should that illness be managed and what level of medical intervention would the person want?'. We frequently admit such patients without any information about their quality of life, prior wishes or capabilities, and it can take several days to get that information. In the meantime, we are faced with decisions about iv fluids, antibiotics, computed tomography scanning, resuscitation and a host of other potential interventions. Alternatively we are told that the person 'suffers from dementia' but we learn nothing about its severity, nor its impact is on the level of function. Martin et al are right to say that 'a value judgement on whether the . . . outcome [of treatment] is worthwhile' should be made by the patient and not by the doctor; the problem is that we almost never have the opportunity to discuss this decision in a timely manner and whether we like it or not some such decisions (for example, on 'do not attempt resuscitation' orders or intensive care admission) simply have to be made by doctors.

> ROGER A FISKEN Consultant Physician Friarage Hospital Northallerton

Lesson of the month

Editor – I found Dr Mir *et al*'s lesson of the month interesting (*Clin Med* October 2007 pp 530–1). It is worth adding to their article by stating a few important points that are perhaps not appreciated by the wider medical community that come into contact with hypertensive patients.

Firstly, the vast majority of patients with primary aldosteronism are normokalaemic. Hypokalaemia is a late feature of Conn's syndrome or aldosterone-producing adenoma. In one series 67% of patients with aldosterone-producing adenomas were normokalaemic.¹ Thus the absence of hypokalaemia does not exclude primary aldosteronism. I would agree that hypokalaemia in a patient with hypertension who is taking thiazides could point towards a diagnosis of primary aldosteronism.

Secondly, the previously held notion that aldosterone-producing adenomas are uncommon has been challenged since the introduction of the aldosterone to plasma renin activity ratio as a screening tool. In

one study there was a fourfold increased removal of aldosterone-producing adenomas resulting in the cure of hypertension in $60\%.^2$

Thirdly, dihydropyridine calcium channel blockers, eg amlodipine and diuretics, can elevate renin levels resulting in a falsely normal aldosterone to renin ratio. Thus amlodipine and bendroflumethiazide could be withdrawn for a period no less than two and four weeks respectively with repeat renin/aldosterone studies. If blood pressure control is required in the interim then non-dihydropyridine calcium channel blocker such as dilitazem (safer than verapamil) or the alpha blocker doxazosin could be used instead.

Finally, it is very important that imaging is only carried out once biochemical confirmation of primary aldosteronism has been established. The incidence of nonfunctional adrenal adenomas increases over the age of 40 years. There are cases of primary aldosteronism arising from the contralateral adrenal gland in someone with an incidental adrenal adenoma in the opposite gland.³

ABBI LULSEGGED Consultant Physician Bromley Hospitals NHS Trust

References

- Hiramatsu K, Yamada T, Yukimura Y et al. A screening test to identify aldosterone producing adenoma by measuring plasma renin activity. Arch Intern Med 1981;141: 1589–93.
- Rutherford J, Taylor WC, Stowasser M, Gordon R. Success of surgery in primary aldosteronism judged by residual autonomous aldosterone production. World J Surg 1998;22:1243–45.
- 3 Stowaser M, Gordon R, Gunasekera T et al. High rate of detection of primary aldosteronism, including surgically treatable forms, after 'non-selective' screening of hypertensive patients. J Hypertens 2003;21: 2149–57.

Lack of access to out-of-hours endoscopy: implications for trainees too

Editor – I wholeheartedly agree with the findings of Gyawali *et al*'s survey (*Clin Med* December 2007 pp 585–8) which concludes that there is significant underprovi-

sion of gastrointestinal (GI) emergency medical services in England. After initial resuscitation and physiological stabilisation of patients with GI emergencies, as a general medical (non-gastroenterology) trainee, I have repeatedly found myself (in centres with no established on-call rota) devoting significant time (with switchboard staff) trying to find an available endoscopist. I would rather spend such time re-evaluating and maintaining the stability of such patients who, as Gyawali et al state, are often extremely sick and complex to manage, requiring prolonged input. Therefore, the delay in accessing an on-call endoscopist (as well as their absence) is another factor which has the potential to impact on patient care in this

Another important issue is that with the reduced number of GI emergencies admitted to some hospitals (for example because of redeployment of gastroenterologists to regional centres or neighbouring units) medical trainees may be underexposed or deskilled in the management of such emergencies in some centres. However, such emergencies can subsequently develop in hospital after admission because of critical illness. It is therefore essential that trainees have experience, confidence and expertise to mange these situations.

In conclusion, a robust and accessible system for on-call endoscopy is the goal but it is a challenging one because of resource and capacity issues. The suggested 'hub and spoke' model with regional centres may well be the solution but general medical trainees must rotate through them to attain sufficient experience and expertise to manage these emergencies whenever they arise.

ANDREW RL MEDFORD Specialist Registrar in Respiratory and General Medicine North Bristol Lung Centre