Potentially fatal vaporizer incident
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EDITOR:
We wish to report a potentially fatal critical incident that occurred during an elective operating theatre list. A similar problem [1] has been recently highlighted by the MHRA but without the emphasis of its potentially fatal nature (Fault in Draeger Vapor 2000/D-Vapor Vaporizer series with the interlocking device, MDA/2006/030) [2].

This series of vaporizers for isoflurane and sevoflurane has an interlocking safety system consisting of a hole with a nib in the coloured disk on the top of the vaporizer, at 180° to each other. This prevents the locking bar being inserted into the hole unless the vaporizer is turned off. It is known that damage to or loss of this plastic nib can result in two vaporizers being turned on simultaneously [3]. The presence of a unilateral nib at the 5% delivery mark masks the absence of the second nib at the 0% delivery mark due to the nature of the vaporizer position on the anaesthetic machine at the left-hand side (Fig. 1). This machine thus passed the routine AAGBI Recommendations for Checking Anaesthetic Equipment [4].

On transfer of the patient to the operating room, initially isoflurane was switched on to 1–2%. The desflurane vaporizer was then mounted as the anaesthetist’s agent of choice and switched on after switching off the isoflurane vaporizer – or so we thought. What happened next is shown in Figure 2. The vaporizer on the left is the isoflurane vaporizer at 5% isoflurane. This was caused when the anaesthetist inadvertently turned the vaporizer dial anticlockwise to switch off instead of in a clockwise direction (i.e. the wrong way) and engaged the interlocking device, which then allowed operation of the desflurane vaporizer. We recognized this mistake a few seconds after it occurred preventing any harm to the patient. The interlocking device required considerable force to disengage from the isoflurane vaporizer.

Two issues need to be addressed:
1. Awareness of this potential error as the number of general anaesthetics given daily with these agents is large, increasing the chances of a similar episode that might not be corrected so speedily.
2. The manufacturer needs to urgently rectify this problem.
This incident emphasizes the fact that despite repeated checking, errors can and do occur and it is necessary to be vigilant for new errors as our anaesthesia delivery systems become more complicated and modernized. We have informed Draeger and look forward to their comments.

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References

Metformin-associated lactic acidosis following contrast media-induced nephrotoxicity
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EDITOR:
Metformin-associated lactic acidosis (MALA) has an incidence of 0.03 per 1000 person years with a mortality rate of about 50% [1]. In a neurosurgical ward, patients with aneurysmal subarachnoid haemorrhage (SAH) may be admitted in an emergency unit requiring early diagnosis and management. Patients on multiple drug therapy require consideration, as the current trend is towards early surgical intervention in treating SAH. Recently, we encountered MALA in a patient with aneurysmal SAH in whom lactic acidosis was precipitated by contrast media-induced nephrotoxicity despite normal renal function before operation.

Case report
A 47-yr-old male, weighing 67 kg, was admitted to the neurosurgical ward with a 2-day history of severe headache and transient loss of consciousness on the previous day. His medical history was significant for Type II diabetes mellitus over the previous 3 yr. Blood glucose concentration was well controlled with glyburide 5 mg and metformin 500 mg twice a day. All routine investigations were normal. Blood was seen in the interhemispheric fissure and basal cistern on a computed tomographic (CT) head scan. A diagnosis of SAH Grade III was made. The patient was taken for emergency digital subtraction angiography under monitored anaesthesia care. It showed aneurysms at the right middle cerebral artery bifurcation and the left anterior communicating artery. Emergency craniotomy and clipping of the aneurysm was planned. Although a dose of metformin had been taken by the patient just before coming to hospital, in view of the emergency nature of the surgery, the neurosurgeon decided to proceed. The anaesthetic and surgical course was uneventful. For the next 12 h, the patient’s general condition remained stable but the blood sugar had risen to 16.2 mmol L⁻¹ despite insulin infusion at the rate of 6 units h⁻¹. The patient became irritable and tachypnoeic. Arterial blood gas analysis showed pH 7.26, PaCO₂ 2.84 kPa, PaO₂ 11.43 kPa, HCO₃⁻ 12 mmol L⁻¹, base excess -10, anion gap 29 and lactate 5 mmol L⁻¹. There were no ketone bodies in the urine. Serum osmolality was 283 mOsm kg⁻¹. A transcranial Doppler examination showed normal blood flow velocities in all major blood vessels. One hour later, the urine output had decreased to 20 ml h⁻¹ despite the administration of adequate intravenous (i.v.) fluids, maintenance of haemodynamic parameters and central venous pressure 16 cm H₂O. Furosemide 40 mg was administered i.v. but the urine output did not improve. Blood gas analysis now showed a metabolic acidosis with pH 7.258, PaCO₂ 2.34 kPa, PaO₂ 12.38 kPa, HCO₃⁻ 7.6 mmol L⁻¹, Na 131 mmol L⁻¹, K 3.7 mmol L⁻¹, Cl 96 mmol L⁻¹, base excess -14, anion gap 27.4 and lactate 7.3 mmol L⁻¹. Serum creatinine was