disorders in AYAs are lacking. This is an area that warrants further investigation.

I believe that there is enough evidence to support education in AYAs about sleep hygiene and the potential effects of smartphone use at bedtime and at night and I applaud the author's advice that good sleep hygiene would 'include not having these devices within the bedroom'. ■

Dr Martin Lee
Consultant rheumatologist, Newcastle-Upon-Tyne Hospitals
Director of www.nophonezone.co.uk

References

1 Bruce ES, Lunt L, McDonagh JE. Sleep in adolescents and young adults. *Clin Med* 2017;17:424–8.
2 Ofcom Communications Report 2016. Ofcom, 2016. www.ofcom.org.uk/research-and-data/multi-sector-research/cmr/cmr16 [Accessed 31 October 2017].
3 Deloitte. *State of the smart: Consumer and business usage patterns. Global Mobile Consumer Survey 2017: UK Cut*. www.deloitte.co.uk/mobileUK/ [Accessed 31 October 2017].
4 Cain N, Gradisar M. Electronic media use and sleep in school-aged children and adolescents: a review. *Sleep Med* 2010;11:735–42.
5 Lemola S, Perkinson-Gloor N. Adolescents' electronic media use at night, sleep disturbance, and depressive symptoms in the smartphone age. *J Youth Adolesc* 2015;44:405–18.
6 Lovato N, Gradisar M. A meta-analysis and model of the relationship between sleep and depression in adolescents: recommendations for future research and clinical practice. *Sleep Med Rev* 2014;18:521–9.
7 Oshima N, Nishida A. The suicidal feelings, self-injury, and mobile phone use after lights out in adolescents. *J Pediatr Psychol* 2012;37:1023–30.

Endocrine abnormalities in lithium toxicity

Editor – Shanks *et al*[1] describe salient physiological lessons from their patient with severe sequelae of lithium toxicity. I wish to suggest further lessons that may be learned from their report to minimise harm in future similar cases. It appears that nephrogenic diabetes insipidus (DI) was not considered until day 10 of the admission. That sodium remained elevated despite fluid resuscitation clearly implicates impaired renal salt handling and the patient's chronic lithium treatment was known. It is pertinent that 0.9% sodium chloride failed to correct the hypernatraemia from the start and so earlier suspicion of DI may have helped. There is no reason to stick doggedly to saline infusions to treat hypercalcaemia and 5% dextrose may have been the preferable resuscitation fluid.

One may justly wonder if the patient would have benefited from earlier ITU admission. Serum sodium was 172 mmol/L at day 4 – a severe medical emergency especially in a young patient – yet she was not transferred to ITU until almost a week later on day 10. This case must surely demonstrate that close monitoring and chasing of fluid and electrolyte goals is very hard on Level 1 wards. I would suggest that the refractory severe hypocalcaemia seen in this case was a known complication of unnecessary bisphosphonate treatment, as calcium will typically drop to safe levels (<3 mmol/L) with sufficient fluids alone. It would be pertinent to know how much improvement in fluid balance and what correction in serum calcium was achieved before the IV bisphosphonate was administered. Finally, with regards the patient's neurological disorder, did the authors consider the possibility of thyrotoxic periodic paralysis? ■

BERNARD FREUDENTHAL
Clinical research fellow, Molecular Endocrinology Laboratory, Imperial College London

Reference

1 Shanks G, Mishra V, Nikolova S. Endocrine abnormalities in lithium toxicity. *Clin Med* 2017;17:434–36.

Regular and frequent feedback of specific clinical criteria delivers a sustained improvement in the management of diabetic ketoacidosis

Editor – Notwithstanding the statement made by the authors of this paper that 'Fluid replacement is the most important initial management [in diabetic ketoacidosis]',[1] the caveat is that intravenous fluid (IVF) replacement is contraindicated when pulmonary oedema is present on admission in a patient with diabetic ketoacidosis (DKA).[2,3] In some of these cases, advanced chronic renal failure is an associated feature.[2] Pulmonary oedema may also be a feature when non-ketotic hyperglycaemia occurs in a patient with chronic renal failure managed by haemodialysis.[4] Both in the context of DKA[2] and non-ketotic hyperglycaemia,[4] one of the underlying causes of pulmonary oedema is the osmotic shift of fluid from the intracellular to the extracellular fluid compartment as a consequence of severe hyperglycaemia. This may overwhelm the pulmonary circulation when there is impaired excretion of that sudden additional extracellular fluid load. In some of these patients the sole use of insulin to correct hyperglycaemia may be instrumental in the resolution of pulmonary oedema.[2,4]

Pulmonary oedema may also be present on admission in a DKA patient with coexisting congestive heart failure.[3] In that context IVF replacement can be withheld, and DKA can be managed solely with intravenous insulin infusion.[3] The associated pulmonary oedema resolves after intravenous administration of frusemide.[3] Also in the context of cardiogenic pulmonary oedema a potential alternative treatment strategy is the use of intravenous nitrate infusion,[5] the latter a well-tried strategy in the management of pulmonary oedema complicating myocardial infarction.[6] The advantage of the latter strategy is that, in a DKA patient concurrently managed with an insulin infusion, hypokalaemia is less likely to be an outcome than might be the case when diuretics are coprescribed with insulin infusion. ■

OSCAR M P JOLOBE
Manchester Medical Society

References

1 Kempegowda P, Coombs B, Nightingale P *et al*. Regular and frequent feedback of specific clinical criteria delivers a sustained improvement in the management of diabetic ketoacidosis. *Clin Med* 2017;17:389–94.
2 Varma R, Karim M. Lesson of the month 1: Diabetic ketoacidosis in established renal failure. *Clin Med* 2016:16:392–3.
3 Jolobe O. Potassium status should be evaluated also when diabetic ketoacidosis is complicated by heart failure. *Am J Emerg Med* 2016;29:955.