Capnography in the early diagnosis of carbon dioxide embolism during laparoscopy

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Venous embolism of carbon dioxide occurred during elective diagnostic laparoscopy in a healthy adult female. The diagnosis of gas embolism was made on the basis of the sudden abrupt onset of systolic and diastolic murmurs. The continuously recorded end-tidal carbon dioxide concentration ($F_{ET}CO_2$) increased abruptly from 3.8 to 4.2 per cent and then slowly decreased to 4.0 per cent over the subsequent 30 seconds. CO_2 insufflation was terminated immediately following the establishment of the diagnosis. The patient recovered uneventfully. A transient but rapid rise in $F_{ET}CO_2$ is suggested as a useful early sign of venous CO_2 embolism during laparoscopy.

Key words
COMPLICATIONS; gas embolism, carbon dioxide embolism; MONITORING; end-tidal carbon dioxide.

Laparoscopy for gynaecologic diagnosis and therapy is a well established technique which, in skilled hands, has an acceptably low morbidity. A serious anaesthesia-related complication is embolization of the insufflating gas which was reported in 15 out of 113,253 gynaecologic laparoscopies under general anaesthesia[1] and in one out of 63,845 laparoscopies for liver biopsy.[2] This low incidence and the relative safety of the procedure[1,3] may have led to complacency and a reluctance to routinely use invasive monitoring systems, such as the central venous line, which have been shown to be effective in detecting and treating air embolism during neurosurgery.[4] However deaths have been reported from embolization of the insufflating gas,[5–7] and therefore an examination of non-invasive monitoring is warranted in order to better predict and perhaps to prevent further mishaps. We have been monitoring laparoscopy patients with a precordial stethoscope and capnography and report our findings from an episode thought to be embolism of CO_2.

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Case report

A 32-year-old woman, ASA physical status class I, weighing 50 kg, with primary sterility, was admitted for elective diagnostic laparoscopy. Heart sounds were normal preoperatively.

After premedication with meperidine 50 mg and scopolamine 0.3 mg IM, the indirect blood pressure was 105/65 and heart rate 95 beats/min. Anaesthesia was induced with intravenous flunitrazepam 0.4 mg and thiopentone 175 mg, and tracheal intubation carried out following gallamine 10 mg and succinylcholine 90 mg. Ventilation was controlled, with a tidal volume of 600 ml, rate 10 breaths per minute, and with a total gas flow of 7 litres/min (nitrous oxide 5 l/min and oxygen 2 l/min). A Bain circuit was used. Fentanyl 0.15 mg and a succinylcholine 0.1 per cent drip maintained anaesthesia. A precordial stethoscope was placed over the left fourth interspace and a Datex CD300 capnograph was connected via a side arm at the tracheal tube connector. (The capnograph was calibrated from two known CO_2 sources and adjusted for the presence of N_2O by an electrical offset switch.[8]) The initial fractional end tidal CO_2 concentration ($F_{ET}CO_2$) was 3.8 per cent, equivalent to a partial pressure of CO_2 in end tidal gas ($P_{ET}CO_2$) of 3.3 kPa (25 mmHg).

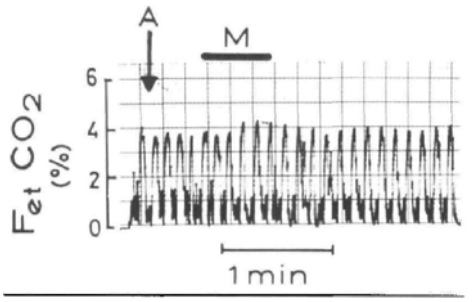

FIGURE 1 Fractional end tidal CO_2 (per cent) vs time. Insufflation of CO_2 started at A. Murmur heard and insufflation discontinued at M.

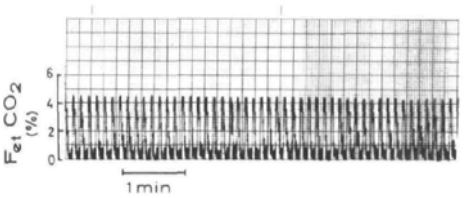

FIGURE 2 Fractional end tidal CO_2 (per cent) vs time. Graph taken during the second insufflation and representative of the record throughout the procedure following the initial abortive insufflation.

The patient was placed in the horizontal lithotomy position and a Verres needle introduced into the abdominal cavity at the umbilicus. Carbon dioxide was insufflated at 3 l/min from a Wisap CO_2-pneu with 2.7–4.0 kPa pressure recorded. Within approximately 20 sec of the start of the insufflation, a loud coarse systolic murmur with a softer diastolic component was heard over the precordium. The heart rate was 90/min, blood pressure 115/80 with no other change in the patient's condition. The insufflation was immediately stopped and the needle withdrawn. A few seconds later the $F_{ET}CO_2$ increased from 3.8 to 4.2 per cent and then slowly decreased to 4.0 per cent over the next half minute (Figure 1). The patient's legs were taken out of the stirrups in preparation for left lateral decubitus positioning, but at this time the murmur softened and then disappeared. The patient was observed carefully for more than 10 min during which time there were no changes in the vital signs. The capnograph record then showed a gradual increase of the $F_{ET}CO_2$ to 4.5 per cent at which level it remained throughout the procedure. The needle was re-positioned and insufflation repeated uneventfully (Figure 2), and the laparoscopy was then completed with the postoperative diagnosis of extensive intraperitoneal adhesions. The patient awakened immediately after the procedure, at which time the ECG, chest *x*-ray and blood gases were normal. She subsequently had an uneventful course and was discharged the next day.

Discussion

Embolization of the insufflating gas during induction of pneumoperitoneum for laparoscopy is a sudden dramatic event caused by accidental puncture of intra-abdominal blood vessels with subsequent intravascular insufflation of the gas. The exact site of intravascular insertion of the needle is usually difficult to determine,[9] though the uterine blood vessels are a likely site of entry of gas following intrauterine insertion of the needle.[10] This approach to the peritonal cavity thus may predispose to gas embolism and is not recommended. Where intraperitoneal adhesions exist, they fix the otherwise mobile omentum to other structures and may similarly predispose to intravascular penetration and gas embolization.[2]

When gas is insufflated intravenously it is rapidly carried to the vena cava and right atrium where it forms a "gas lock." This is true both for air[11,12] and for carbon dioxide,[13] and can result in obstruction to venous return with a precipitous fall in cardiac output. The churning action of the heart breaks the gas up into small bubbles, producing a foam[9] which can advance through the right heart and reach the pulmonary circulation, where it may cause pulmonary hypertension and right heart strain.[9] The amount of gas which will advance in this way is less if the patient is in the left lateral decubitus position,[13,14] or in the steep head down position[15] as the buoyant foam will then be displaced laterally and inferiorly respectively and out of the main blood channel. The size of the bubbles and the rate of the intravenous entry of the gas will also determine the likelihood of passage of the bubbles through the right heart. During neurosurgery the slow entrainment of small bubbles of air is more likely to result in air entrapment in the pulmonary vessels whereas during laparoscopy the rapid insufflation of gas under high pressure probably causes a "gas lock" in the vena cava and right atrium which would

effectively trap subsequent bubbles and prevent gas entry into the pulmonary vessels.[11]

Carbon dioxide is used most frequently for laparoscopy because it is more soluble in blood than either air or oxygen, and therefore a greater quantity of embolic carbon dioxide can be tolerated.[16,17] Absorption of CO_2 is rapid so that dissolution of the foam and reversal of the haemodynamic impairment quickly occur. The platelet aggregation and mediator-induced bronchospasm which occur at air/blood interfaces[18] do not seem to occur with CO_2 emboli. Thus reports of CO_2 embolism have often commented on the rapid reversal of signs and the relatively benign postoperative course [9,10,15,19]

If however the disturbance is unrecognised and there is continued intravascular insufflation of the CO_2, other gases, chiefly oxygen, begin to diffuse into the bubbles.[20] The embolism then will require more time to be absorbed and a vicious cycle could be established. This may account for the reports of permanent injury[6] and deaths[5–7] following CO_2 embolism, and emphasises the importance of prompt diagnosis by the anaesthetist in order to prevent a small embolus from enlarging.

The presenting signs of gas embolism during laparoscopy include sudden profound hypotension, cyanosis, tachycardia and other dysrhythmias, or an alteration in heart tones. The first sign may be pulmonary oedema,[21] and delayed sudden death has also been reported.[5]

Before making the diagnosis of gas embolism, other causes of sudden cardiovascular collapse during laparoscopy must be eliminated, such as intra-abdominal haemorrhage, pulmonary thromboembolism, pneumothorax or pneumomediastinum, diaphragmatic rupture, excessive intra-abdominal pressure, vasovagal reflex, and hypoventilation which may cause dysrhythmias, especially in the presence of halothane.

Aspiration of gas or foamy blood from a central venous line will establish the diagnosis. Preoperative insertion of a central venous line routinely is not, however, always justified for a low risk procedure.

Diagnosis of gas embolism by non-invasive means and at an earlier stage would clearly be more beneficial, and monitoring of the heart sounds with a precordial or oesophageal stethoscope may accomplish this. Heart sound changes reliably detect air embolism[4] and may also help to establish the rate and quantity of air infused.[11] Typical murmurs of air embolism may occur in the absence of clinical signs[4,22] and at times, even before changes in the heart sounds are heard on the Doppler apparatus.[4,23] During CO_2 insufflation for laparoscopy, the sudden onset of "mill wheel" murmurs in two cases,[9,19] and of a coarse sloshy murmur in another case[15] was followed by cardiovascular collapse and was attributed to CO_2 embolism. In our patient the sudden appearance of a murmur which had the characteristics of a "mill wheel" murmur, was highly suggestive of this diagnosis and immediate cessation of the insufflation probably prevented deterioration of the clinical status.

The most sensitive monitor of intra-cardiac gas is the oesophageal Doppler apparatus which can detect as little as 0.05 ml of air.[24] The precordial Doppler is sensitive to 2 ml of CO_2 injected into the right atrium,[23] and has been used during laparoscopy in 100 patients, with no evidence of intra-cardiac gas.[25] This would seem to confirm that gas embolism during laparoscopy does not commonly occur sub-clinically as is the case during neurosurgery.

Other factors which should alert the observant anaesthetist are the presence of blood on aspiration from the Verres needle, pulsations of the flow meter with the patient's pulse during insufflation,[15] and absence of signs of distention of the abdomen in spite of an adequate volume of insufflated gas.

Capnography is a useful non-invasive monitoring technique in anaesthesia which reflects reasonably accurately the arterial CO_2 tension in patients without serious cardiopulmonary disease.[26,27] The $FETCO_2$ immediately reflects changes in ventilation and circulatory status as well as alterations in position and temperature of the patient. It is very sensitive to changes in lung mechanics which may occur during anaesthesia, especially changes in alveolar dead space.[27,28]

The patient described in this report showed a transient 0.4 per cent increase in $FETCO_2$ which began a few seconds after the onset of the murmur. No comparable abrupt rise was noted in any one of 57 patients undergoing gynecologic laparoscopies monitored by capnography by one of us (DS). In these cases there was generally a very gradual rise of $FETCO_2$ over the course of the laparoscopies (mean duration 32 min) which reached its maximum at the end of the procedure. The mean rise at this time was 0.2 per cent (±0.4 per cent) which is

small compared to the mean rise found by Seed *et al.*,[26] probably because we tried to adjust the ventilation to maintain a constant $FETCO_2$, in spite of the steep head down position of the patient. There were no changes in the heart sounds in any of these patients, all of whom were monitored with an oesophageal or pre-cordial stethoscope.

We know of no previous clinical case report of CO_2 embolus where capnographic monitoring was co-incidentally used. Oppenheimer *et al.* demonstrated in dogs that CO_2 embolization caused a biphasic change in $PETCO_2$.[29] The initial increase in $PETCO_2$ was probably the result of excretion of CO_2, which had been absorbed into the blood, by the lungs. The subsequent decrease was explained by blockage of the pulmonary arterioles by bubbles of CO_2 resulting in increased alveolar dead space. This effect is similar to but less marked than that due to air emboli since CO_2 emboli do not produce bronchoconstriction or changes in pulmonary compliance as do air bubbles[18] and also disappear more rapidly from the pulmonary circulation.[18] Thus a small CO_2 embolus may produce only the initial rise in $FETCO_2$ without a significant subsequent decrease.

In our patient there were no obvious causes for the simultaneous appearance of a heart murmur and a transient rise in $FETCO_2$ other than CO_2 embolus. The patient was in the horizontal position and there were no changes in the ventilator settings at that time. Absorption of CO_2 from the peritoneum should not have been significant at the beginning of the procedure,[26] nor were there subsequent disturbances in the otherwise smooth record of $FETCO_2$ (Figure 2). Though not in itself sufficient to establish the diagnosis, the pattern seen on the capnograph record, coinciding with a transient heart murmur, is suggestive of CO_2 embolus. Changes in $FETCO_2$ may in this way complement other signs of CO_2 embolism and help to confirm this diagnosis.

Treatment of gas embolus consists initially of immediate cessation of the insufflation and placing the patient in the left lateral decubitus (Durant's) position.[14] The steep head down position has also been advocated[15] but since the gas bubble may be found at the junction of the inferior vena cava and the atrium,[13] assumption of this position may cause the bubble to enter further into the cardiac chambers and this may increase the disturbance to blood flow. Discontinuing nitrous oxide will allow 100 per cent oxygen to be given, but will not result in reduction of the size of a CO_2 embolus, as is the case with air embolus. This is due to the similarity of the solubilities in blood of N_2O and CO_2[15] so that there will not be significant diffusion of N_2O into or out of the CO_2 bubbles. Hyperventilation will, however, increase CO_2 excretion. If these simple measures are not rapidly effective, a central venous catheter may be introduced for aspiration of the gas, though Alvaran *et al.*[30] have shown in dogs that during air embolus aspiration of air in this way was no more effective than the left lateral decubitus position. Cardio-pulmonary resuscitation may be necessary in order to maintain oxygenation of vital organs.

In summary, we have described the sudden appearance during laparoscopy of a heart murmur that coincided with a transient rise in $FETCO_2$ during insufflation of CO_2 and we have attributed these findings to CO_2 embolism. Early diagnosis and immediate cessation of the insufflation probably prevented progression of the haemodynamic disturbance. We recommend continuous monitoring of the heart sounds routinely during this procedure and, if available, capnographic monitoring which may provide information that will assist the anaesthetist in making the diagnosis of gas embolism early.

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Résumé

Une embolie de gaz carbonique a été détectée pendant une laparoscopie diagnostique chez une femme en bonne santé. Le diagnostic a été posé grâce à l'apparition subite de souffles cardiaques systolique et diastolique caractéristiques. L'enregistrement continu de la pression partielle de CO_2 télé-expiratoire ($F_{ET}CO_2$) a montré une élévation abrupte de 3.8 à 4.2 pour cent puis une descente lente à 4.0 pour cent en 30 secondes. L'insufflation de CO_2 fut arrêtée dès le diagnostic posé. La patiente a récupéré sans problème. Une élévation rapide et transitoire de $F_{ET}CO_2$ semble être un signe précoce utile de l'embolie de CO_2 pendant une laparoscopie.