Anaesth Intensive Care 2002; **30:** 86-89

Cardiovascular Collapse Caused by Carbon Dioxide Insufflation During One-Lung Anaesthesia for Thoracoscopic Dorsal Sympathectomy

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SUMMARY

Carbon dioxide insufflation into the pleural space during one-lung anaesthesia for thoracoscopic surgery is used in some centres to improve surgical access, even though this practice has been associated with well-described cardiovascular compromise. The present report is of a 35-year-old woman undergoing thoracoscopic left dorsal sympathectomy for hyperhidrosis. During one-lung anaesthesia the insufflation of carbon dioxide into the non-ventilated hemithorax for approximately 60 seconds, using a pressure-limited gas inflow, was accompanied by profound bradycardia and hypotension that resolved promptly with the release of the gas. Possible mechanisms for the cardiovascular collapse are discussed, and the role of carbon dioxide insufflation as a means of expediting lung collapse for procedures performed using single-lung ventilation is questioned.

Key Words: ANAESTHESIA: one lung. THORACOSCOPY: carbon dioxide insufflation. COMPLICATIONS: cardiovascular collapse

Thoracoscopic dorsal (thoracic) sympathectomy is now an established surgical technique[1-3], which is usually performed under general anaesthesia with positive pressure ventilation via either an endotracheal tube or a double-lumen endobronchial tube. Where an endotracheal tube is used[2,4,5], carbon dioxide (CO_2) insufflation is required to achieve adequate surgical access. With single-lung ventilation via a double-luman tube, CO_2 insufflation is not necessary to achieve adequate surgical access but in some centres is used with the object of initiating, expediting or improving lung collapse[6-8].

The practice of CO_2 insufflation during single-lung ventilation has been used now for more than a decade, and over this time there have been at least four clinical reports of associated acute cardiovascular collapse[9-12]. A further instance was reported as an incidental account in a study supportive of the practice[13].

In the present report, a sixth instance of cardiovascular collapse is described, the possible underlying mechanisms are considered, and the continued use of the practice questioned.

CASE HISTORY

A 35-year-old woman ASA 1 presented to a vascular surgeon for elective left thoracoscopic dorsal sympathectomy for hyperhidrosis of the hands and axillae. An uneventful right thoracoscopic sympathectomy had been performed eight months earlier by another vascular surgeon without the use of CO_2 insufflation as part of the technique. The patient had ceased smoking six months earlier and was otherwise in perfect health. No weight was recorded for the patient but she was of normal body habitus.

The unpremedicated patient was anaesthetized with fentanyl 100 μg, propofol 200 mg and rocuronium 50 mg. Non-invasive monitoring of cardiovascular and respiratory function included ECG, blood pressure, end-tidal carbon dioxide, pulse oximetry and airway pressure. A 37 French gauge left-sided double-lumen endobronchial tube (Broncho-cath®) was used to achieve lung separation, and the correct position assessed by observation and auscultation whilst alternately hand-ventilating each lung. Anaesthesia was maintained with 40% oxygen, nitrous oxide, isoflurane and morphine 4 mg IV. The patient was turned to the right lateral position and the position of the double-lumen tube rechecked.

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Accepted for publication on September 10, 2001.

When surgery had commenced, single-lung ventilation was initiated and the non-dependent lung was disconnected at the endobronchial tube connector so that the lung was open to air. After insertion of the thoracoscopic port the surgeon observed the left lung partially collapse. To further improve the surgical view and access, CO_2 was insufflated via the thoracoscope port to a maximum "Pressure Setting" of 15 mmHg (Wiest Laparoflator Electronic 3509, Wiest Medizintechnik GmbH, Germany), although there was uncertainty, after the event, about the initial insufflator flow-rate setting. The apex of the lung was being observed at this time and no obvious change or improvement in the surgical field was noted.

Within 60 seconds of the commencement of the CO_2 insufflation the pulse oximeter failed to register on the left hand, which was noted to be white and to have poor capillary return. The end-tidal CO_2 fell to 25 mmHg, the ECG showed sinus bradycardia (heart rate 40) and the blood pressure was 60 mmHg systolic. The surgeon was asked to release the insufflated CO_2, and atropine 300 μg and ephedrine 6 mg were administered intravenously. Within two minutes the acute deterioration in blood pressure, heart rate, end-tidal CO_2 and peripheral perfusion had resolved.

The operation was then carried out successfully without any further CO_2 insufflation and without further untoward event. The left upper lobe was observed to re-expand promptly on resumption of two-lung ventilation. In view of the intra-operative cardiovascular collapse, a chest X-ray was taken in the post anaesthetic care unit. It showed a residual 20 to 30% pneumothorax on the left, and no right pneumothorax. The residual pneumothorax was treated expectantly. Postoperative recovery was uneventful.

DISCUSSION

The dilemma facing the anaesthetist with regard to the use of CO_2 insufflation during single-lung ventilation for thoracoscopic surgery relates to the markedly conflicting messages in the anaesthesia literature. Two recent studies by Wolfer et al[14] and Ohtsuka et al[13] have suggested that CO_2 insufflation can be used with no or minimal adverse cardiovascular effects, while on the other hand there are several accounts of the practice being associated with acute cardiovascular collapse[9-13].

The clinical features of the cardiovascular collapse are profound hypotension[9-13], reduced peripheral perfusion with loss of pulse oximeter signal as in the case described, reduced SpO_2[9-11], and bradycardia[9-11,13], the latter also being present in the case described. It seems likely from these accounts that the potentially life-threatening collapse occurs soon after the start of the insufflation, and resolves convincingly with the release of the insufflated gas[9-12].

It has been suggested that the clinical picture is like that seen with a tension pneumothorax[10,15], which is characterized by cardiovascular collapse and arterial desaturation. Cardiovascular collapse is likely to be the predominant initial feature when pneumothorax presents during IPPV[16,17], whereas during spontaneous respiration, arterial desaturation is the predominant early feature[18,19]. The adverse effects seen during single-lung ventilation and CO_2 insufflation into the contralateral hemithorax will very likely include both acute cardiovascular collapse and early arterial desaturation. The hypotension and reduced peripheral perfusion reflect a reduced cardiac output due largely to decreased venous return, while the arterial desaturation results from intrapulmonary shunting through the non-ventilated lung, of blood with a low $P\bar{v}O_2$. Distortion of the heart and great veins due to mediastinal displacement may well play little or no part in the cardiovascular collapse. This latter opinion is supported by animal studies on tension pneumothorax[18,19] and by the patient study in which CO_2 insufflation was performed for the express purpose of displacing the mediastinum[13].

Unfortunately, the only study looking specifically at the cardiovascular effects of CO_2 insufflation during single-lung ventilation omitted to report the presence or absence of arterial desaturation[20].

It is of interest that five of the clinical case reports of major cardiovascular collapse during CO_2 insufflation, including ours, describe a slowing of the heart rate. None has reported tachycardia. The bradycardia may be the result of a sudden increase in intrathoracic pressure causing vagal stimulation that is not seen with a slower increase in pressure. Alternatively, a sudden increase in pleural pressure preceding a sympathetically mediated compensatory increase in atrial filling pressures may, by reducing atrial distension and the output from atrial stretch receptors, lead to a reduction in sympathetic stimulation of the sinoatrial node[21]. Most likely, the bradycardia will be attributed to the increasingly quoted Bezold-Jarisch reflex[22-24].

Accepting that major cardiorespiratory effects can follow CO_2 insufflation during single-lung ventilation, closer scrutiny is surely indicated of the two studies that have suggested that the practice is safe. In one, the pleural pressure in 32 anaesthetized patients was increased by 2 mmHg every three to five minutes,

up to a maximum pressure of 14 mmHg[14]. This progressive elevation of pleural pressure failed to result in a significant change in mean arterial pressure, heart rate, arterial oxygen saturation or end-tidal CO_2. The only significant change was an early increase in CVP. In the second study, a pleural pressure of 8 to 10 mmHg, insufflated at a rate of 2 to 3 $l.min^{-1}$, was used in 22 consecutive patients to improve surgical access for the thoracoscopic harvesting of the left internal mammary artery for minimally invasive coronary artery bypass grafting[13]. No significant changes were found in arterial blood pressure, heart rate or cardiac index, and only a small increase in CVP and in PAP. However, the authors did specifically warn that in their clinical experience, "rapid delivery of carbon dioxide into the chest cavity was dangerous and caused significant reduction in heart rate and blood pressure". Probably relevant was the fact that the insufflation was not started until it was confirmed that the left lung had already collapsed "satisfactorily".

There are several possible explanations for the failure of these two studies to demonstrate adverse cardiovascular effects. In both, the non-ventilated lung would have been largely collapsed at the time the pleural pressures of 14 and 8 to 10 mmHg respectively were attained. In both, hypoxic pulmonary vasoconstriction would already have been limiting shunting through the non-ventilated lung, and in both there would have been time for sympathetic responses to have compensated, at least in part, for any decrease in venous return. This was certainly not the situation with our patient, and very likely with the others, where the cardiovascular collapse occurred soon after the start of the CO_2 insufflation.

It is postulated that the mechanism underlying the acute cardiorespiratory collapse is as follows. CO_2 insufflation that occurs too rapidly might produce an increase in pleural pressure of sufficient magnitude to result in small airways closure. This could conceivably occur at a time when only a relatively small volume of gas has been introduced into the thoracic cavity. Once airways closure has occurred, further CO_2 insufflation may well serve to increase the intrathoracic pressure without causing further venting of gas from the lung[25]. A resulting relatively rapid increase in intrathoracic pressure will reduce venous return and lower cardiac output, while shunting through the non-ventilated lung will result in accompanying arterial desaturation. A rapid rise in intrathoracic pressure will in theory be more likely if the bronchial cuff of a malplaced left-sided double-lumen tube obstructs either the left upper lobe bronchus during CO_2 insufflation into the left hemithorax, or the right main bronchus during CO_2 insufflation into the right hemithorax.

It is reasonable to conclude, therefore, that if CO_2 insufflation is undertaken during single-lung ventilation, every effort should be made to avoid a sudden increase in pleural pressure. Wolfer et al[14] believe that "careful insufflation of CO_2 under conditions of low pressures (<10 mmHg) and low flow (<2 $l.min^{-1}$) is safe". Perhaps to this should be added the recommendation that following the opening of the thoracic cavity to free access of ambient air, the lung should undergo initial partial collapse before CO_2 insufflation is initiated.

Such precautions may not eliminate the problem, however, and adverse cardiovascular effects have been reported with insufflation pressures as low as 5 mmHg[12]. If the practice is potentially hazardous, the question must be asked whether CO_2 insufflation does in fact result in an increased rate of collapse of a non-ventilated lung. Interestingly, there are no reported studies showing that it does. What is more, a recent study on anaesthetized patients conducted during single-lung ventilation prior to thoracoscopic surgery, found that repeated transient small increases in pleural pressure failed to produce further venting of gas from the non-ventilated lung into a measuring spirometer[25]. After the pleural cavity was opened to ambient air, passive partial collapse of the non-ventilated lung occurred promptly, and the venting of gas into the spirometer had ceased within 120 sec (after being 85% complete in the first 25 seconds and 97% complete in the first 60 seconds).

This rapid phase of partial lung collapse was presumed to reflect the inherent elastic recoil of the lung, and the early cessation of venting to be a consequence of small airways closure as the lung collapsed down. Subsequent transient increases in pleural pressure were ineffective in producing additional venting, and it was therefore concluded that any further collapse of the non-ventilated lung must be due solely to ongoing gaseous uptake and "absorption atelectasis". Thus, the practice of CO_2 insufflation may well be no more effective in increasing the speed of lung collapse during single-lung ventilation than simply opening an adequately sized thoracoscopy port to the ambient air for 30 to 60 seconds. One could hypothesize further and suggest that instead of having no effect, the increased pressure on the lung might, by reducing pulmonary blood flow in that lung, actually serve to slow the ongoing absorption atelectasis.

It appears therefore, that CO_2 insufflation during

thoracoscopic surgery using single-lung ventilation probably has very limited indications. Elective displacement of the mediastinum to improve surgical access for harvesting of the internal mammary artery for minimally invasive coronary artery bypass grafting has been suggested as one such indication[13].

With thoracoscopic surgery performed using two-lung rather than single-lung ventilation, CO_2 insufflation does not carry the same theoretical or reported risk of major acute cardiovascular collapse. It will be expected, however, to lower cardiac output[17], and it will not always allow optimal surgical access[17].

In conclusion, the present case report is a timely reminder of the potential dangers of CO_2 insufflation during thoracoscopic procedures performed with single-lung ventilation. It is yet another account of a major cardiovascular collapse that resolved promptly following the release of the insufflated gas. Thus, where CO_2 insufflation is considered to be indicated, it should probably be started at a low 1 $l.min^{-1}$ insufflation rate and only after the pleural cavity has been opened to free access of ambient air for at least 60 seconds. Although adherence to these recommended precautions may increase the safety of CO_2 insufflation during single-lung ventilation, we believe its role to be extremely limited. As a means of expediting lung collapse, it may well be totally ineffective.

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