Profound hypercapnia due to disconnection within an anaesthetic machine

Mark S. Berner MD CCFP(EM)

A 25-year-old patient received general anaesthesia for an emergency Caesarean section. Arterial blood gases obtained because of prolonged postoperative coma demonstrated profound hypercapnia (PaCO_2 246 mmHg). Examination of the anaesthetic machine revealed a complete disconnection of the metal components of the main gas line downstream from the vaporizer, in a location that was obscured from the anaesthetist's view. Causes of profound hypercapnia are reviewed and preventive measures are discussed.

Hypercapnia is often difficult to detect during general anaesthesia, for two reasons. First, it is unexpected. Second, one of the main manifestations of hypercapnia, increased sympathetic activity, is often seen in response to surgical stimuli. Additionally, in a paralysed, unconscious patient who is being ventilated, other important signs of hypercapnia such as decreasing level of consciousness and tachypnoea are lost.

The following case report describes profound hypercapnia consequent to a major anaesthetic machine leak which was neither apparent nor visible to the anaesthetist. During the course of the anaesthetic the odour of anaesthetic vapour was not noted and the low pressure alarm on the ventilator did not trigger. Many of the usual signs of increased PaCO_2 were absent.

Case report

A 25-year-old primigravida at 38 weeks gestation was

booked for emergency Caesarean section because of fetal distress. She was assessed as ASA physical status IIE, on the basis of her gravid state and obesity (weight increase from 72.6 kg before pregnancy to more than 93 kg at the time of operation).

The anaesthetic machine was an Ohio 30/70 Proportioner with an attached Ohio Anaesthesia Ventilator of the "hanging bellows" type.

Following pre-oxygenation and "pre-curarization," rapid sequence induction of general anaesthesia was achieved with thiopentone and succinylcholine. Anaesthesia was maintained with nitrous oxide ($4.5 \text{ L} \cdot \text{min}^{-1}$) and oxygen ($3.5 \text{ L} \cdot \text{min}^{-1}$ until the umbilical cord was clamped, and then $2.5 \text{ L} \cdot \text{min}^{-1}$) plus enflurane one per cent. Relaxation was maintained with d-tubocurarine (21 mg initially followed by three increments of 6 mg each). Immediately after clamping of the cord, divided doses of fentanyl were given to a total of 200 μg . The patient was ventilated with a tidal volume of 900 ml at 11 cycles $\cdot \text{min}^{-1}$. A Bain anaesthetic breathing circuit was used.

Anaesthesia and surgery appeared to proceed uneventfully. The umbilical cord was clamped 11 minutes following induction. The Apgar scores were 7 at one minute and 10 at five minutes.

Following completion of surgery, the neuromuscular block was reversed with neostigmine 2.5 mg and atropine 1.2 mg. Despite what seemed to be satisfactory respiratory efforts, the patient's breathing was uncoordinated and erratic to the point that manual assistance was required. Assessment with a nerve stimulator showed no evidence of persisting neuromuscular blockade. Two 0.2 mg doses of naloxone produced no appreciable change. At this point, the pulse was 120 bpm and bounding and the blood pressure was 170/80 mmHg. The pupils were dilated, responding sluggishly to light. The gag reflex was absent. No localizing neurological signs were elicited. Arterial blood gases were obtained ($\text{FiO}_2 = 1.0$): PaO_2 95 mmHg, PaCO_2 246 mmHg, pH 6.48, base deficit -29.6. Haemoglobin was $116 \text{ g} \cdot \text{L}^{-1}$, Na 133, K $3.5 \text{ mmol} \cdot \text{L}^{-1}$ and glucose $25.2 \text{ mmol} \cdot \text{L}^{-1}$.

The astounding degree of hypercapnia was greeted with disbelief. In order to provide confirmation, the laboratory

Key words

EQUIPMENT: anaesthesia machine; COMPLICATIONS: hypercapnia; ANAESTHESIA: general, obstetrics.

From The Lady Minto Hospital, Cochrane, Ontario and The Department of Family Medicine, McGill University, Montreal, Quebec.

Address correspondence to: Dr. M. Berner, Family Medicine Centre, St. Mary's Hospital Annexe, 3830 Lacombe Avenue, Montreal, Quebec, H3T 1M5.

FIGURE 1 Site of disconnection as it appeared to the anaesthetist.

instrument was re-calibrated with a nursing supervisor serving as a normal control.

In the meantime, controlled hyperventilation had been immediately begun, initially with the Ohio anaesthesia ventilator affixed to the anaesthetic machine and subsequently with a Bennett MA-1 ventilator brought into the operating room. Three 50 mEq doses of sodium bicarbonate were administered IV.

Within three hours of reversal the patient was extubated. Further blood testing revealed glucose to be $19.3 \text{ mmol}\cdot\text{L}^{-1}$ and potassium $7.4 \text{ mmol}\cdot\text{L}^{-1}$. Six units of regular insulin were administered. Thereafter both clinical and biochemical status remained satisfactory. There were no further difficulties and no permanent sequelae.

Once the patient was stable, the anaesthetic machine was carefully examined. A complete disconnection in the main metal gas line distal to the flow meters and enflurane vaporizer was discovered (Figure 1). Because of the metal flange located on the nut that had come loose, the problem was obscured on visual scanning of the apparatus (Figure 2).

FIGURE 2 Site of disconnection with flanged nut retracted.

Discussion

Generally, hypercapnia with PaCO_2 values in excess of 100 mmHg is unlikely to occur in patients breathing room air, since the associated degree of hypoxia would be incompatible with survival. Gross hypercapnia can only occur if a patient is breathing oxygen-enriched air or is exposed to high concentrations of carbon dioxide in the inspired gas.¹

Four main causes of gross hypercapnia in association with anaesthesia have been described:

- i) Reduced alveolar ventilation with a resultant rise in PaCO_2 .
- ii) Inhalation of exhaled carbon dioxide (as has been noted previously in association with a defective fresh gas flow tube of a Bain circuit).²
- iii) Inhalation of exogenous carbon dioxide.
- iv) Increased metabolic rate, as in malignant hyperthermia.

The systemic effects of hypercapnia affect mainly the central nervous system and cardiovascular system. Increasing levels of PaCO_2 cause a decreasing level of

consciousness. Respiration is initially stimulated; as PaCO_2 further increases, respiratory depression ensues. The general sympathetic hyperactivity more than offsets the degree of myocardial depression with the net result of increased cardiac output and peripheral vasodilatation. Nerve conduction within the heart is impaired, promoting bradyarrhythmias. In addition, myocardial irritability is heightened. Increased bleeding occurs in surgical wounds. Under anaesthesia, many of the above signs may be masked³ or misinterpreted. Consequently, monitoring of exhaled CO_2 is a valuable aid in detecting a potentially lethal problem that may not be apparent clinically.

In this case, the pulse rose to 150 bpm on intubation and then remained at approximately 140 bpm, while the blood pressure rose to 200/110 mmHg before settling to approximately 165/75 mmHg. These values were attributed to a light plane of anaesthesia in an obese patient. During the course of the anaesthetic, none of the features usually associated with hypercapnia was apparent: the colour of blood in the surgical field was never in question, there was no increase of wound bleeding or oozing, peripheral vasodilatation was not seen and there was no arrhythmia, except for the tachycardia which had been present from induction. Throughout, the odour of enflurane was not noted. The low pressure alarm on the ventilator did not trigger. The diagnosis of hypercapnia was made on the basis of arterial blood gases drawn from a nonresponsive postoperative patient.

The treatment of uncomplicated respiratory acidosis is increased ventilation. The use of sodium bicarbonate is potentially hazardous as the carbonic acid equation may be driven to the left, thereby increasing production of CO_2 :

In this situation, the decision to use sodium bicarbonate was an empirical one based on the profound degree of acidosis, and in the absence of an apparent cause, reluctance to believe that the acid-base derangement was purely respiratory.

Once the circumstances were clarified, it became apparent that profound hypercapnia resulted from a major partial leak in the anaesthetic circuit which caused a decreased minute volume due to a decreased ventilating volume. Nevertheless, the disconnection was not a complete one for two reasons:

- 1 In this patient, approximately 129 minutes elapsed from induction to the time of the initial arterial blood gas determination. The total incremental rise in PaCO_2 was 206 mmHg (246 - 40). Assuming a constant rate of CO_2 production, the rate of rise of PaCO_2 is estimated at $1.6 \text{ mmHg} \cdot \text{min}^{-1}$ (206 divided by 129).

For an apnoeic patient, the rate of rise of PaCO_2 is 3-6 $\text{mmHg} \cdot \text{min}^{-1}$.^{3,4}

- 2 If the disconnection had been complete, the ambient gas would have been room air. The principle of apnoeic mass movement oxygenation suggests that had this been the case, then severe hypoxia would have occurred within minutes.⁴ This did not happen. Therefore, at least some of the oxygen-enriched mixture ($\text{FiO}_2 = 0.35$) from the anaesthetic machine was being delivered to the patient.

Assessing the integrity of the anaesthetic circuit

A number of tests have been described to assess the low pressure portion of the anaesthetic circuit for patency and freedom from leaks.

*Pethick's oxygen flush test*⁵ is a method of ensuring that the inner tube of the Bain breathing circuit is intact. It is performed by using the oxygen bypass and observing the reservoir bag. Collapse of the reservoir bag, secondary to the Venturi effect in the outer tube, is an indication that the inner tube circuit is intact.

*The Foex-Crampton Smith Manoeuvre*⁶ is a method of assessing the integrity of the gas line from the flow meters to the patient end of the Bain breathing circuit. It is performed as follows: with oxygen flowing, occlusion of the inner tube with a finger causes the rotameter to descend slightly within its housing. Withdrawal of the finger causes the rotameter to return to its original position. Ghani⁷ has refined this test by suggesting that the tip of a plunger from a 3 ml syringe be used instead of a finger. This ensures precise occlusion of the inner tube without inadvertent partial occlusion of the outer tube. He also notes that when the plunger tip is removed, the very obvious hissing sound heard is a second indication of the integrity of the circuit. Salt⁸ advises that in performing the test, a low flow of oxygen should be used and that occlusion of the inner tube last only 2-3 seconds. The use of very high flows and/or prolonged occlusion can cause very high pressures to build which in turn may cause damage to the anaesthetic machine.

*A Low Pressure Leak Test Procedure*⁹ has been developed for the Ohio 30/70 Proportioner because the high minimum flow ($3 \text{ L} \cdot \text{min}^{-1}$) from this machine does not permit usage of leak testing methods commonly employed for many other anaesthetic machines. The principle of the test is based on the creation of a partial vacuum in a rubber squeeze bulb connected to the anaesthetic machine. When this has been accomplished, one side of the bulb will be in contact with the other side. If a leak exists in the machine circuitry, air from the atmosphere will be drawn in. This fills the vacuum within the bulb thereby causing the bulb to return to its usual

shape more quickly than the usual 15 seconds. The magnitude of such a leak can be determined by dividing the volume of the bulb (35 ml) by the time required for it to return to its normal shape.

This case occurred in a 50-bed hospital located in a semi-isolated northern community. It had been the only anaesthetic of the day. The involved anaesthetic machine had been fully serviced within the previous week. There had not been a previous history of gas leaks.

The preanaesthetic check of the machine consisted of the following:

- the flow meters were checked to ensure appropriate flows of both O₂ and N₂O
- the enflurane vaporizer was verified to be full
- the pressure of the emergency oxygen cylinder mounted on the machine was verified
- the suction apparatus was checked for proper functioning
- the interface of the Bain breathing circuit to the Bain adaptor arm was checked for tightness of fit
- the re-breathing bag was filled with the oxygen flush bypass and the positive manual pressure was applied to the bag (the "pop-off" valve released as calibrated)
- the inner tube of the Bain breathing circuit was assessed as being intact by a normal Pethick test. The Foex-Crampton Smith/Ghani manoeuvre was not performed as it was unknown to the anaesthetist at that time.

Subsequent testing of the anaesthetic machine revealed that performance of the Ohio Low Pressure Leak Test, even with this disconnection, produced a normal result. The service technician indicated the Low Pressure Leak Test must be done with the Bain circuit adaptor removed, though this fact is not noted on the plastic card affixed to the machine that outlines the testing procedure. The practice at this hospital had been to perform this test with the Bain circuit adaptor in place.

Conclusion

This case report has been prepared to alert others to a potentially lethal hazard, especially because the site of the disconnection was hidden.

In order to obviate problems of mechanical malfunction, conscientious routine servicing and calibrating of equipment by appropriate personnel is mandatory. Additionally, prior to each use, the anaesthetist is responsible for checking his equipment, especially the low pressure segment of anaesthesia system for patency and leaks¹⁰ (Table). In this regard, the value of the Foex-Crampton Smith manoeuvre⁶ with Ghani modification⁷ is emphasized as it appears to be the most satisfactory method of assessing the integrity of the gas line from the flow meters through to the patient end of the Bain circuit.

During the course of anaesthesia, the odour of an

TABLE Guidelines for preanaesthetic check

- 1 General inspection for presence of breathing tubes and reservoir bag, suction tip and tubing, as well as laryngoscope (and back-up), masks, endotracheal tubes, eye lubricant, adhesive tape, etc.
 - ensure suction apparatus functions properly
 - ensure laryngoscope functions properly
 - test cuff of endotracheal tube for leaks
- 2 Ensure O₂ and N₂O lines are properly connected to both wall/ceiling central outlets and anaesthetic machine
- 3 Verify flows of O₂ and N₂O with respective dials on anaesthetic machine and verify O₂ flush bypass
- 4 Verify pressure in emergency O₂ cylinder (ensure tank wrench is present)
- 5 Fill reservoir bag with O₂: apply manual pressure to ensure ability to ventilate with positive pressure (this also checks for holes in the bag); where applicable, verify operation of gas evacuator-relief ("pop-off") valve
- 6 Ensure vaporizer(s) is(are) full
- 7 Ensure monitoring equipment - ECG, temperature, O₂ analyzer, Doppler BP, etc. - is calibrated
- 8 Ensure anaesthetic machine has been tested with the appropriate leak test within the prescribed time interval
- 9 Assess breathing circuit:
 - i) rebreathing circle system
 - ensure functional condition of absorbent and ensure canister is properly secured
 - anaesthetist should inhale and exhale through the system to determine competency of directional valves¹¹
 - where applicable, ensure that valve to include/exclude absorber is in the proper position
 - ii) nonrebreathing system - Bain
 - visually inspect tubing to ensure integrity of inner tube
 - Pethick test* to further verify integrity of inner tube
 - Foex-Crampton Smith/Ghani test* to ensure integrity and patency from flow meters to patient end of breathing tube
- 10 Ensure presence of ventilating bags with supplemental O₂ capability for use in case of suspected equipment malfunction or need to evacuate the operating room in an emergency

*See text for details.

increased concentration of escaping vapour may signal the presence of a leak. A soapy solution may be helpful in identifying the leak origin.

Should a patient's condition change unexpectedly during the intraoperative or postoperative period, the early use of arterial blood gases may help to diagnose the underlying problem. If equipment malfunction is suspected, it might be prudent to go back to basics by using a manual ventilating bag with supplemental oxygen.

Finally, in an unconscious patient, the triad of a bounding pulse with tachycardia, elevation of systolic blood pressure and dilated pupils responding sluggishly to light is suggestive of hypercapnia.

Acknowledgments

To Dr. Gordon Sellery my sincere appreciation for his review of my manuscript and helpful suggestions. I would also like to thank Mr. N. Ramanayake, R.T., and Mr. C. Cristofaro for their photographic skills.

References

- 1 Prys-Roberts C, Smith WDA, Nurem JF. Accidental severe hypercapnia during anaesthesia. *Br J Anaesth* 1967; 39: 257.
- 2 Hannallah R, Rosales JK. A hazard connected with re-use of the Bain's circuit: a case report. *Can Anaesth Soc J* 1974; 21: 511.
- 3 Churchill-Davidson HC. A practice of anaesthesia. 5th ed. Chicago: Year Book Medical Publishers, 1984: 117.
- 4 Nunn JF. Applied Respiratory Physiology. 2nd ed. London: Butterworths, 1977: 359.
- 5 Pethick SL. Letter to the Editor. *Can Anaesth Soc J* 1975; 22: 115.
- 6 Foex P, Crampton Smith A. The Foex-Crampton Smith maneuver (letter). *Anaesthesia* 1977; 32: 294.
- 7 Ghani G. Safety check for the Bain circuit (letter). *Can Anaesth Soc J* 1984; 31: 487.
- 8 Salt R. A test for co-axial circuits: a warning and a suggestion (letter). *Anaesthesia* 1977; 32: 675.
- 9 Ohio Medical Products. Operation and maintenance manual for Ohio 30/70 Proportioner Anesthesia Machine, 1978: 10.
- 10 McIntyre JWR. Anaesthesia equipment malfunction: origins and clinical recognition. *Can Med Assoc J* 1979; 120: 931.
- 11 Dripps RD, Eckenhoff JE, Vandam LD. Introduction to anaesthesia. The principles of safe practice. 6th ed. Philadelphia: WB Saunders, 1982: 62.

Résumé

Une patiente de 25 ans a subi une césarienne d'urgence. Les gaz sanguins artériels obtenus à cause d'un coma nous ont permis de diagnostiquer une hypercapnie profonde (PaCO_2 , 246 mmHg). L'examen de l'appareil anesthésique a révélé une dysjonction complète des composantes métalliques de la ligne principale des gaz dans un endroit caché à la vue de l'anesthésiste. On revise les causes de l'hypercapnie profonde et on discute des mesures de prévention.