A 50-yr-old male (BMI 23 kg m⁻²), with a history of spontaneous pneumothorax (PTX) 25 yr before. He was first treated by the emergency services due to severe hypoxia after a large left PTX after an acute coughing period with the need of orotracheal intubation and a chest drainage insertion. At the peripheral hospital, the ECMO team positioned an internal right jugular double-lumen cannula, and transferred the patient to our ICU. The patient responded to ECMO therapy but, at day 10, the day before the scheduled ECMO removal, the patient complained of low back pain, and haemoglobin decreased by 2 g dL⁻² before the scheduled ECMO removal, the patient developed multiple organ failure and disseminated intravascular coagulation and died on day 26.

The second patient was a 50-yr-old male (BMI 23 kg m⁻²), with a history of spontaneous pneumothorax (PTX) 25 yr before. He was first treated by the emergency services due to severe hypoxia after a large left PTX after an acute coughing period with the need of orotracheal intubation and a chest drainage insertion. At the peripheral hospital, the ECMO team positioned an internal right jugular double-lumen cannula, and transferred the patient to our ICU. The patient responded to ECMO therapy but, at day 10, the day before the scheduled ECMO removal, the patient complained of low back pain, and haemoglobin decreased by 2 g dL⁻². A CT scan revealed bilateral iliopsoas haematoma without active bleeding, and surgical indications were not given. ECMO removal was then anticipated. During the next days, disseminated intravascular coagulation made the clinical status worse, and respiratory function was complicated by bilateral spontaneous PTX. Death occurred on day 20.

Iliopsoas haematoma is a rare, but potentially life-threatening, complication of anticoagulant therapy (warfarin or heparin). Common clinical signs of iliopsoas haematoma are groin pain or leg weakness, due to the involvement of the femoral nerve.³ The role of surgical or conservative treatment of iliopsoas haematoma during anticoagulation therapy is not clear.⁴ Few reports are available in medical literature,³⁴ and only one on bilateral haematoma after therapy with heparin.⁵ In our experience, the incidence of this complication is about 3%, but this must be put in the context of a single-centre experience.

It is of interest that, in both cases, activated partial thromboplastin time remained in the therapeutic range (40–60 s, bedside measured every 2 h) and no significant haemostatic changes were identified. Femoral ECMO cannula cannot be considered an absolute risk factor, since in the second patient (bilateral haematoma), the ECMO support used a single double-lumen cannula in the jugular vein. Even BMI cannot be considered a risk factor, since the patients were of very different size.

Despite the fact that during the first season of H1N1 pandemic (2009–10), we experienced a low complication rate and high survival rate in H1N1 patients treated with ECMO support,⁶ during the second pandemic season (2010–1), we observed resistance of the virus to antiviral treatment, with a longer average time of ECMO (14 vs 7 days, respectively). At this stage of ECMO, patients are usually awake and starting physiotherapy to promote lung recovery. Thus, spontaneous movements may have promoted the formation of an iliopsoas haematoma.

Enhanced elimination of phenobarbital using charcoal haemoperfusion in a patient with severe poisoning

Editor—We present data from a patient with severe phenobarbital poisoning who we treated with charcoal haemoperfusion and multiple-dose activated charcoal (MDAC). A 66-yr-old male naïve to phenobarbital therapy was brought to hospital by ambulance, intubated, and manually ventilated, after intentional ingestion of 6 g of phenobarbital. He had a background of unremitting chronic abdominal pain, ischaemic heart disease, hypertension, and congenital partial atonic bowel. Time of ingestion was unclear but <5 h before. Signs at presentation were ventilatory frequency 12 bpm, pulse oximetry 100% (FiO₂, 100%), hypotension (88/58 mm Hg), heart rate 64 beats min⁻¹, Glasgow coma score 3/15, miosis (2 mm bilaterally), areflexia, and hypothermia (33.1 °C). An arterial blood gas (FiO₂, 100%) demonstrated pH 7.29, PaO₂ 72 kPa, PaCO₂ 7 kPa, and lactate...
1.8 mmol litre\(^{-1}\). I.V. normal saline and epinephrine infusions were commenced and a warming blanket was applied. A nasogastric tube was inserted and normal bowel sounds were heard, allowing for the commencement of MDAC 25 g every 2 h. The patient was transferred to the intensive care unit (ICU).

Initial phenobarbital concentration was 409 \(\mu\)mol litre\(^{-1}\) (reference range 65–170 \(\mu\)mol litre\(^{-1}\)) and with serial measurements over the following 7 h, it did not decrease (Fig. 1). In anticipation of a prolonged ICU admission, concern over use of an epinephrine infusion in a patient with coronary artery disease, and potential suboptimal response to MDAC, given the history of partial atonic bowel, charcoal haemoperfusion was commenced. This was performed using a PrismaFlex machine with a Gambro Adsorba 30C cartridge and blood flow 300 ml min\(^{-1}\) for 5 h.

Approximately 1 h after starting haemoperfusion, the patient became hypertensive, so epinephrine was stopped and a glyceryl trinitrate infusion commenced. Over the next hour, spontaneous respiratory effort returned. At the conclusion of the 5 h treatment, the serum phenobarbital concentration was 214 \(\mu\)mol litre\(^{-1}\). Metabolic disequilibria complicating the treatment included hypomagnesaemia 0.55 mmol litre\(^{-1}\), hypocalcaemia 1.55 mmol litre\(^{-1}\), hypophosphataemia <0.3 mmol litre\(^{-1}\), and thrombocytopenia 68 \(\times\) 10\(^9\) litre\(^{-1}\), which were readily corrected. A rebound increase in serum phenobarbital concentration did not occur.

Treatment with MDAC continued overnight and the apparent elimination half-life of phenobarbital was 19.1 h. Progressive clinical recovery was observed, including opening his eyes and spontaneous movement of four limbs. The patient was alert 24 h after stopping haemoperfusion, allowing for weaning of MDAC the following day. Extubation and cessation of mechanical ventilation followed, 59 h after admission. He continued to make rapid improvement thereafter and was discharged from ICU after 77 h.

Using multiple paired blood samples, the initial extracorporeal phenobarbital clearance was calculated to be 163 ml min\(^{-1}\), a potentially 40-fold increase in endogenous clearance,\(^1\) but this decreased progressively with saturation of the charcoal cartridge, amounting to only 64 ml min\(^{-1}\) at the conclusion of the treatment. The total amount of phenobarbital removed by haemoperfusion was calculated to be 2.3 g, more than 30% of the reported exposure.

This case documents apparent clinical and pharmacokinetic benefits from enhanced elimination in the treatment of severe acute phenobarbital poisoning. Improvements in respiratory and haemodynamic status were apparent within hours of treatment. In contrast, intoxication may persist for many days in patients with phenobarbital poisoning who do not receive these treatments.\(^1\) These clinical benefits most likely relate to the marked increase in phenobarbital clearance by haemoperfusion and MDAC. This is possible because phenobarbital has a small volume of distribution, minimal protein binding, and low endogenous clearance.\(^1\) On the basis of the limited literature available, it is not possible to confirm that charcoal haemoperfusion improves clinical outcomes in all patients with severe poisoning.\(^2\) While this technique is less commonly used in recent times, our case demonstrates that with appropriate monitoring, it can be a safe and effective treatment.

**Conflict of interest**

None declared.

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**Do tracheal tubes prevent microaspiration?**

Editor—For decades, it was assumed that cuffed tracheal tubes protected the patient from aspiration. However, studies have demonstrated that current barrel-shaped high-volume, low-pressure cuffs allow aspiration of fluids through channels formed in the cuff.\(^3\) This leakage around the tracheal tube cuff is known to be one of the major causes of microaspiration.\(^2\)\(^3\)

A new taper-shaped cuff (TaperGuard) was recently introduced and claims that the taper shape will decrease the incidence of microaspiration, thereby protecting the lungs. The object of this study was to compare the fluid sealing performance of the new taper-shaped cuff (TaperGuard Evac, Coviden, Boulder, CO, USA) with that of the commonly used barrel-