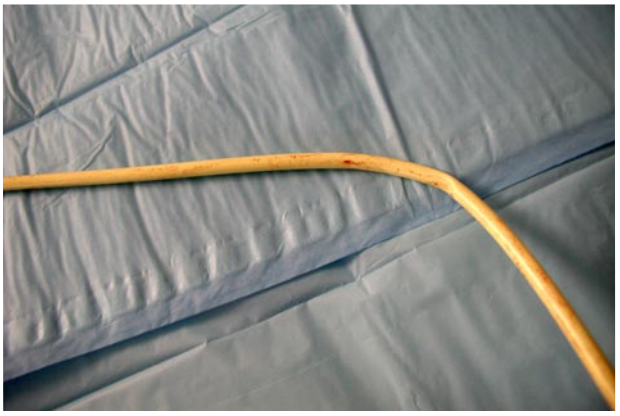

FIGURE A kink in the airway exchange catheter discovered after removal from the airway.

of little use at this point, it was removed along with the MLT® tube, in the presence of the anesthesia team prepared with both a GlideScope® (Saturn Biomedical Systems, Burnaby, BC, Canada) and a fibreoptic bronchoscope and with the surgeon at the bedside. Immediately following removal of the AEC and MLT® from the patient's trachea, her stridor and oxygen saturation rapidly improved and her respiratory effort, blood pressure, and heart rate returned to baseline values. Inspection of the AEC revealed a kink close to the distal end of the catheter (Figure).

Airway exchange catheters are important adjuncts in management of the difficult airway.[1] However, as with most airway devices, they have limitations.[2] In our case, an 11 F AEC was used to minimize obstruction to her already compromised subglottic region. Airway exchange catheters are used to facilitate the reintubation, and to allow for temporary ventilation should reintubation attempts fail.[3] In this patient, however, the AEC reduced the effective subglottic cross-sectional area, increasing the patient's work of breathing and exacerbating her inspiratory stridor. Taping the AEC to the patient's shoulder, in an effort to improve the mask seal while delivering the Heliox mixture, may have resulted in its kinking by creating torsion and changing the natural curvature of this device..

One advantage of the AEC is that this device provides a conduit to allow temporary ventilation in case of an airway emergency.[3] However, in patients with a compromised airway, the use of jet ventilation through this device may be contraindicated since the egress of air may be impaired, increasing the risk of barotraumas.[4,5] Finally, use of a bag-valve device connected to the 11 F AEC may have provided inadequate ventilation in this patient in view of her obesity. Clearly, this approach would only be considered appropriate as a last resort, while a definitive airway was being established.

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Hemoglobin M variant and congenital methemoglobinemia: methylene blue will not be effective in the presence of hemoglobin M

To the Editor:
Hemoglobin M (HbM) Saskatoon is inherited in an autosomal dominant pattern and is associated with congenital methemoglobinemia.[1] In HbM Saskatoon, histidine is replaced by tyrosine at the 63rd amino acid sequence in the β chain of the hemoglobin molecule. Pulse oximeters generate erroneous values in the presence of methemoglobin (MetHb)[2,3] and co-oximeters can be misleading due to HbM.[1] We wish to highlight potential new problems involved in anesthetizing a patient with HbM.

A 60-yr-old lady with HbM Saskatoon and congenital methemoglobinemia was scheduled for mastectomy. Clinical examination confirmed cyanosis but she had no symptoms due to methemoglobinemia and no history suggestive of cardiac or respiratory problems. Her electrocardiogram was normal and arterial blood gas (ABG) values on room air were: pH = 7.52, pO_2 = 104 mmHg, pCO_2 = 28 mmHg, BE = 0.9, lactate – within normal limits. We could not obtain details regarding oxyhemoglobin (oxyHb), reduced hemoglobin and MetHb from our co-oximeter. As the patient had good exercise tolerance and the normal lactate indicated adequate tissue perfusion[4] we planned to proceed with surgery. We monitored the patient by measuring serial ABGs through an indwelling arterial line in addition to routine monitors. Anesthesia was induced with fentanyl and propofol. A spontaneously breathing technique with a 1:1 mixture of oxygen/air and isoflurane was used. Intraoperatively, a representative ABG was as follows: pH = 7.47, pO_2 = 375 mmHg, pCO_2 = 32 mmHg, BE = 0.2; lactate = 1.1 $mmol \cdot L^{-1}$. The perioperative course was uneventful and the patient was discharged home two days later.

Severe MetHb is treated with methylene blue which rapidly reduces methemoglobinemia nonenzymatically.[3] The recommended dosage of methylene blue is 1–2 $mg \cdot kg^{-1}$ iv over a five-minute period. Doses > 15 $mg \cdot kg^{-1}$ can paradoxically cause methemoglobinemia.[1] It is known that methylene blue is ineffective in the presence of nicotinamide-adenine dinucleotide phosphate dehydrogenase and glucose-6 phosphate dehydrogenase deficiencies.[1,3] Methylene blue is not effective in the presence of HbM because HbM leads to the formation of an iron-phenolate complex that resists reduction. Co-oximeters are also misleading in the presence of HbM.[1] The HbM spectrum lacks the characteristic MetHb peak at 630 nm and has a peak near 600 nm. In the presence of HbM, co-oximeters may report normal fractions of MetHb, increased carboxyhemoglobin (COHb) or increased sulfhemoglobin.[1] Our ABG machine (Roche Omni S, Cambridge, UK) failed to record all types of hemoglobin (oxyHb, reduced Hb, MetHb and COHb). We raised this issue with our biochemistry department whose staff concluded that the HbM Saskatoon interfered with the algorithm of the co-oximeter and hence failed to record any form of hemoglobin.

The issues raised by this case include failure of methylene blue to treat methemoglobinemia in the presence of HbM and interference of HbM Saskatoon with the algorithm of the co-oximeter. In severe cases of methemoglobinemia with HbM, exchange transfusion or even hyperbaric oxygen[5] may have to be considered. The role of ascorbic acid in managing such cases remains uncertain.

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Adie syndrome associated with general anesthesia

To the Editor:
The sudden onset of anisocoria indicates the possibility of a life-threatening intracranial lesion. We describe a case of anisocoria which occurred in association with a hypertensive episode after anesthetic induction. Adie syndrome was subsequently diagnosed by a pilocarpine test.

A 61-yr-old previously healthy female was scheduled for a lung biopsy. She had no history of any ophthalmologic disorder, and she had no pupillary abnormalities preoperatively. Diazepam 5 mg *po* and pentobarbiturate 50 mg *po* were administered as premedicants 90 min before surgery. In the operating room a thoracic epidural was inserted following application of routine monitors. After documenting sensory loss in the T3-L1 dermatomes in response to 5 mL of epidurally injected 1.5% mepivacaine, anesthesia was induced with propofol 180 mg *iv* followed by vecuronium 6 mg *iv*. Immediately after laryngoscopy and tracheal intubation, the patient's blood pressure increased suddenly to 205/120 mmHg. Following several minutes of controlled ventilation with 3% inspired sevoflurane, the patient's heart rate