training and multi-source feedback have not given any thought to designing a uniform career pathway for all concerned.

Perhaps it is no surprise that the majority of these ''non-training'' posts are occupied by overseas doctors. With the implementation of MMC and EWTD, the delineation between training posts and service posts will become all the more obvious. Whether this is to the ultimate good of the staff morale, the NHS, and the patients, is anybody's guess.

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Reference

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Antenatally diagnosed single kidney: lack of uniformity in postnatal management practice

Diagnosis of a single kidney on antenatal scan is not uncommon. Unilateral renal agenesis (URA) is reported to occur in 1:1500 prenatal ultrasound studies.[1] Other causes of single kidney are less common and include unrecognised ectopic kidney and fetal involution of a previous unilateral multicystic dysplastic kidney.[2] Forty eight per cent of children with URA are reported to have other urological anomalies, with more than half demonstrating vesicoureteric reflux.[3] Adult patients with URA are reported to have an increased incidence of proteinuria, hypertension, and renal insufficiency.[4]

We conducted a survey to examine how otherwise healthy babies born with antenatally diagnosed healthy looking single kidney are managed in the United Kingdom. A questionnaire was sent to a 219 consultant paediatricians, one in each hospital. Questions were related to postnatal management, including the need for investigations, antibiotic prophylaxis, and follow up.

We received 138 responses (63%). Most of the respondents (n = 131, 94%) agreed for the need for a postnatal ultrasound. Sixty eight (49%) would perform further investigations such as micturating cystogram (MCUG) and/or DMSA/MAG3 if the ultrasound scan was abnormal. However, a significant number (n = 60, 43%) would perform further studies irrespective of the ultrasound result. About half the consultants (n = 65, 47%) would offer their patients prophylactic antibiotics until the radiological assessment excludes the possibility of associated renal anomalies, contralateral ectopia, and residual renal tissue. Forty eight respondents (35%) will follow up their patients for at least one year (30 suggested five years or more), 50% of whom will perform MCUG and/or DMSA/MAG3 as a routine. Ten (7%) will refer to the specialist services and the rest will discharge their patients if the radiological investigations are normal.

This survey demonstrates wide variation in the practice among UK paediatricians in the management of antenatally diagnosed single kidney and suggests the needs for development of national guidelines.

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Breast feeding method should ensure rapid weight gain

McKie *et al* have shown that routine neonatal weight monitoring (with targeted breast feeding advice) does not discourage breast feeding.[1] Breast feeding advice was targeted at babies losing >10% of birth weight or failing to regain birth weight by the age of 14 days. An earlier paper from the same study has shown that 5.3% of babies showed a faltering of weight gain between 10 and 20 days, and that all babies above the 97.5 centile for weight loss had some degree of hypernatraemia.[2] The authors of this earlier paper commented on the increase in dehydration and/or failure to thrive in breast fed babies caused by lactation failure and non-recognition of feeding problems. Exclusively breast fed babies can be expected to grow more quickly than anticipated so that they are above their birth centile (for weight) by the age of 6–8 weeks.[3] Most mothers ceasing to breast feed between the ages of 10 days and 6 weeks state that they do so because of milk insufficiency.[4]

We suggest that breast feeding advice to ensure rapid weight gain (monitored by a structured approach to regular weighing) is likely to increase breast feeding rates and reduce the risks to the neonate.

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Exercise induced dyspnoea: if not asthma, then what?

The report by Seear *et al* found that most children referred to their clinic with a history of exercise induced asthma (EIA) did not have asthma.[1] These investigators conclude that the majority of exercise associated respiratory complaints can be diagnosed and managed without the need for exercise testing. The accompanying editorial concurs with that view.[2] Our own study, recently published, is supportive of the overdiagnosis of asthma as a cause of exercise induced dyspnoea (EID).[3] However, I would argue against the conclusion that formal exercise testing is not indicated. In fact, our experience showed the value of full cardiopulmonary evaluation with breath-by-breath analysis of oxygen consumption and carbon dioxide production for such children during exercise sufficient to reproduce their symptoms.

By reproducing EID in 117 patients, most of whom had been previously diagnosed and treated for asthma, while continuously monitoring their cardiopulmonary physiology, we identified only 11 who had EIA during reproduction of their EID. Fifteen had flattening of the inspiratory portion of the flow-volume loop at the time of reproduction of symptoms. Direct visualisation with a flexible laryngoscope while symptoms were present distinguished two who had exercise induced laryngomalacia from those with vocal cord dysfunction. There were other patients who had what sounded like inspiratory stridor at very high minute ventilation but did not have evidence for upper airway obstruction. They simply moved sufficient air through a normal variant airway that an inspiratory sound could be reproduced during maximal exercise. We also identified 15 who, when their EID was reproduced, attained their maximal minute ventilation with a decreased maximal tidal volume and abnormally high respiratory rate consistent with a component of chest wall restriction not apparent at rest. One patient only had exercise induced hyperventilation, a phenomenon we have previously reported in others.[4] Supraventricular tachycardia demonstrable by ECG was identified in a well conditioned highly competitive teenage athlete whose heart rate jumped suddenly to 220 during maximal exercise in association with his EID. He underwent a successful ablation by our cardiac electophysiologist which completely eliminated this young athlete's EID, enabling him to complete a full basketball game without having to repeatedly stop to recover. Since our publication, we have identified another teenager whose EID was associated with supraventricular tachycardia, also only present during vigorous exercise. Both of these adolescents had been previously treated unsuccessfully for EIA. Of the 117 in whom we reproduced their EID during the treadmill exercise, 74 had only normal physiological exercise limitation, with cardiovascular conditioning among them about equally divided between poor, average, and above average cardiovascular conditioning.

Our experience is that simply identifying the absence of EIA is not sufficient. Even for the 74 with no physiological abnormality, we were able to counsel those with poor cardiovascular conditioning regarding the means to improve exercise ability and could even counsel those with excellent cardiovascular conditioning about the need for training to learn how to manage normal exercise limitation. Moreover, since dyspnoea from any cause is anxiety

producing, identifying the cause of the EID and providing recommendations for a potential solution relieves that anxiety component which was likely to be a factor in seeking medical care and receiving inappropriate treatment in the first place.

In the study by Saeer *et al*, one wonders if all eight children identified as having EIA actually had asthma as the cause of their EID since the diagnosis was based on an FEV_1 decrease $\geq$10% but <15% in six, decreases which may be within normal limits for non-asthmatics.[5] In contrast to the 21% of children with no diagnosis for their EID, we were able to identify the cause of EID in all of our patients in whom full cardiopulmonary monitoring was performed in association with sufficient exercise to reproduce their symptoms.

Thus, while we certainly agree with Seear *et al* and the accompanying editorial that EIA is overdiagnosed, our experience would argue for exercise testing that reproduces the symptoms of EID during continuous cardiopulmonary monitoring for those whose history is otherwise atypical for asthma.

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Accident and emergency: a gateway to improve the management of atopic disease

We read the recent paper by Holgate and Lack[1] with much interest. The use of appropriate allergy testing with the aim of accurate diagnosis is of particular importance in the setting of increasing incidence of allergic disease.[2 3]

We recently audited the impact of atopy on the workload within our paediatric A&E department; 14 369 episodes from a one year period were analysed. Presentations assigned a diagnosis of asthma, eczema, urticaria, angioedema, anaphylaxis, food allergy, drug allergy, or hay fever were felt to be related to atopy. Children under the age of 4 who presented with wheeze associated with a concurrent respiratory tract infection were assigned a diagnosis of viral induced wheeze and not included within the atopy grouping. Of these presentations, 811 (5.6%) were assessed as relating to atopy (fig 1).

We also compared the percentage of admissions and medical follow up between allergic presentations to A&E and non-allergic presentations. Not only did allergic conditions form a significant proportion of the department's workload, but a statistically significant higher proportion of these patients required admission ($p < 0.001$), outpatient follow up ($p < 0.01$), and general practitioner follow up ($p < 0.001$) when compared with the non-allergic population (fig 2).

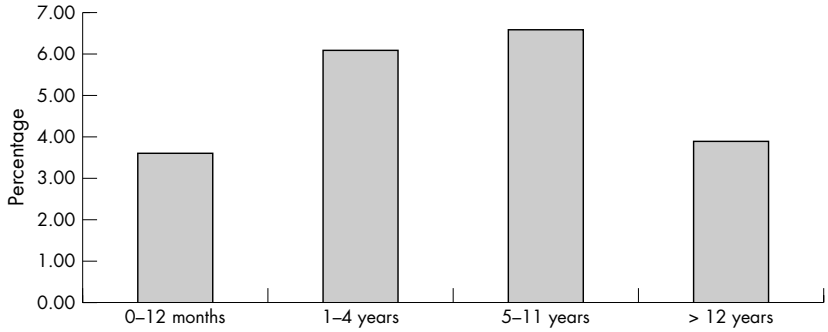

Figure 1 Percentage of allergic presentations with respect to total episodes.

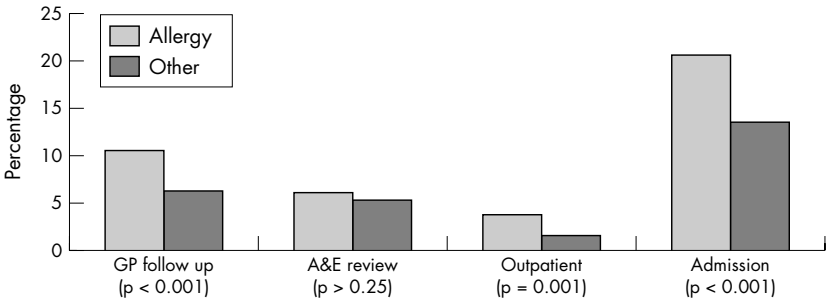

Figure 2 Comparison of National Health Service utilisation for follow up.

The change in the pattern of allergic presentations (the atopic march) across the paediatric age range was evident, with eczema and food allergies being more prevalent in infants and young children and asthma becoming more prevalent in older children. There is evidence that early diagnosis and treatment of atopic disease prevents progression of the allergic march.[4–6]

With the above in mind, the recommendations from Holgate and Lack to utilise specific allergy testing may be useful as a follow up in children presenting to A&E with disease thought to be secondary to atopy. The A&E setting could facilitate early allergic diagnosis, increase parental awareness, and potentially improve patient management.

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Buccal midazolam: is a test dose in hospital needed?

Midazolam has been given via the buccal route for the acute management of prolonged seizures for some years. Buccal administration is preferable to the rectal route for parents and carers.[1] There is evidence that midazolam given in this way is more effective in curtailing seizures than diazepam and that there is no difference in the incidence of side effects.[2]

In some districts, including our own, if the family and the prescribing clinician want to use buccal midazolam the child is admitted as a day case to an acute paediatric ward for a ''test dose''. This has a number of disadvantages, particularly for children with autism or complex neurodisability:

- Inconvenience for families making the trip to hospital
- An extra day off school
- Staying the day in an unfamiliar environment
- The test dose is given to a conscious child and therefore does not mimic its use during a seizure; some consider this unethical
- Use of hard pressed acute care paediatric beds, nursing care, and medical time.

NICE guidelines[3] mention buccal midazolam but do not suggest a test dose. A search of