Effects of carbon dioxide embolism with nitrous oxide in the inspired gas in piglets†


Summary

We have compared cardiorespiratory variables in anaesthetized piglets whose lungs were ventilated with oxygen in nitrous oxide (N₂O group) or nitrogen (N group) after right ventricular carbon dioxide boluses (0.5 or 1 ml kg⁻¹; n = 12) or slow graded injections (n = 6). Boluses affected all variables studied significantly (P < 0.05) except mean systolic arterial pressure. Significant changes in \( P_{\text{CO}_2} \) (\( P = 0.012 \)) and \( P_{\text{A}} \text{O}_2 \) (\( P = 0.048 \)) values were observed in the N₂O group. Changes in \( P_{\text{A}}\text{CO}_2 \) were related to volumes of injected carbon dioxide (\( P = 0.044 \)). Boluses of 1.0 ml kg⁻¹ induced severe circulatory collapse in two piglets in the N₂O group. Slow embolization altered respiratory variables significantly (\( P < 0.001 \)). \( P_{\text{A}}\text{O}_2 \) decreased significantly in the N₂O group (\( P < 0.0001 \)). Mean pulmonary arterial pressure increased significantly over time (\( P = 0.0001 \)) and lasted longer in the N₂O group (\( P < 0.05 \)). Volumes and time required to induce a 50% increase in mean pulmonary arterial pressure differed significantly between groups (\( P < 0.05 \)). We conclude that nitrous oxide worsened the effects of rapid and slow carbon dioxide emboli on cardiopulmonary variables. Rapid carbon dioxide embolism altered respiratory and haemodynamic variables, while slow carbon dioxide embolism changed only respiratory variables. (Br. J. Anaesth. 1996; 76: 428–434)

Key words
Carbon dioxide, embolism. Anaesthetics gases, nitrous oxide. Embolism, carbon dioxide. Pig.

Cases of carbon dioxide embolism with life-threatening consequences have been reported during laparoscopic surgery [1, 2]. As with air embolism, a variety of factors including the volume of the bolus [3, 4], its rate of entry into the vascular compartment and the inspired gas composition [5–8] are likely to determine the consequences of carbon dioxide embolism. In an in-vitro experiment, Nunn [5] noticed that air bubbles enlarged when infused in blood obtained from a patient whose lungs had been ventilated with nitrous oxide–oxygen mixtures. Steffee, Johnson and Eger [9] observed that breathing nitrous oxide resulted in significantly greater and prolonged increases in mean pulmonary artery pressure (PAP) after i.v. injection of carbon dioxide in anaesthetized dogs. These observations [5, 9] suggest that nitrous oxide diffuses from the surrounding blood and alveolar space into the carbon dioxide bubbles as they travel through the venous and pulmonary circulation.

In the present study we have examined the influence of volume and rate of entry into the right ventricle of carbon dioxide emboli, and the role of the inspired gas (nitrous oxide–oxygen vs nitrogen–oxygen) on both haemodynamic state and gas exchange in anaesthetized piglets. Two different experimental conditions were studied: boluses and slow graded infusions of carbon dioxide. These models were intended to mimic either an acute carbon dioxide embolism or a progressive gas embolism, as might be produced by carbon dioxide passing through a venous tear during laparoscopic procedures.

Materials and methods

We studied domestic piglets (Belgian Pietrain) of both sexes (\( n = 18 \)) weighing 23–27 kg after obtaining approval from the Animal Research Committee of Erasme University Hospital, Brussels. Fasting animals were premedicated with atropine 1 mg kg⁻¹ i.m. and anaesthetized with a combination of ketamine 20 mg kg⁻¹ i.m., atropine 0.05 mg kg⁻¹ i.m. and midazolam 0.1 mg kg⁻¹ i.m. [10]. Tracheal intubation was facilitated with administration of pancuronium 0.5 mg kg⁻¹ [11] supplemented with propofol 2–5 mg kg⁻¹ [12]. The lungs were ventilated with a Servo Elema 900B ventilator (Siemens, Solna, Sweden). Anaesthesia was maintained with propofol 25 mg kg⁻¹ h⁻¹ i.v., fentanyl 10 g kg⁻¹ h⁻¹ i.v. and pancuronium 0.4 mg kg⁻¹ h⁻¹ i.v. Fluid infusion of 0.9% NaCl 10 ml kg⁻¹ h⁻¹ was given via a cannula in the external vein of the ear. Inspired gas composition, end-tidal partial pressure of carbon dioxide (\( P_{\text{CO}_2} \)) and

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ventilation rate were monitored continuously using a Datex Capnomac AGM-103 monitor (Datex, Helsinki, Finland). Tidal volume, temperature and pulse oximetry (SpO₂) were monitored using a Datex Cardiomap Ultima SV monitor (Datex, Helsinki, Finland).

The saphenous artery was cannulated for recording of systemic artery pressure and sampling for arterial blood-gas analysis. Both external jugular veins were dissected for the introduction of catheters. A flow-directed thermocatheter (7.5 Fr, Edwards Swan-Ganz, Baxter Healthcare Co, Irvine, CA, USA) was placed in the pulmonary artery for continuous monitoring of PAP and measurements of cardiac output (CO). A second flow-directed thermocatheter (7.5 Fr, Edwards Swan-Ganz, Baxter Healthcare Co, Irvine, CA, USA) and a double-lumen catheter (7 Fr, Deltacath, Becton Dickinson, USA) were introduced into the right ventricle and auricle, respectively, via the opposite external jugular vein. The position of the catheters was confirmed by the presence of an appropriate pressure-wave pattern on the monitor. Systolic arterial pressure (SAP) and PAP were measured continuously using electronic transducers (Uniflow, Baxter Healthcare Co, Uden, Holland) connected to a monitoring system (Sirecust 404-1, Siemens AG, Erlangen, Germany) with zero pressure at the thoracic mid-level as the reference. A 2-MHz Doppler ultrasonic flow transducer (Sonicaid Model D206, Oxford Sonicaid Ltd, Chichester, UK) was positioned above the right heart chambers so that a 10-ml saline injection delivered to the right ventricle would produce an audible change in sound.

A record of the ECG, ultrasonic Doppler signal, \( P_{\text{WCO}_2} \), SAP and PAP was obtained on a six-channel recorder (Model 800, Gould Electronic, Bailleuvilliers, France) for later analysis of these variables. Arterial blood-gas values were determined using an ABL 500 (Radiometer, Copenhagen, Denmark). Measurements of CO were performed in triplicate using a SAT-1 cardiac output computer (American Edwards Lab., Irvine, CA, USA) with 10-ml injections of 0.9 % NaCl via one port of the double-lumen catheter.

The fractional inspired concentration of oxygen \( (F_{\text{I,O}_2}) \) was set at 0.32-0.35 and ventilation adjusted to maintain the arterial partial pressure of carbon dioxide \( (P_{\text{ACO}_2}) \) at 4.5-6 kPa. The animal’s core temperature was monitored via a pulmonary artery thermocouple and maintained at 38–39.5 °C throughout the experiment. Acquisition of baseline data began after a stabilization period of at least 30 min after completion of the surgical procedure. Variables monitored included \( \text{SpO}_2, \text{Sa}_{O_2}, P_{A_{O_2}}, P_{W_{CO_2}}, P_{A_{CO_2}}, \) heart rate (HR), mean SAP, mean PAP and CO.

**STUDY 1: RAPID CARBON DIOXIDE BOLUS INJECTION**

Twelve piglets were allocated randomly in one of two groups to undergo ventilation with either oxygen–nitrogen (N₂ group, \( n = 6 \)) or oxygen–nitrous oxide (N₂O group, \( n = 6 \)) mixture. The carbon dioxide gas was delivered from a container with outflow pressure and volume regulated by a manometer connected to a 50-ml leak-free syringe. A three-way stopcock enabled the syringe to be connected between the outflow tubing of the carbon dioxide container and the distal port of the 7.5-French gauge thermocatheter for embolization. Before embolization, the syringe was flushed five times with carbon dioxide. The injection was then performed manually over a 30-s period. Animals were injected successively with two carbon dioxide volumes (0.5 and 1 ml kg⁻¹) following a crossover design.

**STUDY 2: SLOW GRADED INJECTIONS**

A manually regulated flowmeter (Aalborg Instruments, Monsey, NY, USA) mounted between the manometer of the carbon dioxide container and the distal port of the 7.5-French gauge flow-directed catheter was used to adjust the volume infused per minute. Piglets were embolized with a gradual increase in carbon dioxide flow while the lungs were ventilated with either oxygen–nitrogen (N₂ group, \( n = 6 \)) or oxygen–nitrous oxide (N₂O group, \( n = 6 \)). The infusion was discontinued when mean PAP increased by 50 % from baseline. Initially, piglets were embolized with a carbon dioxide flow of 7.5 ml min⁻¹. At each increment, the embolized carbon dioxide volumes were increased by 50 % from baseline and the infusion was maintained for 3 min at a constant pressure of 14.7 psi. A calibration chart allowed determination of the infused carbon dioxide flows according to readings wherein 10, 20, 30, 40, 50, 60, 70, 80, 90, 100 corresponded to 7.5, 12.6, 17.5, 24, 31, 39.4, 49, 58.4, 69.5, 82.1 ml min⁻¹, respectively.

Between the experimental sequences, the blood-gas variables studied were allowed to return to control values. Ventilation with different gas mixtures was separated by 1 h of ventilation with 100 % oxygen to ensure adequate washout of nitrous oxide and nitrogen. Thereafter, a 30 min-period of washing with either nitrogen or nitrous oxide was allowed before the next experiment was performed.

**STATISTICAL ANALYSIS**

**Study 1**

All variables were analysed using a three-way (time, nitrous oxide \( vs \) nitrogen, 0.5 \( vs \) 1 ml kg⁻¹) analysis of variance (ANOVA) with one-way (time) for repeated measurements. The Newman–Keul test was used for multiple comparisons where appropriate.

**Study 2**

A two-way (time and nitrous oxide \( vs \) nitrogen) ANOVA with one-way for repeated measurements (time) was carried out. The Newman–Keul test was used for multiple comparisons when applicable. The volume of carbon dioxide required to induce an increase of 50 % in mean PAP, duration of injection and the time to recovery of basal \( P_{W_{CO_2}} \) values were compared between groups using the Wilcoxon test for paired data [13].
Data are expressed as mean (SD). Changes were considered statistically significant at $P < 0.05$.

**Results**

Baseline values of the variables after anaesthesia are shown in table 1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean</th>
<th>Range</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beat min$^{-1}$)</td>
<td>119</td>
<td>110–130</td>
<td>6</td>
</tr>
<tr>
<td>Mean SAP (mm Hg)</td>
<td>96</td>
<td>83–110</td>
<td>10</td>
</tr>
<tr>
<td>Mean PAP (mm Hg)</td>
<td>20</td>
<td>17–24</td>
<td>2</td>
</tr>
<tr>
<td>CO (litre min$^{-1}$)</td>
<td>3080</td>
<td>2800–3500</td>
<td>240</td>
</tr>
<tr>
<td>$S_{P_{CO}}$ (%)</td>
<td>96</td>
<td>95–97</td>
<td>1</td>
</tr>
<tr>
<td>$S_{O_{a}}$ (%)</td>
<td>97</td>
<td>96–97</td>
<td>1.5</td>
</tr>
<tr>
<td>$P_{a_{CO}}$ (kPa)</td>
<td>17.4</td>
<td>15.2–18.4</td>
<td>1.1</td>
</tr>
<tr>
<td>$P_{a_{CO}}$ (kPa)</td>
<td>5</td>
<td>4.6–5.3</td>
<td>0.3</td>
</tr>
<tr>
<td>$P_{a_{CO}}$ (kPa)</td>
<td>5.3</td>
<td>5.3–5.6</td>
<td>0.1</td>
</tr>
</tbody>
</table>

**Changes in gas exchange**

After embolization, $P_{e_{CO}}$ decreased significantly over time ($P = 0.001, \text{ANOVA}$) and these reductions were more severe in the $N_2O$ group than in the $N_2$ group ($P = 0.012, \text{ANOVA}$). However, there were no statistically significant differences between the two volumes of carbon dioxide boluses (0.5 vs 1 ml kg$^{-1}$).

At $T + 30$ s, the changes in $P_{e_{CO}}$ were decreased significantly in all groups ($P < 0.05, \text{Newman-Keuls test}$) (fig. 1). In three piglets in the $N_2$ group, analysis of recording charts showed an increase in $P_{e_{CO}}$ up to a mean of 0.3 kPa after embolization with 0.5 ml kg$^{-1}$. These initial increases lasted about 20 s and were followed by typical post-embolic $P_{e_{CO}}$ decreases (fig. 2).

Changes in $S_{P_{O}}$ and $S_{O_{a}}$ were time related ($P = 0.001, \text{ANOVA}$). Decreases in $S_{P_{O}}$ and $S_{O_{a}}$ values were greatest at $T + 0.5$ min ($P < 0.05, \text{Newman-Keuls test}$) compared with other times. $S_{P_{O}}$ decreased by 5 % and 9 % after 0.5 and 1 ml kg$^{-1}$ boluses, respectively, but only in the $N_2O$ groups (fig. 3). $S_{O_{a}}$ and $S_{P_{O}}$ did not differ significantly with different inhaled gas mixtures and volumes of carbon dioxide boluses.

Alterations in $P_{a_{O}}$ were significant in relation to time ($P = 0.0001$) and were more severe in the $N_2O$ than in the $N_2$ group ($P = 0.048, \text{ANOVA}$). In the $N_2$ and $N_2O$ groups, $P_{a_{O}}$ decreased to 81 % and 71 % from baseline after 0.5 ml kg$^{-1}$ carbon dioxide boluses, while after embolization with 1 ml kg$^{-1}$ decreases to 72 % and 59 % were observed. With 1 ml kg$^{-1}$ carbon dioxide boluses, decreased $P_{a_{O}}$ persisted for 30 min in the $N_2O$ group.

Differences in $P_{a_{CO}}$ were significant in relation to time ($P = 0.0001, \text{ANOVA}$) and in volumes of carbon dioxide boluses ($P = 0.044, \text{ANOVA}$). In both groups, $P_{a_{CO}}$ values returned to baseline 30 min after embolization and no significant difference was observed in relation to gas mixture.
Effects of N₂O on CO₂ embolism in piglets

Cardiovascular changes

HR changed significantly over the post-embolization periods (P = 0.029, ANOVA). At T + 5 min and T + 10 min, these differences were significant (P < 0.05, Newman–Keuls test) (fig. 4) compared with other periods. No significant changes were observed in MAP values.

Mean PAP changed significantly in relation to time (P < 0.0001, ANOVA). These changes were observed at T + 30 s and T + 5 min (P < 0.05, Newman–Keuls test).

Changes in CO were observed over time (P < 0.0001, ANOVA) and differences were significant at T + 30 s and T + 5 min (P < 0.05, Newman–Keuls test). In the N₂O group, two piglets suffered cardiovascular collapse after embolization with 1 ml kg⁻¹ carbon dioxide volumes.

There were no statistically significant differences in relation to the gas mixture used for ventilation and to the two volumes of carbon dioxide boluses (table 2).

STUDY 2

An increase in mean PAP of 50 % was used as the reference for completion of embolization. Compared with the N₂O group, the N₂ group required higher cumulative infused carbon dioxide volumes (247 (189) ml vs 72 (60) ml) and a longer delay to reach a 50 % increase in mean PAP (15.5 (7.6) min vs 6.5 (3) min, P < 0.05; paired Wilcoxon test) (fig. 5).

Changes in gas exchange

After embolization, PECO₂ changed significantly with time (P = 0.001, ANOVA). At T + 30 s, PECO₂ showed significant decreases of 20 % in the N₂ group vs 27 % in the N₂O group (P < 0.05, Newman–Keuls test). The time to regain baseline PECO₂ values was longer in the N₂ group than in the N₂O group (11.5 (7.6) min vs 7 (4) min, P < 0.05: unpaired Wilcoxon’s test). The chart recording of PECO₂ during slow embolization showed a plateau profile, despite the stepwise variation in the delivery of carbon dioxide flows used for embolization (fig. 6).

We found significant post-embolic decreases in SPO₂ and SGO₂ over time (P < 0.0001) in both groups and the differences were significant at T + 30 s (P < 0.05, Newman–Keuls test). Values of PAO₂ decreased significantly (P < 0.0001, ANOVA) by 70 % and 63 % from baseline in the N₂ and N₂O groups (P < 0.05, Newman–Keuls test), respectively, at T + 30 s.

Post-embolic increases of 24 % in PAO₂ (P < 0.0001, ANOVA) were noted at T + 30 s in both groups.

Cardiovascular changes

There were no statistically significant changes in HR, mean SAP and CO with time (table 3). Only mean PAP values changed significantly over time (P = 0.01, ANOVA). At T + 30 s and T + 5 min, mean PAP values were significantly greater than those recorded at other times (P < 0.05, Newman–Keuls test). Thirty minutes after termination of

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Table 2: Statistical significance of changes in the variables studied after rapid carbon dioxide embolization (n = 6 in each group)

<table>
<thead>
<tr>
<th>Time</th>
<th>N2 vs N2O</th>
<th>0.5 vs 1 ml kg⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>PECO₂</td>
<td>0.001</td>
<td>0.012</td>
</tr>
<tr>
<td>SPO₂</td>
<td>0.0001</td>
<td>ns</td>
</tr>
<tr>
<td>SGO₂</td>
<td>0.0001</td>
<td>ns</td>
</tr>
<tr>
<td>PAO₂</td>
<td>0.0001</td>
<td>0.48</td>
</tr>
<tr>
<td>PECO₂</td>
<td>0.0001</td>
<td>0.044</td>
</tr>
<tr>
<td>HR</td>
<td>0.04</td>
<td>ns</td>
</tr>
<tr>
<td>Mean SAP</td>
<td>ns</td>
<td>ns</td>
</tr>
<tr>
<td>Mean PAP</td>
<td>0.01</td>
<td>ns</td>
</tr>
<tr>
<td>CO</td>
<td>0.001</td>
<td>ns</td>
</tr>
</tbody>
</table>

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Figure 3: Evolution of SPO₂ from baseline values against time in the N₂ groups after 0.5 ml kg⁻¹ and 1 ml kg⁻¹, and in the N₂O groups after 0.5 ml kg⁻¹ and 1 ml kg⁻¹ against time. Note there are increases only in the N₂O groups compared with baseline. All values are mean (n = 6 in each group).

Figure 4: Changes in heart rate (HR) from baselines values in the N₂ groups after 0.5 ml kg⁻¹ and 1 ml kg⁻¹, and in the N₂O groups after 0.5 ml kg⁻¹ and 1 ml kg⁻¹ against time. Note there are increases only in the N₂O groups compared with baseline. All values are mean (n = 6 in each group).
embolization, residual mean PAP increases were still significantly higher (21% vs 10% from baseline values, *P < 0.05) in the N₂O group than in the N₂ group (fig. 5).

**Discussion**

We have shown that after rapid and progressive carbon dioxide embolism in piglets, cardiorespiratory alterations were worsened by the presence of nitrous oxide in the gas mixture used for mechanical ventilation. These findings confirm the results of previous studies which demonstrated that
nitrous oxide amplifies the effects of venous air [6–8] or carbon dioxide embolism and prolongs the delay to recover from the physiological insults [9]. In agreement with previous studies [7, 9], nitrous oxide reduced the volume required to induce predetermined changes.

Continuous \( P_{\text{CO}} \) monitoring showed some particular trends in addition to the commonly observed sharp decrease after gas embolism. First, abrupt and transient increases in \( P_{\text{CO}} \) were observed after rapid boluses of low volume. This observation confirms the biphasic trend of \( P_{\text{CO}} \) recordings reported previously after carbon dioxide embolism during laparoscopic surgery [2, 14, 15]. Second, while mean PAP continued to increase during progressive embolization, \( P_{\text{CO}} \) values decreased steadily and demonstrated a plateau profile. Moreover, \( P_{\text{CO}} \) values returned to baseline earlier than those of mean PAP and reached a higher level than baseline after slow embolization in the N2 group. These variations in response differed markedly compared with those reported previously after air embolism [16]. Therefore, it must be emphasized that during progressive carbon dioxide embolism, \( P_{\text{CO}} \) monitoring may not allow either early and reliable detection of carbon dioxide embolism or provide relevant quantitative information about the size of the carbon dioxide emboli. The other non-invasive monitorings, including HR and \( S_{\text{O}_2} \), demonstrated changes in values and trends similar to those reported after laparoscopic carbon dioxide embolism.

For invasive monitoring, only \( P_{\text{aO}} \) changed significantly in relation to volume of carbon dioxide boluses and the inspired gas mixture during rapid carbon dioxide embolization. After both rapid and progressive infusions, an increase in mean PAP was observed constantly, immediately after injection of carbon dioxide. In agreement with previous studies on the effects of air [6, 8] and carbon dioxide emboli [9], the rate of increase in mean PAP was related closely to the rate of injection, the volume of the carbon dioxide bolus and the nature of the gases used for ventilation. During slow carbon dioxide embolization, although mean PAP was increased by 50% from baseline, no significant changes in CO were observed. In the presence of N2O, the trends in mean PAP showed higher values without any significant differences. However, its residual values were significantly higher in the piglets whose lungs were ventilated with oxygen–nitrous oxide. This finding confirms the known sensitivity of mean PAP monitoring in detecting gas embolism [16].

Although this model is not strictly similar to the clinical situation, the right intraventricular site for injection was chosen in an attempt to create a better quantitative experimental design for carbon dioxide embolization to observe the relationship between different patterns (volume and rate) and effects. It is well known that gas bubbles introduced into the jugular vein, superior vena cava and right atrium move downwards in some parts of the cardiac cycle and upwards in others [17], and that some fraction of the injected carbon dioxide is absorbed before reaching the pulmonary vasculature.

The haemodynamic changes induced by slow embolization differ from those observed with carbon dioxide boluses, where decreases in CO may be caused mainly by a “bubble-lock” in the right side of the heart. This observation suggests that mechanical block of portions of the pulmonary circulation is only partial but sufficient to contribute to alterations in pulmonary gas exchange. As the arteriole is the major determinant of PAP, one can speculate that an arteriolar effect may be reflected in these changes in PAP [18]. The present study suggests that the post-embolic vascular change may be greater and more prolonged in the presence of nitrous oxide than nitrogen. However, the magnitude of the post-embolic disturbances, may be determined by the combined effect of the size and number of carbon dioxide bubbles. First, a low volume embolism may allow carbon dioxide microbubbles to reach the alveolar capillaries with high alveolar elimination of carbon dioxide and increase in \( P_{\text{CO}} \). This is in agreement with previous reports of an abrupt but transient increase followed by a progressive decrease during carbon dioxide embolism during laparoscopic surgery [2, 14, 15]. Second, larger carbon dioxide bubbles may induce widespread arteriolar block and pulmonary blood flow redistribution resulting in ventilation-to-perfusion (\( V/\dot{Q} \)) ratio alterations including an increased \( V/\dot{Q} \) ratio in the embolized lung [19] associated with opening of intrapulmonary arteriovenous shunts and decreased \( V/\dot{Q} \) ratio in the non-embolized area. The majority of air bubbles ejected from the right ventricle are likely to reach the non-dependent branches of the pulmonary arteries [20, 21]. In terms of regional ventilation-to-perfusion distribution, this implies that inhalation of nitrous oxide would worsen the shift of blood flow to dependent regions with a larger pulmonary shunt and greater deadspace resulting in greater decreases in \( P_{\text{aO}} \) and \( P_{\text{CO}} \) values during rapid embolization.

When differences in solubility and diffusion are considered, the present study agrees with the principle that inhalation of nitrous oxide aggravates existing air and carbon dioxide embolism [22]. The greater partial pressure of carbon dioxide in bubbles compared with surrounding blood and the alveoli results in a direct transfer of carbon dioxide from bubbles to blood with post-embolic increases in \( P_{\text{CO}} \) and higher elimination to alveolar space producing an initial increase in \( P_{\text{CO}} \). Furthermore, the progressive return to baseline values observed in the present study is in agreement with Kunkler and King [23] who demonstrated that carbon dioxide bubbles equilibrated rapidly with the surrounding blood gases and revealed large amounts of other gases, by analysis performed within few seconds on undissolved emboli. Second, considering the greater physiological changes observed in the presence of nitrous oxide, transfer of nitrous oxide into carbon dioxide bubbles may be speculated. The Fick equation to spherical bubble flow in the bloodstream suggests that the rate of gas diffusion is directly proportional to Krogh’s coefficient (the product of solubility and diffusion) and the partial pressure gradient of diffusion for that gas [8]. The higher Krogh’s coefficient for carbon dioxide than for
nitrous oxide and nitrogen suggests that carbon dioxide outflow should be faster than inflow of nitrous oxide and nitrogen for a given partial pressure. Therefore, the magnitude of changes observed during nitrous oxide ventilation may be associated with initial entry of nitrous oxide into the carbon dioxide bubbles until equilibration with the surrounding blood and to the resultant longer lifespan for bubbles, allowing a larger number to induce a cumulative effect. In contrast with nitrous oxide, diffusion of nitrogen into carbon dioxide bubbles would be slower, therefore carbon dioxide bubbles might dissipate more rapidly after embolization.

We conclude that during procedures at risk of carbon dioxide embolism, significant gas exchange alterations should be considered as clinically relevant even in the absence of haemodynamic disturbances. Anaesthetists should keep in mind the study of Eger and Saidman [24] demonstrating that the relationship between nitrous oxide blood concentration and the volume increase of air bubbles is exponential and that for a concentration of between 50% to 75% a two- to four-fold increase may be attained. The present study suggests additional limitations about routine $P_{\text{ECO}_2}$ monitoring during laparoscopic surgery. The assumptions that $P_{\text{ECO}_2}$ monitoring may reliably detect carbon dioxide embolism during laparoscopy with carbon dioxide insufflation should be reconsidered. Because of the increasing number of surgical laparoscopic procedures which may potentially lead to severe gas embolism [1, 2], the presence of Doppler monitoring should be mandatory, as it has been found to be the most sensitive and specific for detection of gas embolism [16, 25].

References

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