

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 µmol litre⁻¹ (reference range 65–170 µmol 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 µmol 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 thrombocytopaenia 68 × 10⁹ 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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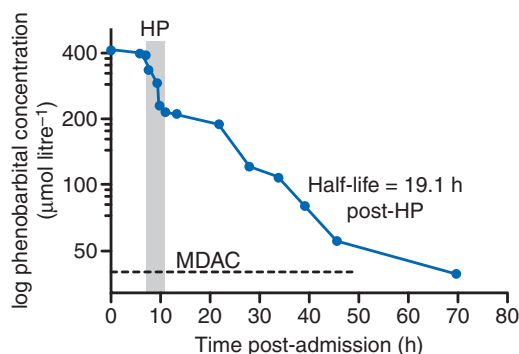


Fig 1 Serial serum phenobarbital levels according to time post-admission. HP, charcoal haemoperfusion; MDAC, multiple-dose activated charcoal.

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.¹ 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, Covidien, Boulder, CO, USA) with that of the commonly used barrel-

Table 1 Summary of tracheal tubes, cuff pressure and results

	Hi-Lo	TaperGuard	P-value
<i>n</i>	7	7	
Mean cuff pressure (cm H ₂ O)	25.2	23.7	<0.2
Tube size (mm)	5–8.5	3–7.0	
	2–9.0	2–8.5	
		2–9.0	
Dye leak	7/7	1/6	<0.005
Ulceration	5/7	1/6	<0.07
Haemorrhagic pneumonia	5/7	1/6	<0.07
Bronchitis	5/7	0/6	<0.016

shaped cuff (Hi-Lo Evac, Covidien, Boulder, CO, USA) in animals undergoing abdominal surgery.

After animal use committee approval, 14 pigs were anaesthetized and the tracheas intubated with appropriately sized Hi-Lo Evac ($n=7$) or TaperGuard Evac ($n=7$) tracheal tubes. Saline based-fluid (pH 7.5/0.3 ml kg⁻¹) stained with Methylene-Blue was instilled into the area below the vocal cords but above the inflated tracheal tube cuff via the Evac port. The cuff pressure was measured every 15 min and maintained between 25 and 30 mm H₂O for both tracheal tubes using a Posey cufflator (Posey Health Care Products, Arcadia, CA, USA). The lungs were ventilated to achieve normocarbida and each pig underwent abdominal surgery. At the end of surgery, the pigs were killed by lethal injection and the tracheal tubes were left in place with the cuffs inflated until the autopsy. At autopsy, a gross visual assessment was made of dye distribution into the trachea and lungs. Gross and histological examination of large, intermediate, and small airway mucosa and pulmonary parenchyma was performed to assess for inflammatory changes related to aspiration.

However, one pig in the TaperGuard group had to be excluded due to accidental cuff deflation. The incidence of microaspiration was significantly less for TaperGuard in the dye leak and bronchitis groups (Table 1). As a result of the dye leak from the accidental cuff deflation, the difference in the other two categories did not reach statistical significance but may be clinically significant. We suggest further animal and clinical studies to confirm our results and impressions.

Conflict of interest

P.R.L. has received travel stipend from Covidien, Boulder, CO, USA. D.M. has performed contract research for Covidien, Boulder, CO, USA. U.B. is an employee of Covidien, Boulder, CO, USA. The research project was funded by unrestricted grant from Covidien, Boulder, CO, USA.

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Frequency band of EMG in anaesthesia monitoring

Editor—In their letter, Chazot and colleagues¹ describe a spectral analysis of a frontally recorded EEG and EMG signal, and they claim that the electrical brain activity in the frequency range of 0.25–40 Hz causes the increase in bispectral index (BIS) and WAVCNS indices. However, the spectrogram shows that the onset of EMG corresponds to the increase in the index values. The frequency range of EMG is actually 0–300 Hz, and therefore, these phenomena must be due to EMG, not EEG. The claim that the EMG frequency range is 30–300 Hz appeared in anaesthesia literature when EEG-derived anaesthesia indices were developed, suggesting that eventual EMG activity does not disturb EEG evaluation while index numbers are calculated, but this is not true.

The frequency spectrum of surface EMG ranges down to 0 Hz.² In fact, the maximum power of frontal muscle activities can be as low as 6 Hz or even lower.^{2–3} In Figure 1A, we present a EEG signal at burst-suppression level under propofol anaesthesia with superimposed EMG, consisting of a single motor unit potential which is regularly firing. The power spectrum of this signal has a maximum at 6.6 Hz and lower peaks at harmonic frequencies. A signal consisting of several motor unit potentials from submental muscles is illustrated in Figure 1B. Notice that the EMG covers the whole 0–30 Hz range.

If the frontal muscle contraction is strong, the muscle activity can totally hide the EEG signal recorded by the monitors. This is often seen in non-paralysed patients during propofol anaesthesia, particularly at induction.⁴ Owing to the overlapping frequencies of EEG and EMG, it is impossible to remove this EMG artifact from EEG by band-pass filtering. As the EMG is often non-stationary, its variable pattern makes it impossible to remove EMG totally from the EEG with any signal processing technique.

Thus, the increase in BIS and WAVCNS numbers in the case presented by Chazot and colleagues is obviously due to the part of EMG that is in the EEG band of the analysis system, although the low resolution of the EEG signal in their Figure 1 makes it impossible to judge how much EMG is really included. From their figure, it is also impossible to