspiratory convective flow, as well as by an end-inspiratory pause. Inspiratory flow during MB is constant (rectangular) up to the end of inspiration, the transition of inspiration to expiration being very fast (<0.1 sec). On the other hand, in SB, inspiratory flow comes down to zero slowly. Since the position of the SI depends on inspiratory flow pattern, particularly at the end of inspiration, we conclude that the different breathing pattern causes the difference in $V_d$. All three mechanisms (end-inspiratory lung volume, end-inspiratory airway pressure and inspiratory flow pattern) reduce $V_d$ in SB compared to MB. We cannot, however, assess the magnitude of the individual effects.

End-tidal concentration of $CO_2$ ($F_{CO_2}$) increased slightly in SB (mean change, $0.2 \, percent$) and the efficiency for alveolar $CO_2$ elimination ($alv_{eff-CO_2}$) was far greater in SB than that in MB. To explain these observations, we discuss three possible mechanisms: first, alveolar ventilation is decreased in MB; second, dead space ventilation is increased in MB; third, pulmonary perfusion is decreased in MB. The change in alveolar ventilation is very small and non-uniform (Table 3), and therefore most probably is not the explanation of our results. It has been shown that in supine, spontaneously breathing patients, inspired gas is preferentially distributed to dependent lung regions. In contrast, during CPPV ventilation, inspired gas is directed to non-dependent lung regions. Although these effects are mixed during IMV, they may lead to large differences of ventilation and perfusion between spontaneous breaths and mandatory breaths. We have measured an increased $alv_{eff-CO_2}$ in the spontaneous breaths compared with the mandatory breaths in each patient. This result suggests that ventilation in high $V_a/Q$ regions in MB is further increased, and that the findings just mentioned may be important even within an IMV cycle. It is not clear what the diaphragm contributes to this difference. Since the diaphragm is usually active in SB, it is very unlikely that no diaphragmatic muscular action takes place in MB.

Displacement of blood from the thorax during positive pressure ventilation has been recognized for many years. Recently it has been demonstrated that, during positive pressure ventilation, pulmonary capillary perfusion is reduced at the end of inspiration. Thus, $V_a/Q$ may vary even within a respiratory cycle. This tidal (temporal) change of $V_a/Q$ was considered to contribute to the increased $V_d/V_t$ during artificial
ventilation. Our results support the hypothesis that such effects also take place within MB during IMV.

Our results suggest that, during spontaneous breaths, either capillary perfusion in non-dependent regions is increased or ventilation is redistributed towards regions of lower $V_a/Q$ ratios, or both.

**IMV Compared with CPAP (Fig 1 and 2)**

CO$_2$ production, as well as cardiac output and a-vDO$_2$, remained constant although in CPAP the patients had to breathe on their own. Therefore, mandatory breathing during IMV does not necessarily reduce the amount of a patient’s work due to breathing, provided an adequate IMV system is used. FRC was decreased in CPAP. There was no significant change in arterial oxygenation and Qs/Qt did not change. Therefore, in situations where a reduced FRC could cause clinical complications, IMV might be an advantage compared to CPAP.

**CONCLUSION**

In conclusion, we summarize our findings as follows: 1) During IMV, alv eff-CO$_2$ is always much larger in SB than in MB. This observation supports the hypothesis that the match between perfusion and ventilation during SB is better than during MB. 2) There is some controversy in the literature over the effect of IMV on Vd/Vt. However, Vd/Vt is composed of series dead space ventilation and alveolar dead space ventilation, and changes of Vds and alv eff-CO$_2$ may depend on different mechanisms. We conclude that Vd/Vt is not a useful index and should not be used to describe IMV. 3) O$_2$ uptake and CO$_2$ elimination were not reduced when changing from IMV to CPAP. We conclude that, even with good equipment, IMV (compared to CPAP) does not necessarily reduce the work of breathing. 4) We have had excellent clinical experience with the IMV system used in this study. We have the impression that IMV makes it possible to start with spontaneous breathing when the patient is still unable to maintain adequate blood gas levels on his own. Our study demonstrates that, in this situation, mean airway pressure can be reduced with a constant FRC, constant oxygenation and less dead space ventilation.

**APPENDIX**

Analog data was entered into an LSI 11/23 microcomputer by means of a 12 bit analog-to-digital converter at a sampling rate of 60/sec. The influence of changing gas viscosity on flow measurement was corrected for in each flow sample, ie, in each instantaneous value. Cracking of CO$_2$ into CO within the mass spectrometer was compensated for. Concentration signals and flow signals were synchronized with a dynamically adjusted delay time according to the measured gas viscosity of the breath under consideration.

All instantaneous values from the A/D converter were read into the computer in an unstructured, continuous data stream. Automatic recognition of inspiration and expiration is therefore necessary for a breath-by-breath analysis, and consequently the algorithm distinguishing between the two phases is the kernel of the computer program. Our algorithm is based on the correlation of gas flow and its CO$_2$ content and is defined in three steps: 1) the start of an inspiratory phase is indicated by the most recent crossing of the flow signal across zero given that CO$_2$ at the mouth rises above 1 vol%, 2) the start of an inspiratory phase is indicated by the most recent crossing of the flow signal across zero given that CO$_2$ at the mouth falls below 2 vol%, and 3) the end of one phase is equivalent to the start of the other phase.

Series dead space volume (Vds) is assessed using the CO$_2$ diagram (CO$_2$ concentration vs expired volume). In a first step, it is focused on phase 2 of the CO$_2$ diagram by looking up the volume (Vap) where $\frac{1}{2}$ of the end tidal CO$_2$ is reached. This volume is doubled and the first derivative of all data points from the beginning of expiration to $2 \times V_a$ is calculated. The rest of the data ($>2 \times Vap$) is discarded. The mean value of the first derivative is calculated and is called the pre-interface expire. This volume is taken to be the series dead space volume.

Arterial PO$_2$ is converted into oxygen saturation using the subroutine of Kelman, taking arterial PCO$_2$, pH and temperature into account. Oxygen saturation of blood is converted into content using the equation CO$_2$ = 1.39 + Hb x ScO$_2$ + 0.003 x PaCO$_2$ with Hb in g/100 ml blood, ScO$_2$ in fraction of 100, and PaCO$_2$ in mm Hg.

During the nitrogen washout, VN$_2$ is measured in each breath and summed up over the entire washout, yielding VN$_{2,wet}$. The washout is finished as soon as the end tidal N$_2$ concentration is less than 1 percent. The accessible pulmonary gas volume (FRC) is calculated using the initial N$_2$ concentration (FN$_2$) and the end tidal N$_2$ concentration (FN$_2$E) of the last breath analyzed:

$$FRC = \frac{VN_2}{FN_2 - FN_2E}.$$  

Since no nitrogen washout lasts longer than 10 min, no correction is made for tissue-nitrogen. Breath-by-breath variations of FRC are assessed by calculating the difference between inspiratory and expiratory volumes ($V_i - V_e$).

The accuracy of the FRC measurements was tested with six washouts with six different FRCs on a simple lung model. The regression analysis between measured and actual FRC gave a slope of 1.02 and an intercept of 41 ml ($r^2 = 0.9996$). To test the reproducibility of the nitrogen washout we performed duplicate measurements in nine patients during CPPV, five patients during IMV and six patients during CPAP. The differences were $-14 \pm 66$ ml, $91 \pm 90$ ml and $\pm 223$ ml for CPPV, IMV and CPAP respectively.

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