Airway obstruction during anaesthesia for anterior cervical cord decompression

To the Editor:

A 73-yr-old man was admitted to our hospital with cervical cord compression at the level of C₄-C₅ and C₆-C₇. Three hours after induction of anaesthesia for anterior cervical cord decompression, an increase in airway pressure suddenly occurred. Causative factors such as inadequate depth of anaesthesia and pulmonary pathology being excluded, the surgeon was asked to halt the procedure whilst the patient's airway problem was being dealt with. As he removed his retractor from the operative site the airway obstruction was suddenly relieved.

The surgeon used an anterior approach to the cervical spine which has been shown to be the safest¹ and the surgical incision made here, in close proximity to the trachea, and the use of a retractor encourages tracheal compression. The retractor was in such a position that it probably compressed the trachea below the level of the distal end of the tracheal tube (TT). Other causes of airway obstruction include kinking of a TT that is *in situ* or deviation of the TT so that the bevel of the tube is intermittently applied to the side wall of the trachea or carina; occasionally herniation of a TT cuff could lead to obstruction.

Monitoring of airway pressure waveform and possibly gas flow throughout the respiratory cycle may assist anaesthetists in assessing the cause of airway obstruction. Obstruction of the airway will lead to a marked increase in initial rise of pressure during the inspiratory phase. Possible techniques of avoiding airway obstruction include insertion of an oesophageal bougie to increase the rigidity of the medial structures; use of an armoured, kink resistant TT with the tube inserted so that its tip lies just above the level of the carina which increases the rigidity of the larynx and trachea and places the bevel of the TT below the site of operation; or the use of a TT in which a Murphy's eye has been cut.

Surgeons and anaesthetists involved in operations performed on the cervical spine should be aware of the possible risk of pressure from surgical retraction causing airway obstruction but careful positioning of an armoured TT and meticulous surgical technique should reduce the risk.

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Subcutaneous emphysema and laparoscopic cholecystectomy

To the Editor:

We believe that there may be a factor more important than the subcutaneous tracking of CO₂ to explain the hypercarbia described by Dr. Pearce¹ which his patient developed. The Bain circuit is one in which partial rebreathing of exhaled gases occurs.² The extent of rebreathing is increased with increased minute ventilation and decreased with increased fresh gas flow.²

In the Clinical Report, prior to insufflation of the peritoneal cavity with CO₂, the ventilation and fresh gas flow were sufficient to maintain the PETCO₂ at 32 mmHg. These same variables, when confronted by the added CO₂ load after insufflation of the abdomen, permitted the PETCO₂ to rise to 45 mmHg. Dr. Pearce increased the minute ventilation by 122% (from 600 ml × 12 bpm to 800 ml × 20 bpm) but only increased the fresh gas flow 33% (from 4.5 L · min⁻¹ to 6 L · min⁻¹). The PETCO₂ increased to 60 mmHg during the next 50 min when N₂O was discontinued and O₂ was continued at 5 L · min⁻¹. With further "rapid manual ventilation" the PETCO₂ climbed to 85 mmHg and the PaCO₂ was measured at 100 mmHg. Probably this further increased PETCO₂ was caused by additional minute ventilation from "rapid manual ventilation" in the face of the reduced fresh gas flow at 5 L · min⁻¹.

In the discussion of this case, Dr. Pearce recommended an increase of ventilation as an intervention in hypercarbia. He comments that, in retrospect, the fresh gas flow should have been higher to be more effective. Certainly, for an increased CO₂ load, an increase in minute ventilation is required to maintain normocarbida. However, when the Bain circuit is used, any increase in minute ventilation must be counteracted by an increase in fresh gas flow to ensure that rebreathing is minimized.

Subcutaneous CO₂ could contribute to an added CO₂ load in the immediate postoperative period and result in prolonged return of PETCO₂ to normal values because of slower absorption from the subcutaneous sites. However, we believe that the combination of increased minute