adult practice. It is possible that the paediatric BIF may prove to be useful in children with difficult airways.

In summary, the paediatric BIF improves visualization of the glottis when compared with DL in the normal paediatric airways, but has a higher incidence of failure to intubate. Caution should be exercised when using newly developed ‘scaled down’ airway devices in paediatric anaesthesia without sufficient data to support their use in children.

Conflict of interest
None declared.

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Improvement of cerebral blood flow patterns in hepatorenal syndrome using sustained low-efficiency dialysis

Editor—We report the case of a 23-yr-old male with cystic fibrosis who had end-stage cirrhosis of his liver and was referred to the intensive care unit (ICU) because of decompensated hepatic encephalopathy. In the ICU, his level of consciousness deteriorated and required tracheal intubation for hypercapnic respiratory failure. He started on haemodialysis for worsening renal failure. Over the night of day 4 and into the early hours of day 5, his clinical condition deteriorated and he suffered a generalized seizure that was associated with hypoglycaemia. He had a short asystolic cardiac arrest from which he was rapidly resuscitated. Through the night, he continued with ongoing seizures requiring benzodiazepines and propofol for suppression. A computed tomographic (CT) scan done that morning revealed diffuse cerebral oedema. That afternoon, the reversal of diastolic flow in the middle cerebral artery and an increased intracranial pressure (ICP) were identified using transcranial Doppler (TCD). Calculated direction of flow index (DFI) was <0.8 (0.77). A study has suggested that if this value is <0.8, recovery to forward flow throughout diastole was never observed and no patient recovered brain stem reflexes (Fig. 1).1

The possibility of an external ventricular drain was explored but due to diffuse cerebral oedema, coagulopathy, and collapsed ventricles, this was not appropriate. To manage this acute crisis, the patient was put on sustained low-efficiency dialysis (SLED) with a high effective osmolality. The investigations done before the SLED included: urea, 19.9; Cr, 288; and Na, 141. Dialysis was started at dialysate flow 550 ml min⁻¹, blood pump speed 250 ml min⁻¹, duration 7 h, and fluid balance = 0. After dialysis, a repeat TCD study revealed a normal DFI. To our knowledge, no previous

Fig 2 Improvements in cerebral blood flow patterns post-SLED, reversal of DFI back to normal.
cases have been reported to date where the DFI was <0.8 and could be reversed to normal by any therapeutic interventions (Fig. 2).

The next morning, TCD again showed the reversal of flow and a CT scan showed massive cerebral oedema. In consultation with the team and with the family, a decision was made to switch his goals of care to palliation and the patient died later that day.

The measurement of deteriorating cerebral perfusion pressure (CPP) non-invasively is possible with TCD. As CPP approaches zero, blood vessels collapse during diastole, followed by absent or reversed diastolic flow. A DFI was defined as DFI = 1 – R/F, where R is the velocity of the diastolic reverse flow and F the velocity of the systolic forward flow. A DFI < 1 indicates reverse flow.

Intracranial hypertension has been reported in patients with fulminant hepatic failure. With a shorter time interval between the start of symptoms and the onset of encephalopathy, there is a greater risk of cerebral oedema.

Intermittent haemodialysis may result in an increase in ICP and has been reported to cause dialysis disequilibrium syndrome (DDS) with induced cerebral oedema that resulted in irreversible brain injury and death. The use of SLED gives a more gradual and stable clearance of urea. DDS has not been reported with the use of SLED.

In our patient with fulminant liver failure and signs of raised ICP, the use of SLED showed a marked improvement in cerebral perfusion and reduction of ICP. To our knowledge, this is the first reported case where any therapeutic manoeuvre managed to reverse the DFI back to normal, indicating that SLED may have a role in reducing ICP and promoting cerebral perfusion, especially in patients with severe liver dysfunction.

**Conflict of interest**

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**Steep Trendelenburg position, intracranial pressure, and dexamethasone**

Editor—I read with interest the article on the effect of steep Trendelenburg position, as I am involved in a lot of laparoscopic abdominal work. These procedures can last up to 6 h and involve a steep head-down Trendelenburg and left or right tilt, to the extremes of the table mechanism. I agree with the findings regarding the changes in stroke volume and cardiac function, as measured by oesophageal Doppler, and was extremely pleased to see the results regarding cerebral oxygen saturations. The authors briefly mention changes in intracranial pressure (ICP) but do not elaborate further. It is my impression from many cases of this sort that the ICP does increase and despite relative normocapnoea and arterial pressure maintenance, some of these patients suffer an acute confusional state after operation. Since the addition of dexamethasone 8 mg into our post-operative nausea and vomiting regime, this only occurred in the diabetic patients who were not given dexamethasone. Owing to this observation, I now give all my patients dexamethasone and monitor and treat the changes in the blood sugar as appropriate. Further, once the surgeons have finished the laparoscopic part of the surgery and are closing the abdominal wounds, I use a reverse Trendelenburg, as much as surgery allows, until the end of surgery. This appears to prevent any postoperative acute confusion. Have the investigators noticed anything similar and how do they manage these patients and this problem?

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Editor—we thank Dr George for his interest in our article and his interesting comments. We also commonly observe a short-lived period of postoperative confusion after prolonged Trendelenburg positioning. While we do not have personal experience with dexamethasone for this indication, in our clinical practice, we do tend to keep the patients sedated for another 60 min after long procedures.

The hypothesis that this confusional state may be caused by cerebral oedema prompted us to perform a follow-up study in which we focused on cerebral perfusion. In this study (currently undergoing peer review), we did not show an influence on cerebral perfusion parameters, but of course this does not exclude a degree of cerebral oedema. Thus, the idea, of using dexamethasone to prevent the