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Thoracic ultrasound experiences among respiratory specialty trainees in the UK

There were some interesting points raised by Sivakumar *et al* in their article on thoracic ultrasound experience among respiratory trainees in the UK.[1] The difficulties with supervision and competent senior clinicians is one which is also seen among acute internal medicine trainees wishing to gain experience in point of care ultrasound (POCUS),[2] and requires much work to overcome. It is reassuring that progress is being made within respiratory medicine.

The fact that a number of trainees claim confidence in diagnosing pulmonary oedema and pneumothorax with ultrasound is interesting, since neither of these pathologies are covered within the Royal College of Radiologists (RCR) curriculum.[3] Indeed, I am not aware either of a training curriculum that sits within respiratory medicine which covers the use of bedside ultrasound for respiratory failure, as opposed to pleural disease which has traditionally been the mainstay of thoracic ultrasound. To my knowledge, the focused acute medicine ultrasound (FAMUS) curriculum[4] is the only training programme for physicians within the UK which covers the use of POCUS in patients with conditions like pneumothorax and pulmonary oedema.

I agree with the sentiment that the current RCR curricular do not entirely fulfil the requirements of respiratory medicine trainees, since they do not cover the use of thoracic ultrasound for respiratory failure. There is increasing evidence for the utility of POCUS for aiding diagnoses of pneumothorax, pulmonary oedema, pneumonia, asthma/COPD and pulmonary embolism,[5] and this should surely form part of a future respiratory medicine ultrasound curriculum. This seems imperative to me given that respiratory medicine (and indeed acute internal medicine) care for the majority of medical patients with acute respiratory failure.

A revision of the current respiratory medicine ultrasound curriculum to include a respiratory failure component would require a significant undertaking from training committees to support both trainees to achieve those competencies, and trainers to deliver them. However, given this is a largely untapped use of POCUS with significant scope to improve diagnostic accuracy,[5] it is a logical extension of the current use of ultrasound within respiratory medicine. ■

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Conflict of interest

The author is a member of the FAMUS working group.

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Sleep in adolescents and young adults

Editor – I congratulate the authors of the recent article about sleep in adolescents and young adults (AYAs).[1] Like the authors, I am aware of the association between poor sleep hygiene and the development of mental health and chronic pain disorders. As a consultant physician working in the UK with an interest in AYA care, I observe the consequences of poor sleep in this group of patients on an almost daily basis. I have an interest in 'smartphone overuse syndrome' (SOS) in AYAs and, in particular, smartphone use at night and the negative effects it may have on sleep. The UK Ofcom communications report published in 2016 highlighted that, on average, we now spend more time on electronic media and communications than we do sleeping.[2] Two-thirds of 16–19-year-olds wake in the middle of the night to check their phones.[3]

A number of theories have been proposed about how smartphone use in the evening and at bedtime can affect our sleep.[4]

- Sleep could simply be displaced by smartphone use at night leaving less time for sleep, sometimes referred to as 'sleep stealing'.
- Smartphone use at bedtime could lead to increased mental, emotional or physiological arousal and therefore interfere with time to onset of sleep.
- Light emission from smartphones that use back-light or 'blue-range' light technology has been demonstrated to interfere with melatonin secretion and our circadian physiology.
- Incoming messages, emails, status updates or calls can disturb sleep and are associated with a reduction in the quantity and quality of deep or 'restorative' sleep.

The comorbidity of depression with sleep problems is common and well documented.[5] A recent meta-analysis reviewing the relationship between sleep and depression in adolescents suggested that sleep disturbance plays a key role in the aetiology of depression during adolescence.[6] A study published in 2012 found a significant association between nocturnal mobile phone use and poor mental health, suicidal feelings and self-harm after controlling for other confounding variables (including sleep length) in 17,920 adolescents.[7]

Despite evidence demonstrating an increase in the use of electronic media and smartphones in AYAs, as well as evidence linking this increased use to sleep and mood disturbances, studies looking at smartphone use, sleep disturbance and chronic pain

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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'. ■

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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? ■

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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. ■

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