proportion of dogs given lethal doses of air with a multi-orificed catheter placed in the mid-atrium. The catheter tip can be placed using ECG guidance, as described by Colley and Artru subsequently [15], thus avoiding the need to irradiate the patient.

Our case highlights a rare complication of anaesthesia and surgery in the prone position. It demonstrates that it is possible for VAE to present late, even after open vessels have been closed. It may present following a change in patient position. We suggest that VAE occurring in the prone position is under detected and consequently under reported. We advocate increased awareness and monitoring in all cases where the operative site is above the level of the heart, and the placement of a CVC in cases that, in light of the discussion above, could be considered to be high risk.

S. F. McDouall
Department of Anaesthetics
Heatherwood and Wexham Park Hospitals NHS Trust
Wexham, Slough, UK

D. Shlugman
Department of Anaesthetics
John Radcliffe Hospital
Headington, Oxford, UK

References

1. Albin MS, Carroll RG, Maroon JC. Clinical considerations concerning detection of venous air embolism. *Neurosurgery* 1978; **2**: 380–384.
2. Palmon SC, Moore LE, Lundberg J, Toung T. Venous air embolism: a review. *J Clin Anesth* 1997; **9**: 251–257.
3. Lang SA, Duncan PG, Dupuis PR. Fatal air embolism in an adolescent in Duchenne muscular dystrophy during Harrington instrumentation. *Anesth Analg* 1989; **69**: 132–134.
4. Sutherland RW, Winter RJ. Two cases of fatal air embolism in children undergoing scoliosis surgery. *Acta Anesthesiol Scand* 1997; **41**(8): 1073–1076.
5. McCarthy RE, Lonstein JE, Mertz JD *et al*. Air embolism in spinal surgery. *J Spinal Disord* 1990; **3**(1): 1–5.
6. Horlocker TT, Wedel DJ, Cucchiara RF. Venous air embolism during spinal instrumentation and fusion in the prone position. Letter to the editor. *Anesth Anal* 1992; **75**: 152.
7. Frankel AS, Holzman RS. Air embolism during posterior spinal surgery. *Can J Anaesth* 1988; **35**(5): 511–514.
8. Albin MS, Ritter RR, Pruett CE, Kalff K. Venous air embolism during lumbar laminectomy in the prone position: report of three cases. *Anaesth Analg* 1991; **73**: 346–349.
9. DiStefano VJ, Klein KS, Nixon JE, Andrews ET. Intraoperative analysis of the effects of position and body habitus on surgery of the low back. *Clin Orthop* 1974; **99**: 51–56.
10. Hagen PT, Scholz DG, Edwards WD. Incidence and size of patent foramen ovale during the first 10 decades of life: an autopsy study of 965 normal hearts. *Mayo Clin Proc* 1984; **59**: 17–20.
11. Butler BD, Hills BA. Transpulmonary passage of venous air emboli. *J Appl Physiol* 1985; **59**: 543–547.
12. Marquez J, Sladen A, Gendell H *et al*. Paradoxical cerebral air embolism without an intracardiac septal defect. *J Neurosurg* 1981; **55**: 997–1000.
13. Bunegin L, Albin MS, Helsel PE *et al*. Positioning the right atrial catheter: a model for reappraisal. *Anesthesiology* 1981; **55**: 343.
14. Colley PS, Artru AA. Bunegin–Albin cateter improves air retrieval and resuscitation from lethal air embolism in dogs. *Anesth Analg* 1987; **66**: 991–994.
15. Artru AA, Colley PS. Placement of multiorificed CVP catheters via antecubital veins using invasive electrocardiography. *Anesthesiology* 1988; **69**: 132–135.

Acetone poisoning – a diagnostic dilemma

doi: 10.1017/S0265021507000245

EDITOR:

Acetone intoxication is a rarely reported substance involved in poisoning [1–3]. A literature search indicated that, in all cases reported, a clear history of acetone ingestion was obtained, thus facilitating management. We present a case where this information was not immediately available, and subsequent difficulties presented in the diagnosis and management.

Case report

A 47-yr-old female presented to A&E with respiratory distress. She had a past history of alcohol abuse and deliberate self-harm and was receiving citalopram for depression. She had no other medical problems of

Correspondence to: Veerappan Kumarvel, Department of Anaesthetics & Intensive Care, Maidstone Hospital, Hermitage Lane, Maidstone, Kent ME16 9QQ, UK. E-mail: seethakumarvel@hotmail.com; Tel: +1622 670554; Fax: +1622 224669

Accepted for publication 23 December 2006 EJA 4244
First published online 11 April 2007

note. On physical examination, she had a slim build and was drowsy but rousable with a Glasgow coma scale of 12/15 and no focal neurological signs. She had a sinus tachycardia of 120 min^{-1} and a blood pressure of 180/120 mmHg. She had a respiratory rate of 40 min^{-1} with a fruity odour on her breath and S_PO_2 of 94% on air. Chest was clinically and radiologically clear with no signs of cardiac, hepatic or renal failure. She was hypothermic (35°C).

Arterial blood gas analysis showed pH 6.9, $PaCO_2$ 1.4 kPa, PaO_2 18.5 kPa and base excess −28.8 mmol L^{-1}. There was negative toxicology screen involving ethanol, salicylate, paracetamol and barbiturates. The blood glucose was 15 mmol L^{-1} and a peripheral blood smear showed neutrophil leucocytosis and white cell count of 26×10^9 L^{-1}. The calculated serum osmolarity was 288 but the measured osmolality was 310, giving an osmolar gap of 22. While the anion gap was 38, the blood lactate level was normal and urinanalysis was mildly positive for ketones. She did not require ventilation, but a bolus of intravenous (i.v.) sodium bicarbonate was administered, and she was transferred to intensive treatment unit (ITU) for further supportive care and management of her profound metabolic acidosis by haemofiltration.

The National Toxicology Centre was contacted and suggested methanol or ethylene glycol, but not acetone as a potential aetiological agent. Treatment with i.v. ethanol was recommended. Gas chromatography of the patient's urine and blood confirmed the presence of acetone. The acidosis improved within hours of haemofiltration and i.v. ethanol was not administered. The osmolar gap, anion gap and pH were completely corrected by 18 h. She was transferred out of ITU after 30 h and subsequently sent home after a few days.

Discussion

Alcohol misuse is associated with concurrent intentional ingestion of acetone as well as methanol, ethylene glycol and acetonitrile (a common component of cosmetic nail varnish remover) [4]. In all of these, the presenting laboratory features of acidosis, with a high anion and osmolar gap, are similar and non-specific. Though acetone alone is a relatively non-toxic compound, methanol and ethylene glycol are converted to the highly toxic breakdown product formaldehyde, and acetonitrile is metabolized to cyanide. The toxicity of acetone is identical to ethanol, although the anaesthetic potency of acetone is much greater [5]. The main route of elimination of acetone is through the lungs. Both acetone and ethanol produce respiratory failure due to central nervous system depression in toxic doses [5]. The elimination of both at high doses follows zero order kinetics. The few reported signs of acetone poisoning, which may help to clinically differentiate ethanol from acetone ingestion, include red and swollen pharynx and soft palate erosions [5,6], but were not seen in this patient. It is likely that the acetone was diluted with another liquid and probably taken over a period of time. Acetone is metabolized to glucose [5,6], and the high blood sugar levels and ketonuria were in keeping with this. Diabetic keto-acidosis was considered as a possible diagnosis but was not in keeping with the overall clinical picture. Measurement of serum osmolality and osmolar gap have limited sensitivity and specificity. Gas chromatography, if available, offers the possibility of a definitive diagnosis.

This case illustrates several interesting points: acetone containing nail polish removers are readily found at home, and acetone ingestion should be kept in mind when considering possible aetiological agents. Unless there is a clear history, it can be very difficult to distinguish clinically between all these agents. Finally, the recommended treatment of methanol, as well as ethylene glycol poisoning, is 100% ethanol. Ethanol administration will exacerbate acetone toxicity. It is important to consider acetone in the differential diagnosis of unexpected metabolic acidosis, especially in the presence of hyperglycaemia.

V. Kumarvel, J. Da Fonseca
Department of Anaesthetics & Intensive Care
Maidstone Hospital
Maidstone
Kent, UK

References

1. Gamis AS, Wasserman GS. Acute acetone intoxication in a pediatric patient. *Pediatr Emerg Care* 1998; 4(1): 24–26.
2. Ramu A, Rosenbaum J, Blaschke TF. Disposition of acetone following acute acetone intoxication. *Western J Med* 1978; **129**(5): 429–432.
3. Kostusiak V, Bekkal R, Mateu P. Survival after drinking lethal dose of acetone. *Intensive Care Med* 2003; **29**: 339.
4. Rainey PM, Roberts WL. Diagnosis of poisoning with the cyanide precursor acetonitrile: nail polish remover or nail glue remover? *Am J Emerg Med* 1993; **11**(2): 104–108.
5. Winchester JF. *Clinical Management of Poisoning and Drug Overdose*, 3rd edn. Philadelphia: W.B. Saunders Company, 1998.
6. Gitleson S, Werczberger A, Herman JB. Coma and hyperglycemia following drinking of acetone. *Diabetes* 1966; **15**: 810–811.

https://doi.org/10.1017/S0265021507000245 Published online by Cambridge University Press