De-airing of a Cardiothoracic Wound Cavity Model With Carbon Dioxide: Theory and Comparison of a Gas Diffuser With Conventional Tubes

Mikael Persson, MSc, and Jan van der Linden, MD, PhD

Objectives: To compare the efficiency of a new gas diffuser with conventional tubes for carbon dioxide (CO_2) de-airing of a cardiothoracic wound cavity model, and to analyze how insufflation flow, outflow velocity, and diffusion affect de-airing.

Design: Technical study _in vitro._

Setting: A nonventilated room at a University Hospital.

Interventions: De-airing by CO_2 insufflation via 3 methods was studied in a symmetric cardiothoracic wound model.

Measurements and Main Results: The studied insufflation devices were 2 open-ended tubes with an inner diameter of 2.5 mm and ¼-in (6.35 mm), respectively, and a gas diffuser (ie, a 2.5-mm tube with a diffuser at the end). CO_2 flows of 2.5, 5, 7.5, and 10 L/min were used. De-airing was assessed by measurement of remaining air content in a set of systematically distributed measuring points in the model. Three-, 2-, and 1-way analysis of variance all revealed significant interaction of device, flow, and depth on air content ($p <$ 0.001). With tubes, the mean air content was 18% to 96% at the studied flows. With the gas diffuser, the mean air content in the cavity was below 0.2% at flows of 5 to 10 L/min. There was an exponential relation between calculated outflow velocity and air content. At a flow of 2.5 L/min, diffusion attenuated de-airing.

Conclusion: These data imply that de-airing of a cardiothoracic wound by CO_2 insufflation depends on flow and outflow velocity. To compensate for diffusion with ambient air, the CO_2 flow should be $\geq$ 5 L/min, and the outflow velocity should be about 0.1 m/s or less to avoid turbulence in the wound. This is only attainable with a gas diffuser.

KEY WORDS: carbon dioxide, diffuser, insufflation device, air embolism, cardiac surgery

RESIDUAL INTRACARDIAC air is an important source of cerebral and myocardial microembolization during cardiac operations.[1-5] Arterial air embolism may cause cerebral injury and myocardial dysfunction/arrhythmia.[2,6-11] Despite standard surgical de-airing techniques, such as squeezing and shaking of the heart, large numbers of air emboli still occur.[3,12] Carbon dioxide (CO_2) insufflated into the chest wound cavity may improve the de-airing. Because CO_2 is $\geq$25 times more soluble in tissue and blood than air,[13,14] arterial CO_2 emboli are much better tolerated than air emboli.[2,7-9,15,16] Furthermore, CO_2 is 50% heavier than air, which facilitates air displacement in a wound cavity.

Although CO_2 has been used for de-airing in cardiac surgery since the 1950s,[17] the method has not become widespread. The conventional device for CO_2 insufflation is a thin open-ended tube, but several studies point to its inability to provide efficient air displacement.[13,18,19] The authors have therefore designed a new insufflation device, which consists of a thin tube with a gas diffuser at the end. Clinical and experimental studies in this field have not thoroughly investigated how CO_2 flow influences air displacement. Furthermore, the aspects of outflow velocity and gas exchange by diffusion have not hitherto been considered.

The first aim of this study was to compare the de-airing efficiency of the gas diffuser with that of conventional open-ended tubes of different diameters in a wound cavity model. The second aim was to analyze how flow, outflow velocity, and diffusion of CO_2 affect the air content in the model.

METHODS

The 3 tested insufflation devices were 2 open-ended tubes with an inner diameter of 2.5 mm and ¼-in (6.35 mm), respectively, and the new patented gas diffuser (Cardia Innovation AB, Stockholm, Sweden). The latter consists of a cylindrical diffuser, which is made of soft polyurethane foam with open cells and has a diameter of 18 mm and a length of 14 mm. It is attached to a tube with an inner diameter of 2.5 mm via a plastic disc at the proximal area of the diffuser.

The air displacement efficiency of the CO_2 insufflation devices was tested in a cylindrical model (diameter 16 cm, depth 8 cm, Fig 1), which was based on intraoperative measurements of the open chest wound cavity of 5 adults undergoing cardiac surgery through a standard sternotomy. The mean depth of the studied cavities (average of cranial and caudal depths) during cardiopulmonary bypass with an empty heart was 7 cm (range 6.5-7.5 cm). The corresponding mean length (midline) and width of the wound opening were 19 cm (range 17-20 cm) and 10 cm (range 9-12 cm), respectively. The shape of the opening of the cavities was elliptical. The area of the circular opening of the model is 200 cm^2, which corresponds to an elliptical opening with a length of 21 cm and a width of 12 cm. The slightly oversized opening of the model maximized the influence of diffusion. The model is similar to the one used by Selman et al.[18]

The study was performed during controlled static conditions (ie, without atmospheric movements) to reveal the true flow characteristics of the insufflation devices and to isolate the fundamental gas dynamics that are involved in CO_2 de-airing. The ventilation system of the study room was shut off. The temperature was around 20°C in the room throughout the experiment.

Each insufflation device was fixed to the brim of the model with laboratory stands and with the help of a supporting metal rod. The devices were pointed toward the center of the model, with the orifice positioned 4 cm inside and 2 cm below the brim (Fig 1). The CO_2 was delivered from a medical gas cylinder (AGA AB, Stockholm, Sweden) via a flow regulator with a flowmeter.

The air content inside the model was analyzed at CO_2 flows of 2.5, 5, 7.5, and 10 L/min by sampling the oxygen (O_2) concentration. The air content (%Air) is given by the following equation:

From the Department of Cardiothoracic Surgery and Anesthesiology, Huddinge University Hospital; and Division of Medical Engineering, Department of Medical Laboratory Science and Technology, Karolinska Institute, Stockholm, Sweden.

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Address reprint requests to Jan van der Linden, MD, PhD, Department of Cardiothoracic Surgery and Anesthesiology, Huddinge University Hospital, SE-141 86, Stockholm, Sweden. E-mail: jan.vanderlinden@thsurg.hs.sll.se

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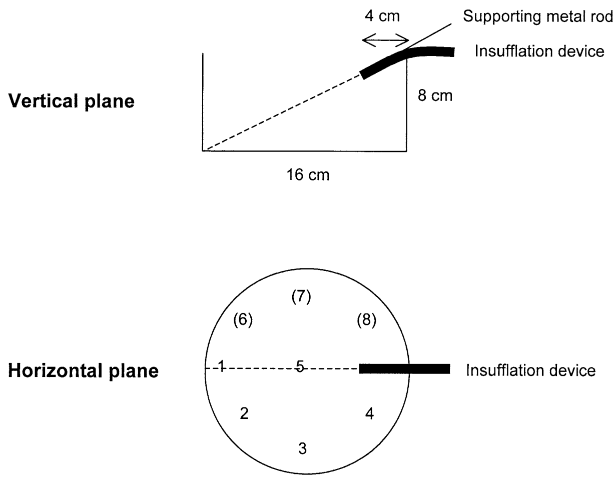

Fig 1. Wound cavity model, position of insufflation devices and horizontal measuring positions No. 1–5. Because of symmetry positions, No. 6–8 are represented by measurements at position No. 2–4. Position No. 5 is in the center of the model and coincides with the direction of the CO_2 insufflation at half the depth of the model. All other positions are located 2 cm inside the wall of the model separated by a horizontal angle of 45°. The dashed line marks the direction of the CO_2 insufflation.

$$\%\text{Air} = \frac{\%O_2}{\%O_2(\text{ref})} \cdot 100$$

where $\%O_2$ is the measured O_2 concentration and $\%O_2$(ref) is the normal O_2 concentration in atmospheric air (20.95% near sea level).[20] The O_2 concentration inside the model was analyzed by measurements at 5 horizontal positions (No. 1-5, Fig 1) at every second centimeter of the depth below the opening. Four of the horizontal measuring positions (No. 1-4) were located 2 cm inside the wall of the model, separated by a 45° angle, and 1 measuring position was located in the center (No. 5). Because of symmetry, position No. 6 to 8 were represented by measurements at No. 2 to 4.

A stable O_2 concentration was considered to be present when values were fluctuating around a constant value over a period of 30 seconds when the O_2 concentration was recorded 10 times in succession, once every 5 seconds ($n = 10$). Before changing CO_2 flow or device, the insufflation was shut off and the remaining CO_2 was removed with a standard vacuum cleaner when air movements around the model were left to settle for one minute.

The impact of gas diffusion on the air content in the model was studied by continuously measuring the air content at 0, 2, 4, 6, and 8 cm depth, respectively, in the middle of the model (position No. 5, Fig 1). Initially, the model was completely filled with CO_2. To reach an air content near 0% in the whole cavity, the opening of the model was covered with 2 plastic plates during CO_2 insufflation with the gas diffuser. The insufflation was discontinued, and the plates were removed horizontally outward at the start of the measurements, which then continued for 10 minutes.

The CO_2 flow was measured with a back-pressure-compensated oxygen flowmeter (AGA AB) because a flowmeter for medical CO_2 was unavailable at the time of the study. The O_2 reading scale was adjusted for CO_2 by a universal flowmeter (ABB/Fisher & Porter, Göttingen, Germany) because of the higher density of CO_2 gas. The universal flowmeter consisted of a measuring tube (FP ¼-16 G-5/81) with a spherical stainless steal float (SS-14). The universal flowmeter was not used for measurements in the study because of its lack of back-pressure compensation. During calibration, this problem was avoided by measuring the CO_2 outflow at the distal end of the insufflation device. The reading scale of the universal flowmeter was calculated for the used gas (medical CO_2, AGA AB) at 20°C and at 760 mmHg with a computer program (FlowSelect version 2.0, ABB/Fisher & Porter). The CO_2 gas was room temperature at the calibration site.

The O_2 concentration was measured with a heated ceramic O_2 sensor (CheckMate 9900; PBI Dansensor, Ringsted, Denmark), which has an accuracy of 1% of the measured value in the range 0.0001% to 100% O_2. The response time of the O_2 sensor is <2 seconds, and the sampling volume is <2 mL. The sampling probe was a 1.5-mm thick Teflon tube (Habia Teknoflour AB, Knivsta, Sweden) that was fixed with a vertical metal rod. It was held in place with a laboratory stand. The O_2 instrument was connected to a personal computer that recorded the O_2 concentration every 5 seconds.

To study the relation between the outflow velocity of the CO_2 and the remaining air content in the model, the studied CO_2 flows were converted to the corresponding calculated outflow velocities for each of the insufflation devices. The mean outflow velocity was calculated by dividing the CO_2 flow with the dispersing area of the insufflation device. The dispersing area of an open-ended tube corresponds to its inner cross-sectional area, πr^2, and that of the diffuser is equal to its dispersing surface, $\pi r^2 + 2\pi rl$, where r is radius and l is length. The mean air content ($n = 80$) at half the depth of the model was plotted versus the calculated outflow velocity (m/s) of the 3 insufflation devices at CO_2 flows of 2.5, 5, 7.5, and 10 L/min, giving a total of 12 points.

Differences were considered statistically significant if $p < 0.05$. One-, 2-, and 3-way analysis of variance (ANOVA), Tukey Honestly Significant Difference test, Student *t* test, and Mann-Whitney *U* test were used whenever appropriate. Curve fitting by nonlinear regression was performed with the computer software NLREG version 5.3 (Brentwood, TN).

RESULTS

Four independent measurable variables were studied: CO_2 flow, gas-dispersing area of the insufflation device, and horizontal and vertical position in the model. The dependent variable was air content, expressed as a percentage of the gas mixture present in the model. Fig 2 shows the remaining air content inside the model resulting from CO_2 insufflation with the various devices at CO_2 flows of 2.5, 5, 7.5, and 10 L/min, respectively. Each mean value of air content at each depth is based on 80 values, 10 per horizontal measuring position. With the 2.5-mm tube (Fig 2A), the mean air content remained between 88% and 96% at all CO_2 flows and at all depths of the cavity. The ¼-in tube (Fig 2B) produced a mean air content between 18.2% and 86% at the studied CO_2 flows and depths. The gas diffuser (Fig 2C) produced a striking drop in air content compared with the tubes. With the gas diffuser, the air displacement was statistically most efficient at a CO_2 flow of 7.5 L/min at a depth of 4 cm and deeper ($p < 0.001$, *t* test). At 7.5 L/min, the air content was 0.30% ± 0.10% (mean ± standard deviation) at 2 cm depth, 0.16% ± 0.02% at 4 cm, 0.15% ± 0.01% at 6 cm, and 0.15% ± 0.01% at 8 cm. Similar low mean air contents were attained at 5 and 10 L/min, 0.65% and 0.33% at 2 cm depth, 0.22% and 0.18% at 4 cm, 0.19% and 0.17% at 6 cm, and 0.18% and 0.17% at 8 cm, respectively. With a CO_2 flow of 2.5 L/min, the air content was significantly higher at all depths ($p < 0.001$) with mean values of 7.1%, 2.0%, 1.7%, and 1.7% at a depth of 2, 4, 6, and 8 cm, respectively. At 0-cm depth (the level of the opening of the

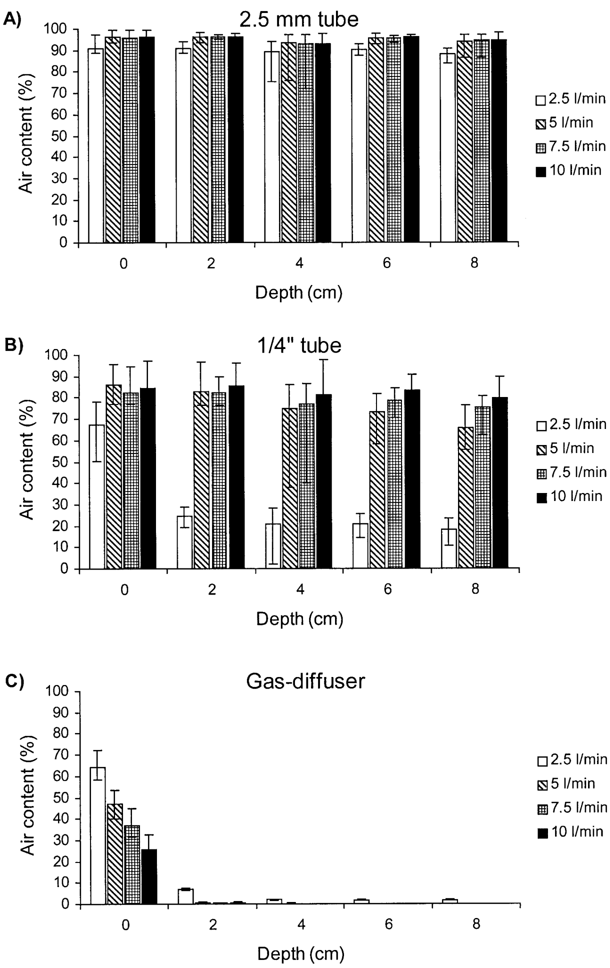

Fig 2. The diagrams show the air content in % (mean and range, n = 80; n = 10 for each position No. 1–8) at each depth inside the model resulting from CO_2 insufflation with (A) a 2.5-mm tube, (B) a 1/4-in (6.35 mm) tube, and (C) a gas diffuser at CO_2 flows of 2.5, 5, 7.5, and 10 L/min.

model), the air content was much higher than at other depths, with the air content inversely related to CO_2 flow.

With 1-way ANOVA, the effect of insufflation device on air content was significant ($p < 0.001$). The following Tukey HSD test showed that all the devices were significantly different from each other ($p < 0.001$). With 2-way factorial ANOVA, the interaction device $\times$ CO_2 flow was significant ($p < 0.001$). Three-way interaction and all 2-way interactions of device, CO_2 flow, and depth were tested with 3-way factorial ANOVA. The three-way and all 2-way interactions were significant ($p < 0.001$).

Fig 3A shows the mean air contents (n = 10) in the vertical plane that include positions No. 1 and 5. Fig 3B depicts the mean air contents (n = 10) in the horizontal plane, at half the depth of the model, which includes the positions No. 1 to 8. The values correspond to a CO_2 flow of 7.5 L/min. Both Fig 3A and B show the superiority of the gas diffuser for de-airing of the model, with an air content close to 0% at all measured positions at a depth of 2 cm or deeper. The tubes produced a significantly lower air content in the CO_2 jet, measured at the center of the model, than at adjacent measuring positions ($p < 0.01$, Mann-Whitney *U* test).

In Fig 4, the mean air content (n = 80) at half the depth of the model was plotted versus the calculated outflow velocity (m/s) of the 3 devices at the 4 different CO_2 flows (2.5-10 L/min). The curve is an exponential regression, air content (y) as a function of the calculated outflow velocity (x), $y = 94.1*(1\text{-}e^{-x/2.56})$, $R^2 = 0.97$. All 4 values of air content for the gas diffuser and the corresponding outflow velocities were all close to zero. With the gas diffuser at a CO_2 flow of 2.5, 5, 7.5, and 10 L/min, the mean air content was 2.0%, 0.22%, 0.16%, and 0.18%, respectively. The corresponding calculated outflow velocities of the gas diffuser were 0.04, 0.08, 0.12, and 0.16 m/s. At these flows, the corresponding calculated outflow velocities for the 2.5-mm tube and the ¼-in tube varied between 1.3 and 34 m/s.

Fig 5 shows the effect of diffusion. The curves show the air content at 0, 2, 4, 6, and 8 cm depth in the middle of the model (position No. 5) during the first 10 minutes after discontinuation of CO_2 insufflation when the cavity, initially completely filled with CO_2, was exposed to the surrounding air. The diffusion flux was highest near the opening of the model (depth 0 cm) during the initial low air content inside the cavity. Thereafter, the diffusion flux decreased with time as the air content inside the cavity increased. The air content was almost 100% in the whole model after 10 minutes.

DISCUSSION

The main component of air, nitrogen, dissolves poorly in blood and tissue. Experimental animal studies have shown that arterial embolization of air to the brain and the heart may not only cause cerebral and myocardial dysfunction but may also lead to convulsions, infarctions, ventricular fibrillation, and increased mortality.[2,7-9,15,16] Air emboli may obstruct vessels and cause tissue ischemia, damage endothelial cells, and initiate other thromboinflammatory effects.[21,22] Even obstruction of cerebral arterioles by air microbubbles (25 μL) for less than 30 seconds may still disrupt brain function.[10,23] Massive air embolism (>20 mL) is an infrequent but well-documented risk of cardiopulmonary bypass (CPB).[24,25] A recent study by Borger et al[11] has shown neuropsychologic impairment after coronary bypass surgery as an effect of air microemboli during perfusionist interventions. In contrast, animal studies have shown that injection of carbon dioxide is much better tolerated than air.[2,7-9,15,16]

Intracardiac air is frequently observed after termination of CPB in patients undergoing cardiac surgery despite systematic use of available surgical de-airing techniques. In fact, new episodes of air bubbles are even noticed in the heart up to 20 minutes after weaning from CPB.[3,5,11,12] The main cause seems to be air from the wound cavity that is trapped in the highest parts of the heart and great vessel (ie, pulmonary veins, superior part of the left atrium, the left ventricular apex, the left atrial appendage, and the right coronary sinus).[4] Trapped air in these pockets is only mobilized when the heart is ejecting blood, especially during and early after weaning from CPB.[3,5] Usual surgical measures to prevent air embolism during cardiac surgery include atrial venting, aortic vent suction, Trendelburg

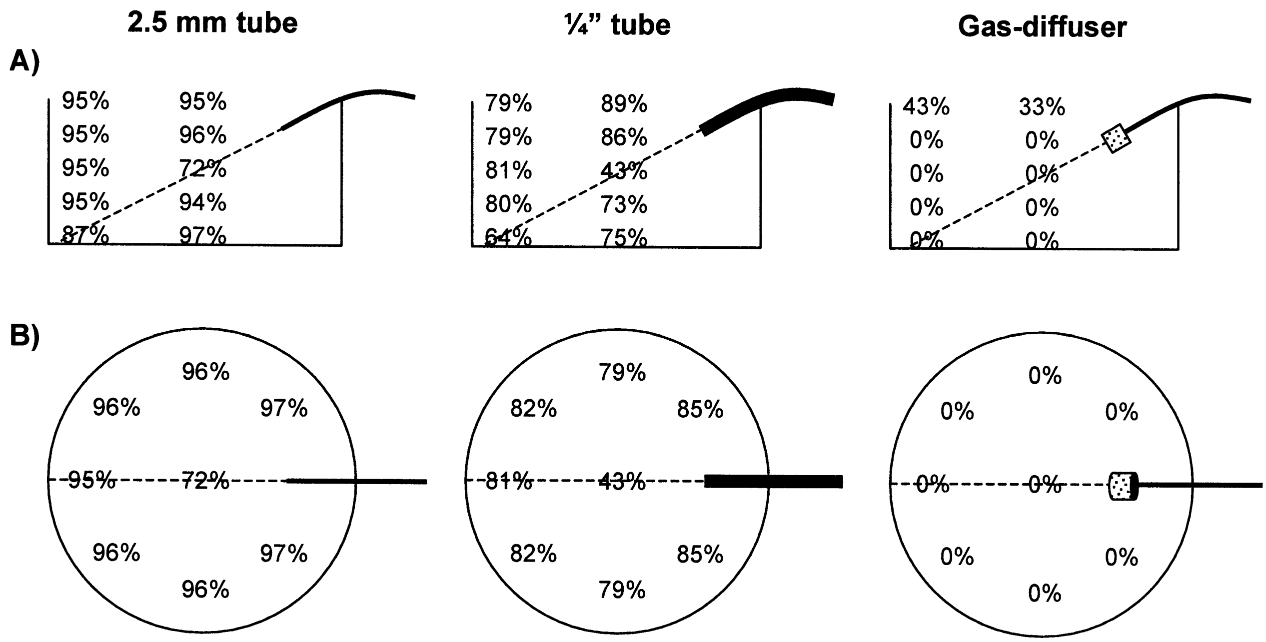

Fig 3. Distribution of mean air contents in % (n = 10) in (A) the vertical plane in the direction of the CO_2 insufflation and (B) in the horizontal plane at half the depth of the model, during CO_2 insufflation with a 2.5-mm tube, a 1/4-in (6.35 mm) tube and a gas diffuser at a CO_2 flow of 7.5 L/min. The dashed line marks the direction of the CO_2 insufflation. The air content in positions No. 6, 7, and 8 in the horizontal plane was not actually measured but was because of symmetry assumed to be equal to positions No. 2, 3, and 4, respectively.

position (without effect in a clinical trial),[26] ventricle emptying by compression, and evacuation of trapped air (diagnosed by transesophageal echocardiography)[5] by gravitation or aspiration. Moreover, the cardiothoracic wound may be insufflated with CO_2. The authors are only aware of 1 single study of the effects of CO_2 insufflation, in which a reasonably high degree of de-airing of the cardiothoracic wound has been obtained. In an experimental study with dogs Eguchi et al[2] exposed the mitral valve to the open cardiothoracic wound by opening the left atrium widely for 2 minutes. In the control group, air appeared in the coronary arteries within a few seconds after atriotomy and produced arterial obstruction, and evidence of coronary insufficiency appeared. The heart became dilated, and the cardiac action became so weak that cardiac massage was necessary to maintain the circulation in 70% of the control group. In the treatment group, in which the cardiothoracic wound cavity was insufflated with CO_2, the cardiac action was satisfactory and cardiac massage was not necessary except in 1 case. Ventricular fibrillation occurred in 37.5% of the control group versus 0% in the CO_2 group. The corresponding figures for electrocardiographic changes were 87.5% versus 28.6%. The gross and microscopic examination of the brain revealed

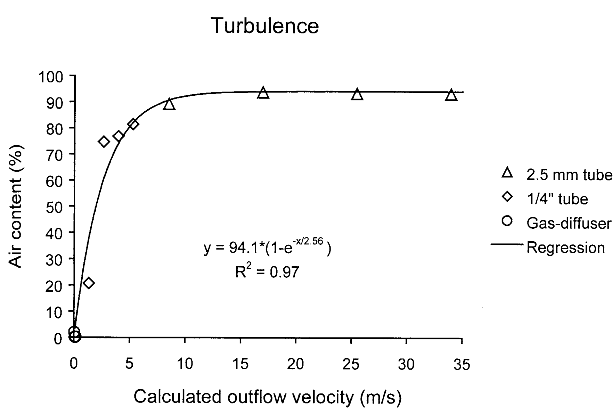

Fig 4. The mean air content in % (n = 80) at half the depth of the model plotted versus the calculated outflow velocity (m/s) of the 2.5 mm tube, the 1/4-in tube, and the gas diffuser at CO_2 flows of 2.5, 5, 7.5, and 10 L/min. The 4 values of the gas diffuser are located close to the origin of coordinates because of low air contents caused by low outflow velocities. The curve represents an exponential regression of the data.

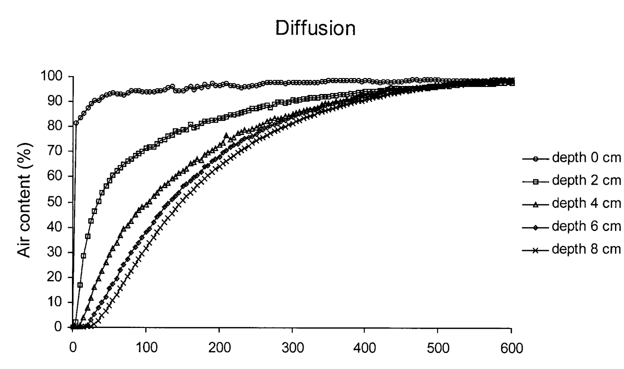

Fig 5. The curves represent the air content in % at 0, 2, 4, 6, and 8 cm depth, respectively, in the middle of the cardiothoracic wound cavity model (initially completely filled with carbon dioxide) during the first ten minutes after discontinuation of CO_2 insufflation.

pathologic findings in 75% of the control group versus 28.6% of the CO_2 group. In the experimental animals CO_2 gas was insufflated into the operative field at a flow of 5 L/min. However, the insufflation device was not described. The air content in the dogs' wound cavities was between 10% and 20%.

In a recent clinical study in cardiac surgery, Martens et al[19] did not find a difference in neuropsychologic outcome when CO_2 insufflation was applied in the cardiothoracic cavity compared with a control group without CO_2. However, they did not achieve efficient de-airing (mean air content 56%, range 14%-92%). They used an open-ended perfusion line with an inner diameter of 2 mm for CO_2 insufflation at a flow of 2 L/min. It was concluded that, "For effective reduction of cerebral and coronary artery emboli, higher levels of CO_2 must be achieved in the operating field by more sophisticated means of application."[19]

An optimal CO_2 insufflation device should provide efficient de-airing of the wound cavity without disturbing the surgeon. In the present study, a tube with an inner diameter of 2.5 mm was studied as a control to the gas diffuser because many cardiac surgeons use a thin tube of similar size to a CO_2 insufflation device.[19,27] The ¼-in (6.35-mm) tube was studied because it has been used as an insufflation device in the authors' department as well as in earlier studies.[18,28] Larger tubes were not studied because the authors consider them to be too bulky for clinical use.

Although air is a mixture of several gases, including nitrogen and oxygen (20.95% near sea level),[20] atmospheric air acts as one gas at normal atmospheric pressure and temperature. Because nitrogen is difficult to detect, air displacement by CO_2 insufflation can be assessed by measuring the content of either CO_2 or O_2[13,18] present in the wound cavity. Avogadro's law states, "At constant pressure and temperature equal volumes of different gases contain equal amounts of gas molecules". This means that for every 5 CO_2 molecules that are supplied to a cavity, 5 air molecules are displaced, of which approximately 1 is an O_2 molecule. Thus, when the content of 1 of the gases is measured, the content of the other gases are indirectly measured at the same time.

The O_2 instrument with a heated ceramic sensor that was used can assess air displacement more accurately and faster than commonly used CO_2 sensors that use an optical infrared sensor technique to measure CO_2 concentrations up to 100%. The O_2 sensor's accuracy is 1% of the measured value in the range 0.0001% to 100% O_2, which means that the accuracy increases when the O_2 and the air content decrease. Moreover, the O_2 sensor requires only a 2-mL gas sample volume and has a response time of <2 seconds. The resulting sampling flow of <0.06 L/min should cause minimal interference with the de-airing because the studied CO_2 flows were much higher, ≥ 2.5 L/min. In contrast, CO_2 sensors using the infrared technique usually have a constant accuracy of approximately $\pm$ 2% units CO_2 over the entire range of measurement 0% to 100% CO_2, a larger required sampling volume, and a longer response time, usually >10 seconds. The quick response modality of the O_2 sensor used enabled the detection of rapid variations of air content over time. Thus, the authors consider the O_2 sensor used to be more suitable for evaluation of CO_2 de-airing during steady state and during changes than optical infrared CO_2 sensors.

When a cavity is insufflated with CO_2, air and CO_2 will spontaneously mix because of diffusion. Just as a temperature difference is the driving potential of heat transfer, a concentration difference is the driving potential of diffusion. The higher the concentration difference (ie, diffusion gradient), the higher is the diffusion flux.[29] Diffusion of 2 gases, a and b, can be expressed according to Fick's law in a simplified 1-dimensional form as:

$$J = \frac{D_{ab} \cdot A \cdot \Delta C}{L}$$

where J is the diffusion flux of 1 of the 2 gases in mol/s (may be converted into L/min), D_{ab} is a diffusion coefficient for gas a and b (CO_2 and air), A is the area through which diffusion occurs (opening of the cavity), ΔC is the concentration difference of 1 of the 2 gases between 2 points along the direction of the diffusion transfer (depth of the cavity), and L is the length between the 2 points (depth below the cavity opening). Thus, the diffusion flux is (1) greater when the opening of the wound cavity is larger, (2) greater when the air content inside the cavity is lower, and (3) greater near the opening of the cavity. The last 2 statements were confirmed in the study (Fig 2C, Fig 5).

The shallowest part of a cardiothoracic wound cavity is the anterior part of the aortic root, usually 3 to 4 cm below the wound edge in adult patients. However, the data imply that diffusion had a minimal effect on the de-airing already at a depth of 2 cm and deeper when CO_2 was supplied at a flow of ≥ 5 L/min (Fig 2C). The effect of diffusion could only be observed with the gas diffuser because of minimal turbulence and low air contents in the model. The impact of diffusion on air displacement was only prominent at a CO_2 flow of 2.5 L/min with significantly increased air contents at all depths in the cavity. Higher CO_2 flows compensated for the CO_2 that diffused out of the cavity and for the air that correspondingly at the same time diffused into the cavity. Earlier studies have suggested a minimum required CO_2 flow between 2 to 5 L/min,[2,13,18] but the reason for this has hitherto not been explained.

The major cause of insufficient air displacement with tubes was consequently turbulence induced by high outflow velocities of the CO_2 jets. The same phenomenon occurs when trying to fill a pail with water using a garden hose. Most of the water splashes out of the pail. By contrast, the pail is quickly filled if the hose is provided with a multiperforated nozzle resulting in a reduced outflow velocity. Fig 4 shows how the air content in the cavity increased exponentially when outflow velocity increased. Thus, a slight increase in velocity of the CO_2 gas significantly impairs CO_2 de-airing. The authors are not aware of any earlier report regarding CO_2 de-airing that shows this important relationship.

The larger the dispersing area of the insufflation device, the lower is the outflow velocity (mean outflow velocity = flow/area). The area of the dispersing surface of the chosen diffuser size ($\pi r^2 + 2\pi rl$, 10.5 cm^2), which corresponds to a tube with an inner diameter of 36 mm, is more than 30 times greater than the inner cross-sectional area of the ¼-in tube (πr^2) and more than 200 times greater than the inner cross-sectional area of the 2.5-mm tube. In terms of maximal de-airing efficiency, the gas diffuser was more than 120 and 580 times more efficient than the ¼-in and the 2.5-mm tube, respectively. The difference in de-airing efficiency between the 2.5-mm tube and the gas diffuser (Figs 2-4), which was made of the same tube, shows the decisive effect of the diffuser.

Fig 4 is based on the mean air content at half the depth of the model ($n = 80$), where the influence of air diffusion was minimal (Fig 2C). The transformation of CO_2 flows to outflow velocities (flow/dispersing area of the device) made its relationship to the remaining air content in the model independent of type of insufflation device. Although the delivered CO_2 flows varied (2.5-10 L/min) among the points in the diagram, they still show an exponential relation. This was most probably because of the fact that turbulence (because of high outflow velocities) was the dominating cause of increased air content in the cavity. Diffusion was the primary cause of the increased air content only at a CO_2 flow of 2.5 L/min with the gas diffuser (low outflow velocities).

CO_2 de-airing of a standard cardiothoracic wound cavity has to include the cannulation sites of the aorta and the incisions of the heart. As seen in Fig 3, the air content was somewhat lower in front of the CO_2 jet from the tubes than beside and behind it. Thus, the de-airing efficiency of tubes is direction dependent, and they can therefore not satisfy the above requirement. The fact that the air content in the CO_2 jet was still high even at close range may be explained by ambient air being sucked down with the jet, the so-called ejector effect. In sharp contrast, the gas diffuser produced almost complete air displacement that was independent of the direction of the diffuser, which provides a multidirectional outflow of CO_2.

The wound cavity model had to be insufflated with a CO_2 flow of $\geq$5 L/min to counteract the diffusion. Only the gas diffuser was able to deliver these CO_2 flows with sufficiently low outflow velocity and minimal turbulence. Air displacement was most efficient with a flow of 7.5 L/min. However, a flow of 5 or 10 L/min caused only a fractional and clinically unimportant increase in air content, probably because of diffusion at 5 L/min and to the just slightly higher outflow velocity at 10 L/min. Although a high degree of de-airing was obtained at a CO_2 flow of 5 L/min, higher flows may be necessary intraoperatively to compensate for turbulence from hand movements, convective air currents from ventilation, and for use of suction. Because the air content increased rapidly after discontinuation of CO_2 insufflation (Fig 5), CO_2 should be supplied continuously throughout the operation.

To analyze and solve the problem with inefficient CO_2 de-airing,[13,18,19] the important variables have to be studied separately in a controlled setup. The authors' approach was an *in vitro* study with accurate and systematic measurements in a symmetric cardiothoracic wound model positioned in a nonventilated room. The diffusion curves in Fig 5 show the minimal presence of disturbing air movements in the study room because they show almost no fluctuation.

CONCLUSION

There may be a solution to the problem with inefficient CO_2 de-airing. First, the CO_2 flow must be high enough to counteract diffusion with ambient air. Secondly, the delivered CO_2 must have a low velocity to avoid turbulent mixing with ambient air. Conventional open-ended tubes provided a poor and varying de-airing of the wound cavity model (18%-96% remaining air) because of CO_2 jets with calculated velocities between 1.3 and 34 m/s. The gas diffuser provided an almost complete de-airing of the model (<0.2% remaining air) at flows of 5 to 10 L/min. This was a result of a uniform distribution of CO_2 with calculated velocities of about 0.1 m/s.

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REFERENCES

1. Muth CM, Shank ES: Gas embolism. N Engl J Med 342:476-482, 2000
2. Eguchi S, Sakurai Y, Yamaguchi A: The use of carbon dioxide gas to prevent air embolism during open heart surgery. Acta Med Biol 11:1-13, 1963
3. van der Linden J, Casimir-Ahn H: When do cerebral emboli appear during open heart operations? A transcranial Doppler study. Ann Thorac Surg 51:237-241, 1991
4. Orihashi K, Matsuura Y, Hamanaka Y, et al: Retained intracardiac air in open heart operations examined by transesophageal echocardiography. Ann Thorac Surg 55:1467-1471, 1993
5. Oka Y, Inoue T, Hong Y, et al: Retained intracardiac air. Transesophageal echocardiography for definition of incidence and monitoring removal by improved techniques. J Thorac Cardiovasc Surg 91:329-338, 1986
6. Fries CC, Levowitz B, Adler S, et al: Experimental cerebral air embolism. Ann Surg 145:461-470, 1957
7. Kunkler A, King H: Comparison of air, oxygen and carbon dioxide embolization. Ann Surg 149:95-99, 1959
8. Goldfarb D, Bahnson HT: Early and late effects on the heart of small amounts of air in the coronary circulation. J Thorac Cardiovasc Surg 46:368-378, 1963
9. Spencer FC, Rossi NP, Yu S-C, et al: The significance of air embolism during cardiopulmonary bypass. J Thorac Cardiovasc Surg 49:615-634, 1965
10. Hindman BJ, Dexter F, Subieta A, et al: Brain injury after cerebral arterial air embolism in the rabbit as determined by triphenyltetrazolium staining. Anesthesiology 90:1462-1473, 1999
11. Borger MA, Peniston CM, Weisel RD, et al: Neuropsychologic impairment after coronary bypass surgery: Effect of gaseous microemboli during perfusionist interventions. J Thorac Cardiovasc Surg 121:743-749, 2001
12. Tingleff J, Joyce FS, Pettersson G: Intraoperative echocardiographic study of air embolism during cardiac operations. Ann Thorac Surg 60:673-677, 1995
13. Ng SW, Rosen M: Carbon dioxide in the prevention of air embolism during open heart surgery. Thorax 23:194-196, 1968
14. Mitz MA: CO_2 biodynamics: A new concept of cellular control. J Theor Biol 80:537-551, 1979

15. Eguchi S, Bosher LH Jr: Myocardial dysfunction resulting from coronary air embolism. Surgery 51:103-111, 1962

16. Moore RM, Braselton CW Jr: Injection of air and of carbon dioxide into a pulmonary vein. Ann Surg 112:212-218, 1940

17. Nichols HT, Morse DP, Hirose T: Coronary and other air embolization occuring during open cardiac surgery. Prevention by the use of gaseous carbon dioxide. Surgery 43:236-244, 1958

18. Selman MW, McAlpine WA, Albregt H, et al: An effective method of replacing air in the chest with CO_2 during open heart surgery. J Thorac Cardiovasc Surg 53:618-622, 1967

19. Martens S, Dietrich M, Wals S, et al: Conventional carbon dioxide application does not reduce cerebral or myocardial damage in open heart surgery. Ann Thorac Surg 72:1940-1944, 2001

20. Lindsay B: Mechanics, in Gray DE. (ed): American Institute of Physics Handbook, New York, McGraw-Hill, 1972, pp 134

21. Warren BA, Philp RB, Inwood MJ: The ultrastructural morphology of air embolism: Platelet adhesion to the interface and endothelial damage. Br J Exp Pathol 54:163-172, 1973

22. Herren JI, Kunzelman KS, Vocelka C, et al: Angiographic and histological evaluation of porcine retinal vascular damage and protection with perfluorocarbons after massive air embolism. Stroke 29:2396-2403, 1998

23. Helps SC, Parsons DW, Reilly PL, et al: The effect of gas emboli on rabbit cerebral blood flow. Stroke 21:94-99, 1990

24. Mills NL, Ochsner JL: Massive air embolism during cardiopulmonary bypass. Causes, prevention, and management. J Thorac Cardiovasc Surg 80:708-717, 1980

25. Tovar EA, Del Campo C, Borsari A, et al: Postoperative management of cerebral air embolism: Gas physiology for surgeons. Ann Thorac Surg 60:1138-1142, 1995

26. Rodriguez RA, Cornel G, Weerasena NA, et al: Effect of Trendelenburg head position during cardiac deairing on cerebral microemboli in children: a randomized controlled trial. J Thorac Cardiovasc Surg 121:3-9, 2001

27. Olinger GN: Carbon dioxide displacement of left heart chambers. J Thorac Cardiovasc Surg 109:187-188, 1995

28. Nadolny EM, Svensson LG: Carbon dioxide field flooding techniques for open heart surgery: Monitoring and minimizing potential adverse effects. Perfusion 15:151-153, 2000

29. Incropera FP, DeWitt DP: Diffusion mass transfer, in Fundamentals of Heat and Mass Transfer. New York: John Wiley & Sons, 1996, pp 784–785