A non-airway management use of the video laryngoscope (GlideScope®)

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EDITOR:
We report a case in which we used the GlideScope® to facilitate the successful insertion of nasogastric tube.

A 66-yr-old male was scheduled for an elective left-sided hemi-mandibulectomy with radial free flap reconstruction for squamous cell carcinoma of the oral cavity. On assessment, the patient's airway was Mallampati 2 and mouth opening was three finger-breadths wide. We induced anaesthesia and intubated the trachea (Cormack & Lehane grade 2 on direct laryngoscopy). We attempted and failed to insert a nasogastric tube with digital manipulation as well as under direct vision with a MacIntosh blade. At this point, we inserted the video laryngoscope (GlideScope®). The view was Cormack & Lehane grade 1 of the laryngeal inlet with an endotracheal tube *in situ*. We lifted the epiglottis with the GlideScope tip, which improved the view of entrance to the oesophagus. We could insert the nasogastric tube under direct vision with digital manipulation. The position was confirmed with gastric contents on aspiration and the appropriate pH of the aspirate.

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The GlideScope has been designed to facilitate tracheal intubation by achieving a clear view of the anterior segment of the glottis without the need for a direct line sight. Even in difficult intubations, the GlideScope achieves Cormack & Lehane view I and II in 99% of patients [1]. It requires less force than conventional laryngoscopy, hence it is less traumatic and minimizes the laryngoscopic stress response. Its slim blade provides a good working space not only for intubation, but also for nasogastric tube placement. It is easy to learn, use and master the technique.

Even though there are no clinical trials available to support this use, it may be a useful technique to use the GlideScope to insert a nasogastric tube, especially in intubated patients. It may also theoretically reduce the stress response to laryngoscopy as it requires less force than the traditional laryngoscope.

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Lactate gap and ethylene glycol poisoning

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EDITOR:
We assayed lactate levels in plasma using point-of-care analysers and obtained a fallaciously high value when compared to the value obtained from the central laboratory. The divergence in the lactate values suggested the possibility of ethylene glycol poisoning, but due to the limited information valuable time was lost in initiating treatment.

A 36-yr-old male was brought to the hospital in an unconscious state with a core temperature of 32.9°C. Glasgow coma score on arrival in the accident and emergency department was 3. Clinical evaluation and an urgent computed tomography (CT) scan of the head ruled out intracranial pathology. The patient was moved to the critical care unit for further management.

Blood gas analysis breathing 50% oxygen showed a pH of 6.8, PCO_2 1.4 kPa, PO_2 34 kPa, HCO_3^- 1.0 mmol L^{-1}, base excess −26 mmol L^{-1} and lactate 33 mmol L^{-1}. Routine blood tests showed a lactate of 15.8 mmol L^{-1}. Past medical history revealed a suicidal tendency with previous admission to the hospital with paracetamol overdose. Toxicology screening was sent. A portable ultrasound of the abdomen was performed and contrast CT was planned to rule out intra-abdominal conditions. Fluid resuscitation,

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haemodynamic support, respiratory support and later renal replacement therapy were initiated. There was no improvement in acidosis despite treatment.

All the arterial blood gas samples analysed from the critical care unit using a Radiometer ABL 725 (Radiometer Medical A/S, Bronshoj, Denmark) blood gas analyser showed consistently high levels of lactate despite a sodium bicarbonate infusion and haemofiltration. Thus the cause of the metabolic acidosis remained obscure. The lactate levels from the clinical chemistry laboratory taken at the same time did not match those from the ICU analyser. These differing results aroused the suspicion of ethylene glycol poisoning and further samples were sent for methanol/ethylene glycol detection to the regional toxicology laboratory. It was not before another 6 h that 333 mg L^{-1} of ethylene glycol was detected in the blood and appropriate treatment (4-Methylpyrazole) was instituted. Within an hour of this treatment the acidosis began to improve and the cardiovascular support could be reduced. The patient's condition progressively improved and the lactate had fallen to 0.5 mmol L^{-1} by the 4th day. He was discharged to a tertiary care unit for further management of his renal failure.

We analysed some samples of plasma with a known quantity of glycolic acid and compared the results obtained from our ICU blood gas analyser with those from our central clinical chemistry laboratory (Cobas Integra 400 plus analyser; Roche Diagnostics, Basel, Switzerland). The levels of glycolic acid added and the discrepancies in the two sets of observations are shown in Table 1. This clearly demonstrates the lactate gap in all the readings, which increases linearly as the glycolic acid levels in plasma increased.

The delay in diagnosing this case of ethylene glycol poisoning could have been possibly averted by awareness of this artifactual elevation of lactate levels by our point-of-care analyser and facilities for the prompt detection of ethylene glycol in blood. The lactate gap has been described as divergent lactate levels obtained from a single sample when measured using two different modalities. Recently an erroneous reading by a Radiometer 700 analyser (Radiometer Limited, Crawley, UK) resulted in the patient having an emergency laparotomy [2]. Our patient presented to us approximately 12 h after ingestion of anti-freeze, an interval corresponding with high levels of toxic metabolites. Clearly, ethylene glycol metabolites were causing falsely elevated lactate levels. This was attributed to the large dose of ethylene glycol consumed and delay in the time of arrival at the hospital.

The turning point in the diagnosis and subsequent management in our case were two different lactate values measured from the analyser in our critical care unit and that in the clinical chemistry laboratory. The method for lactate measurement in the latter equipment utilises a lactate oxidase method [3]. This enzyme converts lactate to pyruvate and produces hydrogen peroxide (H_2O_2). The peroxide reacts with 4-aminoantipyrine and other unspecified reactants to form a coloured product that is quantified colorimetrically. This method has the practical advantage of having improved reagent stability when compared to alternative methods based on lactate dehydrogenase. However, lactate oxidase may be less specific for the substrate lactate than lactate dehydrogenase. The L-lactate analyser such as that in our ICU, which is widely used to monitor lactate levels in critical care units, measures lactate using the L-lactate oxidase method. However, it uses an electrochemical principle. Lactate determination is accomplished by the enzymatic reaction of lactate oxidase and the detection of H_2O_2 [4]. It seems that most lactate oxidase-based systems respond to glycolate. The difference in response probably depends on the way in which the reaction is monitored. The false-positive results from the ICU equipment occur because ethylene glycol metabolites are substrates for L-lactate oxidase. In contrast, ethylene glycol metabolites cause minimal lactate elevation with the Bayer, iSTAT and Vitros devices [3].

Metabolites of ethylene glycol cause a worse acidosis than the parent compound itself. These may continue to remain in blood for a variable period of time. Increased glycols are measured as lactate in blood [1]. However, despite the equipment being used so frequently for blood gas analysis in the critical care unit, it has not been an object of attention for its erroneously high lactate readings in the presence of glycolic acid. Glycolic acid may account for as much as 96% of the anion gap in patients poisoned with ethylene glycol [5]. Point-of-care test systems may not mark the reaction course as atypical or erroneous. Therefore, having

Table 1. Glycolic acid measured as lactic acid.

Glycolic acid	Radiometer ABL725 lactate reading	Cobas Integra lactate reading
0	1.0	1.1
3	6.9	2.6
6	12	4.2
9	18	6.0
12	24	7.2
18	34	10.2
24	43	12.6
30	51	14.9

A known concentration of glycolic acid was added to a sample of plasma and then measured as lactate by the two systems. Data are all mmol L^{-1}.

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the local laboratory check a high lactate value is prudent, particularly if the diagnosis is not firmly established. In this regard it is useful to have previously established the response to glycolate of both point-of-care and laboratory systems. This case has highlighted the fact that more understanding of equipment and its mechanisms of action are critical in interpreting the data.

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Low bispectral index values in a 2-yr-old with a large bifrontal porencephalic cyst

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EDITOR:
Bispectral index (BIS) is routinely used as a monitor of depth of anaesthesia. Unusually low BIS values may be seen during cerebral ischaemia [1], in neuroradiology during glue embolization [2], in patients with dementia [3] and in persons with genetically determined low-voltage electroencephalographic (EEG) signals [4]. Recently, we detected persistently low BIS values in a patient diagnosed with large porencephalic cysts in the frontal lobes.

A 2-yr-old male child, weighing 12 kg, was admitted to our neurosurgical ward with major complaints of increasing head size since 4 months of age, two episodes of generalized tonic-clonic seizures a week ago and delayed milestones. The child responded to commands, recognized his mother but had difficulty in holding his head with a tendency to fall while walking. Contrast-enhanced computed tomography showed two large cystic intraparenchymal lesions in the frontal lobe, suggestive of porencephalic cysts with paucity of periventricular matter and prominent ventricles. Magnetic resonance imaging showed bilateral communicating frontal lobe porencephalic cysts with secondary aqueductal stenosis and secondary corpus callosal hypoplasia (Fig. 1). He was scheduled for an elective frontal craniotomy and fenestration of frontal porencephalic cyst with right cystoperitoneal shunt. In the operating theatre, routine monitors (electrocardiogram, non-invasive blood pressure and pulse oximetry) were attached. A BIS monitor (A-2000; Aspect Medical Systems, Newton, MA, USA) was also attached. The BIS sensor (paediatric) was applied to the forehead and left temporal area. A low BIS value of 39 was observed. Lower values persisted till induction of anaesthesia. General anaesthesia was induced with thiopental sodium 40 mg after fentanyl 20 μg. Rocuronium 10 mg facilitated tracheal intubation. Anaesthesia was maintained with sevoflurane in an oxygen and nitrous oxide mixture (1 : 2). Intermittent boluses of fentanyl and rocuronium were given to facilitate mechanical ventilation to target an end-tidal carbon dioxide value of 32 ± 1 mmHg. Throughout the surgical procedure, the BIS values remained between 30 and 40 with a signal-quality index (SQI) of more than 90%. Due to the low BIS values, the sevoflurane concentration was reduced from inspired concentration of 1–0.2%. Still, no changes in the BIS values were observed. No burst and suppression pattern was observed and the electromyography value remained below 30. At the end of an uneventful surgery, the anaesthetics were discontinued and neuromuscular block reversed. The trachea was extubated and the child cried actively. The BIS value continued to remain low, even 6 h later in the neurosurgical ICU.

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