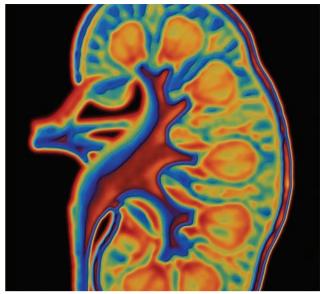
THIS MONTH



Key questions
Food allergies
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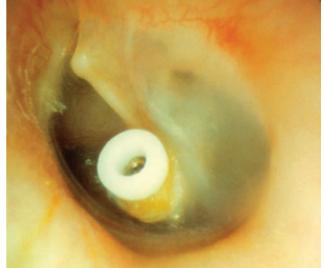
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KEY QUESTIONS

Food allergies

Paediatric allergy consultant Dr Adam Fox and allergy GPSI
Dr David Mass answer questions from GP Dr Sara Ritchie
on cow's milk allergy, urticaria and when an adrenaline
autoinjector is needed

What should make GPs suspect cow's milk protein intolerance in a bottlefed baby? How can the diagnosis be made with certainty?

Cow's milk protein intolerance is quite an outdated term now. Non-IgE mediated cow's milk allergy is probably better as it reflects that these are immune-mediated reactions.

This is different from lactose intolerance which, in infants, is usually because of viral gastroenteritis eroding the lactose enzyme in the brush border of the gut, causing diarrhoea for about a month.

Typical symptoms of non-IgE mediated cow's milk allergy include reflux, colic, vomiting, diarrhoea – particularly with blood and mucous – and eczema. Faltering growth can be a sign, but is not always present. The more systems that are involved, the more likely there is to be an allergic cause – so, for example, the presence of eczema alongside gut symptoms needs to be taken seriously. The relationship between symptoms and cow's milk exposure is key.

Look for significant worsening of symptoms after moving from breastfeeding to bottlefeeding. Remember that there is cross-reactivity between cow's milk and soya milk, which can confuse things.

Less frequently, IgE mediated reactions to cow's milk occur within seconds to minutes of exposure to milk protein. Symptoms include urticaria, runny nose and eyes, angioedema, diarrhoea and vomiting, shortness of breath and decreased blood pressure.

The only way to make a clear diagnosis is to do an exclusion diet followed by reintroduction four to six weeks later.

What dietary advice should we give to the parents of children older than one year who still have cow's milk protein allergy?

There is real potential to get things wrong with children who have cow's milk allergy and this can have significant nutritional consequences.

Infants are less reliant on milk once they reach the age of one, and the important thing is that they have a good source of calcium. It is essential that any infant with milk allergy has contact with a dietician.

It is also worth bearing in mind that

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children with a history of food allergy are more likely to have ongoing food aversions and fussy eating, and early intervention by an experienced dietician can make a real difference.

If urticaria in children is delayed in onset, and not significantly improved with chlorphenamine, should a viral cause be suspected rather than food allergy?

Viral urticaria is often mistaken for food allergy and there is a growing tendency, when a child develops hives, for parents to ascribe it to a food.

There are a few important differences that will help you to distinguish an allergic reaction from viral urticaria. Viral urticaria often takes hours to develop and then lasts for a number of days, while allergic reactions tend to develop and then pass very rapidly. Also, with viral urticaria the rash will often calm down a little with chlorphenamine but then flare up again. With an allergic reaction, once the rash has been effectively treated with an antihistamine it will most likely stay away.

Obviously the timing of the rash and exposure to possible allergens is also important, so for example if the child went to bed perfectly well but woke up in the morning with hives, a food allergy is very unlikely.

In a small number of children, milk seems to exacerbate their eczema. Is a trial of milk exclusion with subsequent re-challenge appropriate if eczema is severe and difficult to control?

There are certainly children, particularly infants, whose eczema is exacerbated by a food allergy. These children can sometimes make a dramatic improvement when moved off cow's milk formula onto a hypoallergenic formula, or when their mother, if breastfeeding, removes cow's milk from her diet.

Both the NICE food allergy and eczema guidelines^{1,2} recommend that infants with early onset of severe eczema that is resistant to conventional treatments should be considered for a four- to six-week trial of a hypoallergenic formula. Children who have gastrointestinal symptoms with significant eczema also need careful consideration for a trial exclusion diet followed by reintroduction of cow's milk.



My understanding of the NICE guidelines is that all patients with IgE mediated food allergy should be referred to a dietician. Do you think this is always necessary?

Yes, it is essential that all children with food allergy have dietetic input. Dietetic advice ensures patients are educated and empowered to avoid further reactions, and that they have an appropriate nutritious diet, despite the restrictions.

There is clear emerging evidence that growth parameters are better in children with food allergy as a result of dietetic involvement. Parents often find it difficult to shop for a child with food allergy so having the skills to understand advisory labels and risk assessment are essential for successful management of food allergy, and this help is best delivered by dieticians.

If a patient presents with urticaria after eating nuts other than peanuts, but has no history of angioedema, should they be advised to carry an adrenaline autoinjector?



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Many allergy clinics will check specific IgE levels annually to predict the development of tolerance and help to decide when oral challenges are indicated.

And with IgE mediated reactions, food challenges should usually be done in a supervised environment.

Allergy clinics also carefully look to identify and manage other allergic co-morbidities in food-allergic children as these are extremely common.

The annual review is an opportunity for a specialist to spend a significant amount of time with each child, considering their allergy holistically in the context of likely eczema, asthma, rhinitis, and other issues.3 So this cannot easily be done in primary care.

Is RAST testing necessary to 'prove' allergy if the food culprit seems obvious - and in severe reactions, should the GP request **RAST testing on all likely foods?**

RAST testing has now been replaced by specific IgE testing, which detects the amount of allergic antibody to a specific allergen. The larger the number, the more likely it is that there is true clinical allergy, while smaller values may simply represent sensitisation.

It is definitely worth doing a specific IgE test to confirm a food allergy, even with a very clear history.

Parents sometimes confuse allergens. for example peanuts and other nuts, and occasionally – especially if the reaction is not reported until some time after the event – tolerance could have developed.

Also, having a baseline value provides useful prognostic information. Lower initial values are linked to an increased chance of tolerance developing. And a gradual decline in the amount of specific IgE on further testing can indicate when a child has outgrown the allergy.

Testing for other common allergens needs to be directed by the history. There is no point testing for foods that are already tolerated as this can produce unhelpful false positives, but, it can be useful to test for commonly associated allergens that have not yet been demonstrably tolerated. For example, in children with an egg allergy there is a 20-30% risk of peanut allergy and a negative test would be reassuring, and in peanut-allergic children there is a significant risk of sesame allergy so it is worth looking for this if there is no clear history of tolerance.

Dr Adam Fox is a consultant in paediatric allergy at Guy's and St Thomas' Hospitals, London, and Dr David Mass is an allergy GPSI in north west London. Dr Sara Ritchie is a GP in Stoke Newington, north London.

• If you are interested in learning more about allergy, there are many courses and seminars available through the King's College London Allergy Academy: allergy academy.org

allergy syndrome may notice oropharyngeal reactions when they eat raw fruit between birch pollen and rosacea fruits such as apples, pears, peaches and

plums. There is a similarity between the main allergenic protein in birch pollen and in these fruits, so once hay fever has developed, patients may start to notice oropharyngeal reactions when they eat raw fruit. There will usually be a history of

tolerating the fruit for many years before the symptoms start. Symptoms are often variable - affecting patients on some occasions and not others, and the fruits can be eaten when they are processed or cooked. Severe reactions are rare and diagnosis can be based on positive allergy testing to birch pollen together with the appropriate history. These reactions can sometimes start to affect an increasing number of foods and become a real problem, in which case a specialist referral would be worth considering.

I understand that allergy clinics do annual specific IgE tests in children to check when a food allergy may have resolved. Could these be undertaken in primary care?

Children with oral

Extra Q&As

Go to the online version of this article to see two extra Q&As on: adrenaline autoinjector education in primary care explaining when testing for food allergies is not necessary

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ka-food

How can GPs distinguish oral allergy syndrome from more serious reactions? When should we consider referral?

The decision to carry adrenaline

likelihood of future anaphylaxis and it is

important that this is not overly tied in

important risk factors for future severe

and co-morbid asthma. These are both

absolute indications for children with food allergies to carry adrenaline

injectors.

of life.

reactions are a history of severe reactions

There is data to suggest nuts are the

cause of most severe reactions and this

includes tree nuts as well as peanuts, but

there have also been fatalities as a result

injectors also needs to take into account

remoteness from medical care and the

likely impact on the family's quality

of fish, shellfish and milk allergy

The decision to prescribe adrenaline

autoinjectors relates to the

with the specific allergen. The most

Oral allergy syndrome results from cross-reactivity, most commonly

NEW SERIES

CKD IN PRIMARY CARE

Benign decline or CKD?

Consultant nephrologist Dr Robert Lewis advises on kidney decline in older patients

The line between healthy aged kidneys and true CKD is poorly defined.

How aging affects the kidneys

After 18 years of age, there is a progressive decline in an individual's number of nephrons – about 7000 'drop out' per year. This loss has no effect on renal function until about the fourth decade, when renal function declines progressively, with the GFR falling by about 1ml/min/1.73m² per year.

Aging is associated with a number of morphological changes within the kidneys which are thought to lead to this nephron drop-out. One is glomerulosclerosis – glomeruli show compensatory hypertrophy on histological examination of the kidney, presumably because of the demands placed upon them as other glomeruli fail. The renal tubules also progressively atrophy, causing loss of tubular function.

Renal blood flow declines after the age of 40 and vascular resistance increases, so that less blood reaches the glomeruli for filtration. Atherosclerotic damage to the larger vessels supplying the kidneys, further reduces renal perfusion.

As a result of ischaemia, atrophy, sclerosis and fibrosis, the kidneys lose mass. Kidney measurements on ultrasound usually show shrinkage in elderly patients, similar to that seen in CKD. So is loss of renal function in association with aging a disease or a normal phenomenon? Does a low GFR have the same health implications in the elderly as it does in younger patients?

The effect of age on eGFR

The graph (right) represents the relationship between GFR (measured accurately by isotope methods) and age. These are measurements of 'normal' kidney function stratified by age. In the graph, superimposed on the GFR ranges at each age, are the eGFR-based criteria for the various stages of CKD. There is an overlap between 'normal' renal function in some people over 70 (and most of those over 80) and CKD stage 3.

Although this decline in GFR (and by implication, eGFR) is accepted, the classification of CKD, and the treatment guidelines, take no account of age. According to NICE guidelines, the management of a 30-year-old and an 85-year-old, each with an eGFR of 50ml/min/1.73m², is the same. It might be argued that the MDRD formula includes a correction for age. But the validity of this in those aged 75 or more has not

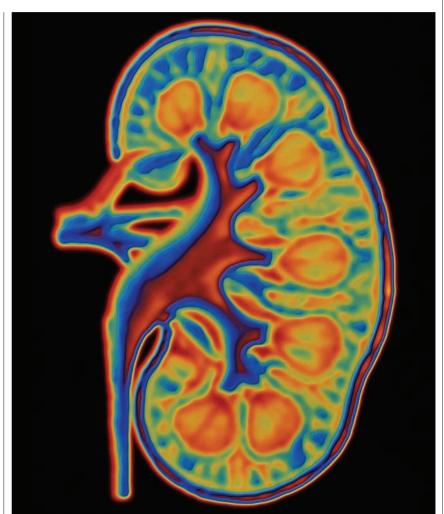
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The line between healthy aged kidneys and true CKD is poorly defined

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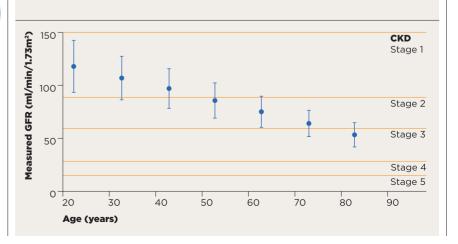
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Normal kidney function with increasing age



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been established. Also, the relationship between eGFR and true renal function is less reliable when the degree of renal impairment is small. So the formula is least reliable at the tipping point between normal renal function and stage 3a CKD. As the graph shows, accepted normal renal function in the very elderly is close to the defined lower limit of normal renal function. If there is an error in the eGFR that underestimates renal function in the elderly, a disproportionate number of otherwise healthy individuals will be defined as having CKD stage 3a.

Since the introduction of eGFR measurement, there has been a great increase in the number of people diagnosed with CKD. Just under 10% of the general population have CKD stages 3a-5, and most of these people are elderly. In fact, in individuals aged over 85, CKD 3a-5 is more common than normal renal function. In the absence of an upper age limit for entry onto primary care CKD registers, these elderly patients are contributing to the so-called 'epidemic' of CKD and may receive the same monitoring and treatment as their younger counterparts. Is this justified?

Even when a correction is made for comorbidities, there appears to be a correlation between low eGFR and increased mortality. But the impact of this effect decreases with increasing age. While some studies haven't identified any relationship between mortality and eGFR in subjects over 75 years, others have shown that a significant adverse effect on outcomes only becomes measurable when the eGFR is below 45ml/min/1.73m² (CKD stage 3b or worse).

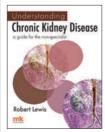
It is significant that individuals over 75 with stage 3a CKD may be at no increased risk of death compared with those with an eGFR greater than 6oml/min/1.73m2. And interventions to reduce an illusory risk are likely to do more harm than good. The fact that 40% of elderly patients on primary care CKD registers are unaware of their diagnosis suggests that GPs understandably use their judgement to assess the significance of stage 3a CKD in elderly people. The following factors may be relevant in guiding decision-making:

1 Proteinuria – an albumin:creatinine ratio >30mg/mmol (protein:creatinine ratio >50mg/mmol) identifies individuals at increased risk of mortality and adverse cardiovascular outcomes. Proteinuria is not a feature of the aging kidney. 2 Subjective assessment – most healthcare professionals can judge the difference between a 'good 80-year-old' and someone with a limited prognosis. 3 Views of the patient - many older people express a reluctance to take lots of drugs to manage renal and vascular risk.

Dr Robert Lewis is a consultant nephrologist at Portsmouth Hospitals NHS Trust

Key points

- Using the accepted definition based on eGFR, CKD is present in about 65% of people over 85 years of age.
- Renal function declines with age; the significance of an eGFR of 45-60ml/ min/1.73m² in an otherwise well individual over 75 years remains unsubstantiated.
- In this age group, evidence of an adverse effect on outcomes first becomes apparent at an eGFR less than 45ml/ min/1.73m² and, even then, is much less striking than in younger subjects.
- Inappropriate diagnosis of CKD can cause harm: individuals suffer unnecessary anxiety, may be financially disadvantaged (for example, with increased payments for holiday and life insurance) and may receive unnecessary treatment.
- Other factors can help guide decision making, such as proteinuria. subjective assessment and patient preference.



This article is an extract from **Understanding Chronic Kidney** Disease: A guide for the non-specialist by Dr Robert Lewis. ISBN: 9781905539741 (M&K Publishing); available from: mkupdate.co.uk priced £25.00.

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TOP TEN TIPS



Slipped upper femoral epiphysis is most common in overweight boys

The limping child

Professor Helen Foster, Dr Hannah Dean and Dr Eve Smith's

hints on assessing a child with an acquired limp

A limp persisting beyond four weeks warrants referral

A limp which has been present since the child started walking may be a congenital or neuromuscular disorder but this article will cover assessment of an acquired limp. Assess limping children carefully to exclude rare but serious causes of limp.¹

If the diagnosis is not clear then it is important parents know when to reattend. A limp persisting beyond four weeks without a confirmed diagnosis warrants a referral to assess for childhood arthritis. Reactive arthritis – arthritis following infection – shouldn't persist beyond six weeks.

Age is key in the differential diagnosis

Age is very helpful in determining the differential diagnosis of the limping child. Trauma, infection, reactive arthritis, malignancy, referred pain and juvenile idiopathic arthritis should be considered at any age. But other conditions are more likely in different age groups.

Preschool: toddler's fracture, missed developmental dysplasia of the hip, neuroblastoma, acute lymphoblastic leukaemia.

Four to 10 years: transient synovitis,

Perthes' disease, acute lymphoblastic leukaemia.

11 to 16 years: anterior knee pain, slipped upper femoral epiphysis, primary bone tumours.

Do not attribute limp to growing pains

Growing pains can occur in children between three and 12 years and importantly do not cause daytime symptoms (including limp), do not affect daily activities, are always symmetrical, and are not limited to joints – these symptoms are red flags.

A feverish child with a limp needs same-day assessment

Most paediatricians prefer that feverish children with a limp be referred same-day to exclude septic arthritis and osteomyelitis – especially where there is complete non-weight bearing, refusal to move the limb or severe pain. Even if a GP is considering doing X-rays and bloods followed by observation, we recommend referral anyway - these children will then be in the system if features of septic arthritis appear.

Immunocompromised children may not display typical features of sepsis so careful assessment is crucial. Also consider urgent assessment if a child is systemically unwell, under two years, non-weight bearing, or if there is concern about non-accidental injury (NAI). Never ignore red flags for malignancy or sepsis:

- systemic upset
- non-remitting pain
- night pain
- weight loss
- localised bone pain
- night sweats
- pallor
- unexplained bruising
- fever
- lymphadenopathy
- hepatosplenomegaly.

Look for a history of trauma

It is important to consider trauma, especially in younger ambulant children from whom the history may not be forthcoming. But trauma is common and often a swollen joint is only noticed after a coincidental injury. Consider the mechanism of injury – does it sound significant enough to cause a limp, or is the trauma a red herring?

Think beyond the hip as a cause of limp

The history in small children can be vague and history alone has been shown to be poor at localising pathology to a particular joint or even limb.² All joints should be screened in children with a limp. A quick and simple musculoskeletal examination called pGALS – paediatric gait, arms, legs and spine examination - can help identify abnormal joints which can then be more closely examined using a "look, feel, move" approach.³

It will also help to identify joint pathology elsewhere which may otherwise be missed. Limp can also be a presentation of non-musculoskeletal problems. It is important to consider the possibility of referred pain from the abdomen (e.g. urine infection, hernia, appendicitis), chest (pneumonia), testes (torsion) or the spine.

Insidious onset limp – with worsening pain on activity – should prompt you to think of Perthes' disease

Perthes' disease is an avascular necrosis of the femoral head and can be bilateral. It is most common in boys between four and eight years old but can also occur in girls. Children present with insidious onset limp or pain which may be referred to the thigh or knee and is worse with activity. Children may be non-weight bearing with an acute or chronic presentation. Examination of the hip may reveal limited abduction and internal rotation, and prompt orthopaedic review is essential.

Consider non-accidental injury if the history doesn't fit what you're seeing

Consider NAI, particularly in younger children with delayed presentation or if the history does not fit with the presentation or child's



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pulsetoday.co.uk/ ttt-skin-reactions developmental stage. Other red flags for NAI include unkempt appearance and previous or multiple injuries. Concerns require urgent assessment and should trigger local safeguarding policies.

Consider slipped upper femoral epiphysis in overweight boys over 10

Slipped upper femoral epiphysis is most common in overweight boys over the age of 10 but can also occur in tall, thin teenagers who have recently undergone a growth spurt. An acute epiphyseal slip is painful but a chronic slip is more common and symptoms tend to be indolent. Between 25% and 40% of cases are bilateral. Pain may be felt in the hip or referred to the knee, and is made worse by running and jumping. Trendelenburg gait may be apparent. Diagnosis relies on anteroposterior and 'frog lateral' radiographs of both hips, and diagnostic delay can result in much worse outcomes.

Juvenile idiopathic arthritis presents very differently to adult rheumatoid arthritis

JIA is defined as joint swelling for more than six weeks, presenting before the age of 16 in the absence of other causes. There is a risk of chronic uveitis which is usually asymptomatic and which - if not detected and treated - can result in visual loss. Presentation is variable; pain may not be a prominent verbalised feature, especially in the very young. Parents may notice a limp or refusal to stand, especially in the morning or after periods of inactivity, due to joint stiffness. On probing, parents may report a change in mood or activities (such as returning to bottom shuffling). Joint swelling may be subtle, especially in bilateral disease or in a chubby toddler. Even if musculoskeletal examination is inconclusive, refer if the history is suggestive of JIA.4

There are no diagnostic tests for JIA – investigations may be normal. Don't delay referral while waiting for results.

Professor Helen Foster is a consultant paediatric rheumatologist, Dr Hannah Dean is a GP registrar, Dr Eve Smith is a clinical research associate in paediatric rheumatology, all at Great North Children's Hospital, Newcastle-upon-Tyne. Dr Sharmila Jandial, consultant paediatric rheumatologist, also contributed to this article

• This article was produced with the British Society for Paediatric and Adolescent Rheumatology. Membership is open to healthcare professionals involved in the care of children and adolescents by paediatric rheumatology departments. BSPAR aims to improve provision and quality of healthcare for patients with rheumatic disease in childhood and adolescence, supporting clinical care, research, education and training. Go to bspar.org.uk

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Further reading

To watch the pGALS video and get further information, go to Arthritis Research UK's education pages at arthritisresearch.uk.org

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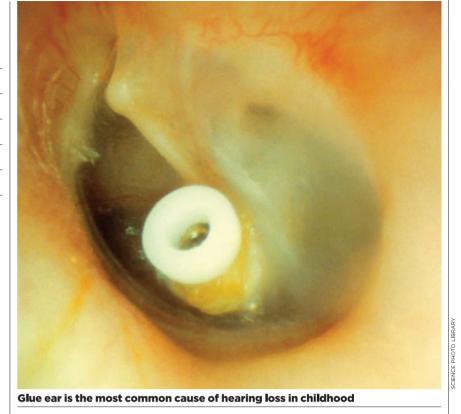
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THE INFORMATION

Glue ear

Consultant ENT surgeon

Mr Peter Robb discusses how
to manage this common
primary care presentation,
using PUNs and DENs



The patient's unmet needs (PUNs)

A five-year-old girl presents with her mother who has concerns about the girl's hearing. She has to turn the TV up loud, tends to shout and sometimes misses what her teacher says at school – although her mother thinks that, at times, she simply isn't concentrating. On examination, the child is a mouth breather and both tympanic membranes look dull and grey. You diagnose likely glue ear. The mother is keen for you to 'test her hearing', and wonders whether her daughter should be referred because her older sibling required grommets.

The doctor's educational needs (DENs)

Can the GP realistically perform a valid screening hearing test in the surgery? Or should all such children be referred for formal audiology?

It is difficult to perform a valid hearing assessment in a small child during a busy GP surgery. While there is now universal neonatal hearing screening in the UK, a child may pass the screening and then develop sensorineural hearing loss, either because of intrauterine infection, or acquired postnatal infection.

If the parents are concerned and there are positive clinical findings of glue ear, formal audiology would be warranted. Depending on local access, this will normally be to a local children's ENT clinic for pure tone audiometry.

Children younger than four years should initially be referred to the local second tier paediatric community audiology clinic as it will not be possible to perform reliable pure tone audiometry in an ENT clinic.



Extra Q&As

Go to the online version of this article for advice on surgical options to treat glue ear pulsetoday.co.uk/

Further reading

info

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What is the natural history of this condition?

Glue ear (otitis media with effusion) is the most common cause of hearing loss in childhood. Prevalence peaks at around two years, and again at around five years of age. By the age of 10, about 80% of children will have had glue ear.

Glue ear may be asymptomatic and parents may not notice hearing loss. In young children, poor balance, and speech and language delay are common presenting features. In school-age children, teachers may note inattention or poor social behaviour where background noise is a problem. When asked, older children may be aware that they cannot hear well. Untreated, most cases resolve within three months and 90% within six months.

What conservative measures can be tried, and for how long?

The initial management of glue ear is hearing assessment and active monitoring. This depends on timely referral for hearing testing, repeated three months later to assess resolution or the need for intervention. Reassure parents that resolution is common and medical treatment is not helpful. There is no evidence that antibiotics, decongestants, antihistamines or intranasal steroids are beneficial. And there is no evidence for alternative or complementary remedies. Nasal autoinflation of the Eustachian tubes with a balloon may be helpful during the period when natural resolution can occur, but the need to use the balloon three or more times daily is often a drawback for children.

Which children should be referred, and when?

A child with a history and new diagnosis of glue ear should be referred for hearing assessment and active monitoring. Most children will have to wait about three months to be seen. Referral does not imply the need for surgical treatment, but to document the hearing thresholds and middle ear tympanometry, repeating these over a three-month period, after which persistent glue ear might require intervention.

Are there 'special groups' who warrant more urgent action – and is the family history of any relevance?
Children with some conditions, such as Down's syndrome and cleft palate, are more likely to develop persistent, recurrent glue ear. These children are usually under surveillance by a developmental paediatrician or in a regional cleft service. If not, early referral is recommended. For children where persistent glue ear is predictable, hearing aids are a helpful option to avoid repeated operations.

The genetics of otitis media with effusion are currently being studied, and it is likely that a familial disposition will be shown.

Mr Peter Robb is a consultant ENT surgeon at Epsom Hospital and Surrey Children's ENT Clinic, Ashtead Hospital.

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PICTURE QUIZ

Environmental rashes

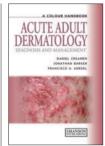
These four patients presented with rashes. Look at the pictures and case histories below to make the diagnosis



This woman has just returned from a holiday in Cuba – and this intensely itchy rash appeared on both arms during the first week. Now that she has been back in the UK for a couple of days, it is already starting to improve.



According to this woman's partner, who attends with her, this odd rash had appeared on the patient's arm overnight. There are no lesions elsewhere, and – unlike her partner – she seems remarkably unconcerned about the rash.



These cases are taken from Acute Adult Dermatology – A Colour Handbook by Daniel Creamer, Jonathan Barker and Francisco A. Kerdel. ISBN 9781840761023 (Manson Publishing); available from mansonpublishing. com/colour handbooks and all good booksellers priced £29.95



This woman developed red nodules on the side of each thigh over the previous week. Initially, they were itchy but are now quite painful. She recalls similar episodes the last two winters.



Only hours after arriving for a short break in the Canary Isles, this young woman developed a very itchy rash on her neck and forearms. She is on no medication and otherwise feels well.



Extra Q&A
Go to the online
version of this
article for an extra
case: a woman
presents in the
emergency
surgery with
painful lesions on
her forearm.
pulsetoday.co.uk/

enviro-rashes

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Answers

Polymorphic light eruption is an inflammatory dermatosis caused by a reaction to the UV component of sunlight. The eruption only involves to eight hours of exposure. Lesions are small, red, intensely itchy papules. Plaques, vesicles and blisters may also be seen. Following sun avoidance, besions usually resolve without scarring after one to three days.

4 Polymorphic light eruption

леека.

Chilblains are localised inflammatory lesions which occur as an abnormal reaction to cold. Chilblains can be associated with systemic disorders cuch as systemic lupus erythematosus, leukaemia and anorexia nervosa. Chilblains are develop on acral skin (fingers, toes, heels, thighs, nose and ears). Keep the affected area warm - chilblains usually stfected area warm - chilblains usually resolve spontaneously after a few

3 Chilblains

The lesions of dermatitis artefacta are inflicted by the patient—who is usually fully aware of their actions. Exposed areas, such as face, hands, arms and legs, are commonly involved and the clinical history often lacks detail.

Lesions may be unusual shapes – straight lines and angulations – and monomorphic. This case was probably monomorphic. This case was probably caused by iron wool or a cheese grafter. Occlusive bandaging allows lesions to heal, but the patient may require assessment by a psychiatrist.

2 Dermatitis artefacta

Miliaria rubra is a sweat rash which occurs most commonly in hot, humid environments and is known as 'prickly heat'. Miliaria rubra consists of tiny, monomorphic, red papules which occur predominantly on the torso and in the flexures. The lesions produce in the flexures. The lesions produce prickling discomfort rather than itching. Keep the affected body parts cool - for example with a cool shower and use emollient therapy.

Filiaria F

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A CASE THAT CHANGED MY PRACTICE

An anxious patient with a headache

Dr David Kernick, headache GPSI, discusses the case of a woman who was worried about her worsening headaches

The case

My patient – a 35-year-old nursing sister – had a long history of migraine. But over the last three months her headaches had been increasing in frequency and she was now getting headaches almost every day. She was taking regular cocodamol on five days each week.

I diagnosed migraine with medication overuse headache but she insisted on having an MRI – a colleague of hers at work had had a similar headache and developed a brain tumour. So, against my better judgement, I ordered an MRI.

The outcome

The MRI showed a small, 4mm aneurysm in the posterior cerebral circulation which was totally unrelated to her headache.

She had an appointment with the neurosurgeon who told her that the natural history of lesions of this size was not known. If he operated she would have an 8% risk of stroke and a 2% risk of death, and he suggested that she was imaged again in a year's time.

Now my anxious patient had become a nervous wreck!

What I learned

The increasing sensitivity of modern imaging technology has overtaken our ability to understand the abnormalities it reveals. This is particularly relevant in the case of MRI brain scans, where incidental abnormality rates of up to 10% have been reported. Not only do these results have implications for patient anxiety but also for their future insurance applications.

The incidence of primary brain tumours is between six and 10 per 100,000 population per year. Some 72% of brain tumours will present above the age of 50 and 10% of tumours will present

Do you have a story to share?

Submit your own 'a case that changed my practice' feature – in the format of Dr Kernick's article – to rhiannon. smith@pulsetoday.co.uk. Advise your GP colleagues on what you learned. There is a £100 honorarium for every article published.





I now think carefully about why an investigation is needed

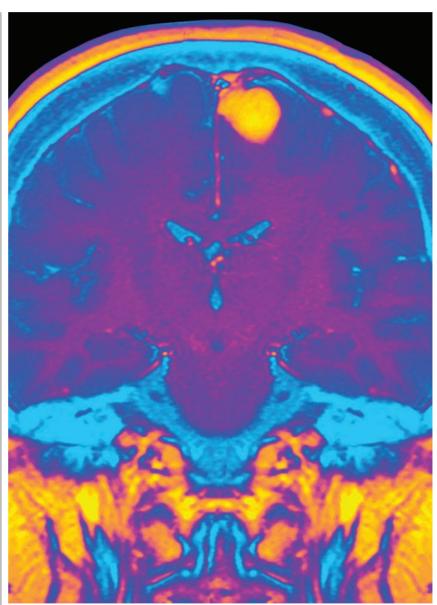
References
1 NICE. Headaches:

diagnosis and

NICE CG150

management of

people and adults. September 2012.



Incidental abnormality rates of up to 10% have been reported on MRI brain scans

with isolated headache. Only 0.09% of all headache presentations in primary care are because of a primary tumour. Brain metastases occur in 20-40% of patients with cancer elsewhere.

How this changed my practice

I now think more carefully about why an investigation is needed when patients present with headache, and provide them with more comprehensive information

on the pros and cons of screening. The new NICE headache guidelines¹ advise that imaging should not be done for reassurance alone and if imaging is considered it should be for a good clinical reason and following discussion with the patient on the benefits and drawbacks of this investigation.

Dr David Kernick is a headache GPSI in Exeter and RCGP headache champion

CE PHOTO LIBRARY

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Latest modules



Hot topics

Skin and nail infections 2 CPD HOURS

This case-based module is an update on common skin and nail infections and also Lyme disease, hidradenitis suppurativa and PVL-positive *Staphylococcus aureus* infection.



Guideline debrief

Ectopic pregnancy and miscarriage 2 CPD HOURS

Medical and expectant management of miscarriage as alternatives to surgery have shifted more of this work into primary care and this module will bring you up to speed with the new NICE guidance.

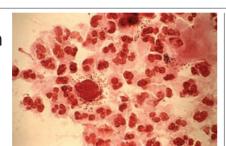


End-of-life care

Advance care planning 1 CPD HOUR

GP and hospice physician Dr Peter Nightingale's guide to initiating the discussion, assessing capacity and the three options for formalising a decision on end-of-life care.

Also new this month



Key questions

Vaginal discharge 1.5 CPD HOURS

'Simple, interesting, informative but at the same time comprehensive' *Dr Shalini Gadiyar*



Key questions

Movement disorders in the elderly 1.5 CPD HOURS

'Excellent, succinct and very helpful to GPs'

Dr Chris Waldrum



Prescribing

Controlling your prescribing spend 1 CPD HOUR

'Well written with all aspects of prescribing covered - I strongly suggest all GPs read this' Dr Abdulrehman Rajpura



Guideline debrief

Psoriasis 2 CPD HOURS

'Excellent. Very comprehensive and will alter my management' Dr Andrew Doyle



Hot topics

Iron deficiency anaemia 2 CPD HOURS

'Excellent, very educative and relevant to primary care' Dr Kamla Mahto



Guideline debrief

Perinatal mental health 2 CPD HOURS

'Excellent update on a complex clinical problem'

Dr Matthew Hughes

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> Applications & CV to Mr. D. Hicken laurel.house@nhs.net

Please Note: Applications from agencies will NOT be considered

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- Send your application to careers@rfdsse.org.au

Royal Flying Doctor Service

PEVERLEY

From the cradle to the grave

When the last World War Two veteran on his list died, Peverley found himself weighing up the pros and cons of abandoning paperwork to pay his respects

Dr Phil Peverley is a GP in Sunderland and was PPA and BSME columnist of the year in 2012. Read more Peverley columns at pulsetoday. co.uk/peverley t is almost never a good idea to lie to your patients and it is difficult to imagine a situation when doing so would have a good outcome. As family GPs, we have a duty of honesty.

But I had to tell a lie the other day, and it's troubling me. I went out on a home visit to see an old gentleman patient of mine, who has been gradually dying of an unknown primary cancer for several months. It's not been a bad illness, as these things go. He's been OK on the whole, in the circumstances.

I had got into the habit of visiting every week and, as usual, I parked my car and walked around the corner to his house. Where, to my horror, I found him being loaded into a hearse by six blokes dressed as extras out of *Oliver Twist*; top hats, black coats, the works. The only things missing were the black-plumed horses. His daughter dashed over. 'Oh doctor, how good of you to come and pay your respects! Dad would have been so pleased!'

Dilemma! Seconds before I had been expecting my usual chat with my patient about Rommel and Montgomery (he had been a Desert Rat) and now suddenly I'm in deep doo-doo.

Do I tell the truth? ('Sorry love, I didn't know he was dead. I wish you'd let us know earlier. I've got a lot of paperwork back at

the surgery, I could have been doing that.')

Or do I lie? ('I couldn't let him pass without seeing him off. You must all be devastated. How *are* you? Can I do anything to help?')

I'll let you guess which option I chose, on the spur of the moment, but I'm bloody glad she didn't notice the doctor's bag

I was carrying and ask herself just why I would have brought that to a funeral.

'Would you come to the crematorium with us doctor? And then back here afterwards? Dad would have been *so* pleased!'

I politely declined and skedaddled, but the incident set me thinking. Actually, I would have liked to attend his funeral; he was, I think, the last proper World War Two veteran under my care, and his passing was the passing of an era, for me.

And I genuinely liked him and enjoyed his company. Why should I have to forgo an act of genuine respect just because I've got a pile of bloody insurance forms to fill in? Which, in the long term, is more important?



In practical terms, we just can't do three funerals a week (the average in our practice), even though the savings in mini pork pies and sandwiches with the crusts cut off would be significant. Time just does not allow. But I

like the idea. Bevan's vision of cradle-to-grave healthcare would be realised, in very literal terms.

But then again, not all my patients are like the Desert Rat. Some funerals I would be honoured to go to; some would just be a profoundly irritating and hypocritical waste of time.

How would you choose? Would a two-tier 'deathcare' system develop, with some patients blessed with their GP's attendance at their funeral and others not? Would we end up with a bloody funeral rota?

Maybe some things are best left the way they are.

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1. Scheltens P, et al. J Alzheimers Dis, 2012

2. Scheltens P, et al. Alzheimers Dement. 2010 Jan;6(1):1-10.e1.

3. Kennedy EP Weiss SB (1956) J Biol Chem 222(1):193-214



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References: 1. Rose JE et al. Nicotine & Tobacco Research 2009; 11 (9):

Adverse events should be reported. Reporting forms and information can be found at http://www.mhra.gov.uk/yellowcard. Adverse events should also be reported to GlaxoSmithKline Consumer Healthcare 0500 100 222.

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