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Influence of SIMV plus inspiratory pressure support on $\dot{V}_A/\dot{Q}$ distributions during postoperative weaning

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Abstract. Since the introduction of synchronized intermittent mandatory ventilation (SIMV) several advantages have been attributed to this ventilatory mode, one of them being a more homogeneous distribution of ventilation and perfusion than during controlled mechanical ventilation (CMV). Up to now no data are available to confirm whether this is true when SIMV is used in combination with inspiratory pressure support (IPS). Therefore, we compared the influence of CMV and SIMV+IPS on the distributions of ventilation and perfusion in 9 patients undergoing weaning from postoperative mechanical ventilation. Continuous distributions of ventilation and perfusion were assessed using the multiple inert gas elimination technique (MIGET). SIMV+IPS did not induce any change in the hemodynamic or oxygenation parameters, in particular CI and PaO_2 remained constant. Physiological dead space ($\dot{V}_D/\dot{V}_T$) increased, but $PaCO_2$ remained unchanged due to increased minute ventilation (from $9.5 \pm 0.9\ l \cdot min^{-1}$ to $11.3 \pm 1.2\ l \cdot min^{-1}$). The perfusion distributions remained unaltered; there was no change in $\dot{Q}_S/\dot{Q}_T$ nor in the perfusion of the low $\dot{V}_A/\dot{Q}$ lung regions. This result was underscored by the unchanged dispersion of the perfusion distribution (log SD$\dot{Q}$). The increased $\dot{V}_D/\dot{V}_T$ was caused by increased inert gas dead space (from 22.0 ± 9.6 to $26.8 \pm 8.7\%$) which was accompanied by increased ventilation of lung regions with high $\dot{V}_A/\dot{Q}$ ratios ($10 < \dot{V}_A/\dot{Q} < 100$) in 3 patients. These results show that in our group of patients partial removal of CMV together with pressure support assistance of spontaneous ventilation did not induce a clinically significant loss of the efficiency of the breathing pattern. Since the unchanged hemodynamic parameters were accompanied by increased minute ventilation, arterial blood gases did not deteriorate. Hence, SIMV+IPS proved to be useful for weaning from postoperative mechanical ventilation.

Key words: Inspiratory pressure support – Synchronized intermittent mandatory ventilation – Ventilation/perfusion distribution – Weaning

Synchronized intermittent mandatory ventilation (SIMV) has an important place during weaning from controlled mechanical ventilation (CMV). Since its introduction [1] several advantages have been attributed to this ventilatory mode. One of these potential advantages is a more homogeneous distribution of ventilation and perfusion ($\dot{V}_A/\dot{Q}$) than during CMV. Weismann and coworkers [2] hypothesized that the spontaneous breathing during SIMV improves the ventilation of dependent lung regions: spontaneous breathing preferentially distributes ventilation to basal lung regions and thus compensates for the mechanical positive pressure breaths which favour the ventilation of non-dependent lung regions due to the inactive diaphragm. In fact, Wolff and coworkers [3] found increased efficiency of alveolar ventilation for the spontaneous breaths during SIMV compared to CMV in patients after open-heart surgery.

Up to now no data are available whether this assumption is also valid when the spontaneous breath is assisted by inspiratory pressure support (IPS), a ventilatory mode which is frequently combined with SIMV in order to reduce the work of breathing, in particular its components attributable to the resistance of the ventilatory circuit and the endotracheal tube [4]. Therefore the aim of our study was to compare the effects of SIMV combined with IPS with those of CMV on the distributions of ventilation and perfusion.

Patients and methods

Nine consecutive patients (male, age 61.4 ± 9.3 years) were studied during weaning from postoperative controlled mechanical ventilation after major abdominal aortic surgery. The mean ventilation period was 30 h (range 24–36 h). None of the patients had a history nor clinical or radiological signs of chronic obstructive lung disease (COPD). All pa-

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tients had a pulmonary artery wedge pressure (PAWP) below 15 mmHg and a cardiac index (CI) above 2.5 l/min·m^2. Radial artery and thermodilution pulmonary artery catheters had been placed for routine perioperative monitoring. The patients' lungs were ventilated via an endotracheal tube with a volume-cycled Servo 900 C respirator (Siemens Elema, Sweden) with the patients comfortably resting in the semirecumbent position. During the study period the positive end-expiratory pressure, FiO_2 (mean 0.38, range 0.3–0.45), i.v. fluid administration and treatment with vasoactive or cardiotonic drugs were kept at maintenance levels used before the study. The study protocol had been approved by the local ethical committee and was carried out according to the principles embodied in the Declaration of Helsinki.

The following parameters were obtained: 1) tidal volume (V_T) and minute ventilation ($\dot{V}_E$) with a calibrated ultrasound spirometer (model VW 90, Bourns Inc., Riverside, California); 2) inspiratory PO_2 (PiO_2) of a gas sample from the inspiratory limb of the ventilator and mixed expired PCO_2 ($PeCO_2$) from a mixing box for the expired gas samples (ABL 30, Radiometer, Copenhagen); 3) arterial (a) and mixed venous ($\bar{v}$) pH, PO_2, PCO_2 (ABL 30); 4) total hemoglobin (Hb) and hemoglobin oxygen saturation (SO_2) by spectrophotometry (OSM2 Hemoximeter, Radiometer, Copenhagen); 5) systemic (SAP), mean pulmonary artery (PAP), right atrial (RAP) and pulmonary artery wedge pressures (PAWP) (Combitrans transducers, Braun Melsungen, Melsungen, FRG), all pressures being taken at end-expiration. The values reported during SIMV+IPS represent an average over 30 s of the end-expiration measurements; 6) cardiac output by thermodilution (monitor 560A, modul 582, Kone, Finland), the values being the mean of 4–5 injections of 10 ml 0°–8 °C saline randomly spread over the respiratory cycle. The coefficient of variation (SD/mean·100) of the cardiac output determination was 7.4%.

The vascular pressures and a lead II electrocardiogram were continuously recorded on a VP 95 thermoprinter (Saikosha, Japan).

Oxygen uptake ($\dot{V}O_2$), physiological dead space ($\dot{V}_D/\dot{V}_T$) and venous admixture ($\dot{Q}_{VA}/\dot{Q}_T$) were calculated using standard formulae.

$$\dot{V}O_2 = (CaO_2 - C\bar{v}O_2)\cdot CI$$

$$\dot{V}_D/\dot{V}_T = (PaCO_2 - PeCO_2)/PaCO_2$$

$$\dot{Q}_{VA}/\dot{Q}_T = (CcO_2 - CaO_2)/(CcO_2 - C\bar{v}O_2)$$

where CI is cardiac index, $PaCO_2$ is arterial PCO_2, CaO_2 and $C\bar{v}O_2$ are arterial and mixed venous oxygen contents calculated as $Hb\cdot SO_2\cdot 1.34 + PO_2\cdot 0.0031$ and CcO_2 is ideal capillary oxygen·content calculated as $Hb\cdot 1.34 + P_AO_2\cdot 0.0031$. The alveolar PO_2 (P_AO_2) was derived from the P_iO_2 with the alveolar gas equation assuming a respiratory coefficient (RQ) of 0.8.

Continuous ventilation-perfusion ($\dot{V}_A/\dot{Q}$) distributions were assessed using the multiple inert gas elimination technique (MIGET) of Wagner et al. [5] as described previously [6]. Briefly, Ringer's lactate equilibrated with 6 inert gases (sulphur hexafluoride (SF_6), ethane, cyclopropane, halothane, ether and acetone) was infused into a peripheral vein. Arterial and mixed venous blood and mixed expiratory gas samples taken from a specially designed heated mixing box were analyzed for the inert gases with a gaschromatograph (SiChromat 1, Siemens, Cologne, FRG) equipped with a sample splitter and a flame ionisation and an electron capture detector. The coefficient of variation of inert gas concentrations was 3.6% for SF_6 and 2.5–3.8% for the other 5 gases in subsequently obtained blood samples. Inert gas solubilities in blood were measured for each patient and retention-solubility and excretion-solubility curves were calculated. Continuous ventilation-perfusion distributions ($\dot{V}_A/\dot{Q}$) in a 50-compartment lung model were computed using an appropriate algorithm [7]. The remaining sum of squares was 6.1±7.4 indicating compatibility between measured and calculated inert gas elimination data [8]. Computer-assisted analysis allowed to discriminate: right-to-left shunt ($\dot{Q}_S/\dot{Q}_T$, $\dot{V}_A/\dot{Q} < 0.005$), perfusion of lung regions with low (low $\dot{V}_A/\dot{Q}$, $0.005 < \dot{V}_A/\dot{Q} < 0.1$) $\dot{V}_A/\dot{Q}$ ratios, ventilation of lung regions with high (high $\dot{V}_A/\dot{Q}$, $10 < \dot{V}_A/\dot{Q} < 100$) $\dot{V}_A/\dot{Q}$ ratios and inert gas dead space ($\dot{V}_D/\dot{V}_T$ (IG), $\dot{V}_A/\dot{Q} > 100$). The logarithm of the standard deviation of the mean for the ventilation and the perfusion (log $SD\dot{V}_A$ and log $SD\dot{Q}$) distributions as well as the mean $\dot{V}_A/\dot{Q}$ ratio for ventilation and perfusion (mean $\dot{V}_A$ and mean $\dot{Q}$) were calculated to evaluate the dispersion and a putative shift, respectively, of these distributions.

Statistical analysis was performed using a non-parametric Wilcoxon rank sign test for paired variables. Significance was assumed when the p value was below 0.05.

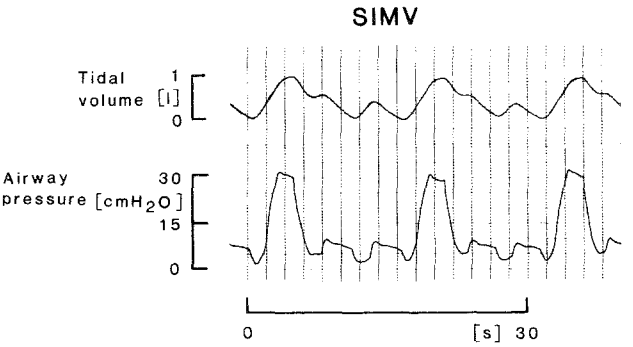

Fig. 1. Original recording of tidal volume (*above*) and airway pressure (*below*) during SIMV+IPS. The time scale demonstrates that the mechanical respiratory rate was reduced to 4 breaths/min in this patient. The upper panel registration demonstrates that the spontaneous tidal volumes reached about 30–50% of the mechanical breath

Protocol

The order of the ventilatory modes was randomized. During CMV the patient were ventilated with a tidal volume of 14–16 ml/kgBW and a respiratory rate of 8–10 breaths/min. During SIMV+IPS the mechanical respiratory rate was reduced to 4–5 breaths/min, i.e. 50% of the CMV value. The spontaneous breaths were assisted by an IPS, its level being adjusted so that the spontaneous tidal volumes reached about 50% of the mechanical breaths (IPS = 5–8 cm H_2O). A representative recording of airway pressures and tidal volumes is presented in Fig. 1. For each ventilatory mode the respective set of measurements was obtained after 45 min had elapsed at stable hemodynamic and respiratory conditions, i.e. vascular pressures and CI were constant as well as respiratory rate and minute ventilation.

Results

When switched to SIMV+IPS, none of the patients showed any signs of respiratory muscle dysfunction, such as hypercapnia, tachypnea, dyspnea or paradoxic breathing [9]. Consequently, in all patients the weaning attempts were successfully continued resulting in extubation within 4–8 h after the study period.

The hemodynamic and the O_2 and CO_2 exchange data are summarized in Table 1. SIMV+IPS did not induce any changes of the intravascular pressures or the CI. The oxygenation parameters PaO_2, $\dot{Q}_{VA}/\dot{Q}_T$ and calculated oxygen uptake remained unaltered as well. In all patients physiological dead space ($\dot{V}_D/\dot{V}_T$) increased during SIMV+IPS (from 37.2±9.2% to 42.5±8.5%, $p < 0.05$). This rise in $\dot{V}_D/\dot{V}_T$ was accompanied by increased minute ventilation (from 9.5±0.9 l·min^{-1} to 11.3±1.2 l·min^{-1}, $p < 0.05$), resulting in constant $PaCO_2$ values.

The overall inert gas data are summarized in Table 2. The perfusion distributions remained virtually unaltered,

Table 1. Hemodynamic, O_2 and CO_2 exchange data: heart rate (HR), mean systemic arterial pressure (SAP), mean pulmonary artery pressure (PAP), right atrial pressure (RAP), pulmonary artery wedge pressure (PAWP), cardiac index (CI), minute ventilation ($\dot{V}_E$), respiratory rate (RR), arterial PO_2 (PaO_2), arterial PCO_2 ($PaCO_2$), physiological dead space ($\dot{V}_D/\dot{V}_T$), venous admixture ($\dot{Q}_{VA}/\dot{Q}_T$) and calculated oxygen uptake ($\dot{V}O_2$). All values are mean ± standard deviation, and *asterisks* denote a significant difference between the two ventilatory modes ($p<0.05$)

	CMV	SIMV+IPS
HR (1/min)	85 ±11	87 ±14
SAP (mmHg)	84.8±16.7	86.9±17.0
PAP (mmHg)	22.6± 7.4	22.7± 6.6
RAP (mmHg)	6.9± 3.1	7.5± 2.2
PAWP (mmHg)	8.9± 5.0	9.5± 3.2
CI (l/min·m^2)	3.3± 0.5	3.6± 0.7
$\dot{V}_E$ (l/min)	9.5± 0.9	11.3± 1.2*
RR (1/min)	9 ± 1	16 ± 2*
PaO_2 (mmHg)	106.9±25.3	117.9±19.7
$PaCO_2$ (mmHg)	34.0± 6.1	34.5± 6.4
$\dot{V}_D/\dot{V}_T$ (%)	37.2± 9.8	42.5± 8.5*
$\dot{Q}_{VA}/\dot{Q}_T$ (%)	18.2± 6.2	16.0± 5.9
$\dot{V}O_2$ (ml/min·m^2)	153 ±25	155 ±20

Table 2. Inert gas data during CMV and SIMV+IPS: right-to-left shunt ($\dot{Q}_S/\dot{Q}_T$, $\dot{V}_A/\dot{Q}<0.005$), perfusion of lung regions with low$\dot{V}_A/\dot{Q}$ ratios (low$\dot{V}_A/\dot{Q}$, $0.005<\dot{V}_A/\dot{Q}<0.1$), ventilation of lung regions with high$\dot{V}_A/\dot{Q}$ ratios (high$\dot{V}_A/\dot{Q}$, $10<\dot{V}_A/\dot{Q}<100$), inert gas dead space ($\dot{V}_D/\dot{V}_T$(IG), $\dot{V}_A/\dot{Q}>100$), logarithm of standard deviation of mean $\dot{V}_A$ (log SD$\dot{V}_A$), logarithm of standard deviation of mean $\dot{Q}$ (log SD$\dot{Q}$), mean $\dot{V}_A/\dot{Q}$ ratio for ventilation (mean $\dot{V}_A$) and mean $\dot{V}_A/\dot{Q}$ ratio for blood flow (mean $\dot{Q}$). All values are mean ± standard deviation, *asterisks* denote a significant difference ($p<0.05$) between the two ventilatory modes

	CMV	SIMV+IPS
$\dot{Q}_S/\dot{Q}_T$ (%)	12.6 ±7.6	14.3 ± 8.6
low$\dot{V}_A/\dot{Q}$ (%)	6.4 ±7.0	1.9 ± 3.1
high$\dot{V}_A/\dot{Q}$ (%)	5.2 ±6.3	7.6 ±11.6
$\dot{V}_D/\dot{V}_T$ (IG) (%)	22.0 ±9.6	26.8 ± 8.7*
log SD$\dot{Q}_T$	1.15±0.5	0.88± 0.5
log SD$\dot{V}_A$	0.87±0.4	0.96± 0.5
mean $\dot{V}_A$	1.67±0.69	2.15± 1.39*
mean $\dot{Q}$	0.61±0.24	0.78± 0.5

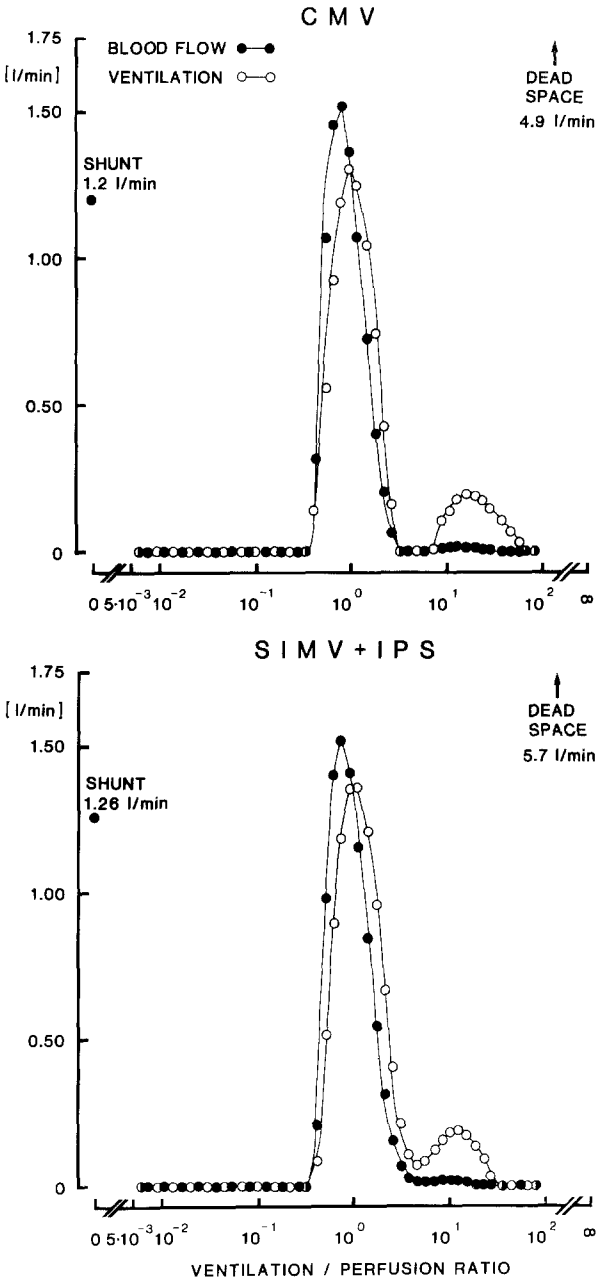

Fig. 2. Continuous ventilation-perfusion distributions of a representative patient during CMV (*above*) and SIMV (*below*). The *open circles* represent alveolar ventilation, while the *closed circles* are pulmonary blood flow. Note that while inert gas dead space increased, the ventilation dispersion and the distribution of blood flow remained virtually unchanged

in particular there was no increase in $\dot{Q}_S/\dot{Q}_T$ nor in the perfusion of low $\dot{V}_A/\dot{Q}$ lung regions. The increased physiological dead space was due to an increased inert gas dead space, which was accompanied by an increased ventilation of lung regions with high $\dot{V}_A/\dot{Q}$ ratios in three patients. Consistent with these results the mean $\dot{V}_A/\dot{Q}$ ratio for perfusion remained unchanged while the mean $\dot{V}_A/\dot{Q}$ for ventilation increased significantly. The dispersion indices log SD$\dot{V}_A$ and log SD$\dot{Q}$ were not influenced during SIMV+IPS. Fig. 2 shows continuous $\dot{V}_A/\dot{Q}$ distributions obtained from a representative patient.

Discussion

This study was to investigate the influence of SIMV combined with IPS on the distributions of ventilation and perfusion. Gas exchange alterations were analyzed by using the multiple inert gas elimination technique (MIGET) which allows good resolution in terms of $\dot{V}_A/\dot{Q}$ ratios independently of O_2 and CO_2 partial pressures in the blood and the respiratory gases [5].

SIMV+IPS did not induce any hemodynamic changes, in particular CI remained constant. Although removal of CMV generally causes an increase in CI due to increased venous return [10], this was unlikely to occur in our patients: in contrast to previous reports [10, 11]

mechanical ventilation was not completely removed during SIMV+IPS. Moreover, the PEEP level was maintained, and mean intrathoracic pressure probably did not change substantially because of the inspiratory pressure support for the spontaneous breaths. Hence, venous return probably did not change significantly, resulting in unchanged CI.

No change was calculated for $\dot{V}O_2$ during SIMV+IPS, evidence that spontaneous respiration did not account for a substantially increased work of breathing. Although spontaneous breathing has been reported to increase oxygen demands in mechanically ventilated patients [12], such an effect could not be demonstrated in our patients: on the one hand the spontaneous breaths were assisted by an inspiratory pressure support which is known to considerably reduce the work of breathing [4] and, hence, to restrain oxygen demand of the respiratory muscles [13]. On the other hand, none of our patients exhibited any signs of respiratory insufficiency, and all were successfully weaned and extubated. In fact, Shikora et al. recently showed that $\dot{V}O_2$ did not increase in a group of patients who could be weaned from ventilatory assist [14]. Hence, it can be assumed that the work of breathing probably accounted for only a small fraction of total body oxygen uptake similar to the value found in healthy normal subjects. In healthy resting subjects the work of breathing only represents about 2% of total oxygen uptake [15]. Such minor differences of $\dot{V}O_2$ are unlikely to be detected when $\dot{V}O_2$ is calculated based on various measurements (Hb, SO_2, PO_2 in arterial and mixed venous blood; CI) which all have inherent errors of about 5%.

A major finding of this study was that despite a slight increase in inert gas dead space SIMV+IPS hardly influenced the $\dot{V}_A/\dot{Q}$ distributions in our patients. In particular, neither the mean $\dot{V}_A/\dot{Q}$ ratio for perfusion nor the dispersion indices log $SD\dot{Q}$ and $SD\dot{V}_A$ were altered. These results differ from previous studies comparing CMV with pure spontaneous ventilation in patients during weaning from mechanical ventilation after elective cardiac surgery [16] or after exacerbation of COPD [11]: while the mean $\dot{V}_A/\dot{Q}$ for perfusion was reported to decrease in both of these studies, a substantial rise of the dispersion indices was found during spontaneous ventilation in the COPD patients [11]. Any differences between our study and the previous ones are probably due to the use of IPS: Beydon et al. recently reported that IPS improved $\dot{V}_A/\dot{Q}$ matching in patients who had difficulties during weaning from CMV [17].

The unchanged perfusion distributions together with the unaltered extrapulmonary factors influencing arterial oxygenation, namely CI and $\dot{V}O_2$ [18], explain why PaO_2 did not change. The increased minute ventilation, a third extrapulmonary factor influencing gas exchange, only accounted for increased dead space and eventually, increased ventilation of high $\dot{V}_A/\dot{Q}$ lung regions, and hence, did not interfere with pulmonary oxygen exchange.

In our patients we did not find any alterations in $PaCO_2$, although physiological dead space increased during SIMV+IPS. In contrast, Wolff and coworkers [3] had observed increased $PaCO_2$ values during SIMV breathing in patients after open heart surgery. These differences are probably due to the use of an IPS in our patients: IPS allowed for increased total minute ventilation because of relatively high spontaneous tidal volumes (about 5–8 ml/kgBW) and, hence, it compensated for the increased "wasted" ventilation yielding unchanged $PaCO_2$.

Inert gas analysis demonstrated that these increased physiological dead space was due to increased inert gas dead space ($\dot{V}_D/\dot{V}_T$ (IG)). These results are in accordance with the data published by Wolff and coworkers reporting increased physiological dead space during SIMV due to increased serial dead space. It has to be noted, however, that in Wolff's study this increased serial dead space was associated with improved efficiency of alveolar CO_2 elimination for the spontaneous breaths. Although in our patients only inert gas dead space was significantly increased during SIMV+IPS, additional effects on the alveolar level cannot be definitely ruled out. Two major reasons have to be considered to explain putative differences between Wolff's study and our data. First, Wolff and coworkers used breath-to-breath analyses based upon PCO_2 measurements which provided means to discriminate between mechanical and spontaneous breaths. In contrast to that, MIGET only allows an overall description of steady state $\dot{V}_A/\dot{Q}$ distributions [5]. Therefore, differences between the two breathing modes during SIMV+IPS could not be detected. Second, apart from the different methodological approach the application of IPS in our patients might explain the conflicting results in the two studies. During IPS breathing the inspiratory flow profile is accompanied by steeper and more pronounced increases in airway pressures which would preferentially direct alveolar ventilation to the lung apex [19]. Together with a reduced tidal volume this phenomenon might account for increased serial dead space and a decreased efficiency of alveolar CO_2 elimination as well.

Argument in favour of the latter is based on the results of MIGET. Because of the finite solubility of acetone, the most soluble gas used for MIGET, inert gas dead space not only represents anatomical dead space, but also includes all lung regions with $\dot{V}_A/\dot{Q}$ ratios above 100. The latter would reflect changes on the alveolar level. The de novo appearance and/or the increase of the ventilation fraction to lung regions with high $\dot{V}_A/\dot{Q}$ ratios ($10 < \dot{V}_A/\dot{Q} < 100$) adjacent to inert gas dead space in three of our patients suggest that beyond any putative effects on serial dead space SIMV+IPS might have induced ventilatory alterations on the alveolar level as well. These results are underscored by the values for the log $SD\dot{V}_A$ in these patients which, in contrast to the unchanged overall mean, was augmented from 1.1 to 1.4 during SIMV+IPS documenting a broader distribution of alveolar ventilation.

The use of MIGET raises another methodological problem: The remaining sum of squares (RSS) for the $\dot{V}_A/\dot{Q}$ distributions during CMV was 4.9 ± 6.9 (mean $\pm$ SD) while it reached 7.3 ± 7.9 during SIMV+IPS, a value which is relatively high though still within the acceptable range [8]. This may be due to the fact that during SIMV+IPS two completely different modes of ventila-

tion are combined which might jeopardize the maintenance of a steady state, one precondition for the applicability of MIGET. Hence, greater variations for the inert gas partial pressures might be anticipated resulting in higher values for RSS.

In summary, in our patients without significant lung pathology SIMV combined with IPS did not induce a clinically significant loss of the efficiency of the ventilatory pattern. In particular, no change was observed for the perfusion distribution nor for the dispersion indices. An increase in dead space ventilation did not result in increased $PaCO_2$ because of increased minute ventilation. Hence, in our patients SIMV+IPS proved to be nearly as efficient as CMV during weaning from postoperative mechanical ventilation. Further investigation is warranted whether this is true in patients during weaning from acute respiratory failure.

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