

- in the age group 10-20 years. *Int J Ther Rehabil* 2006;13:511-6.
- 3 Frank AO, Ward JH, Orwell NJ, McCullagh C, Belcher M. Introduction of the new NHS Electric Powered Indoor/outdoor Chair (EPIOC) service: benefits, risks and implications for prescribers. *Clin Rehabil* 2000;14:665-73.
 - 4 Evans S, Frank A, Neophytou C, De Souza LH. Older adults' use of, and satisfaction with, electric powered indoor /outdoor wheelchairs. *Age Ageing* 2007;36:431-5.
 - 5 Davies A, De Souza LH, Frank AO. Changes in the quality of life in severely disabled people following provision of powered indoor/outdoor chairs. *Disabil Rehabil* 2003;25:286-90.
 - 6 May M, Rugg S. Electrically powered indoor/outdoor wheelchairs: recipients views of their effects on occupational performance and quality of life. *Br J Occup Ther* 2010;73:2-12.
 - 7 Frank AO, Neophytou C, Frank J, De Souza LH. Electric Powered Indoor/outdoor Wheelchairs (EPIOCs): users views of influence on family, friends and carers. *Disabil Rehabil* (2010).
 - 8 Department of Health. *Powered indoor/outdoor wheelchairs for severely disabled people*. London: DH, 1996.
 - 9 Williams E. *Electronic assistive technology: a working party report of the British Society of Rehabilitation Medicine*. London: British Society of Rehabilitation Medicine, 2000:1-34.
 - 10 Richardson M, Frank AO. Electric powered wheelchairs for those with muscular dystrophy: problems of posture, pain and deformity. *Disabil Rehabil Assistive Technol* 2009;4:181-8.

Polymyalgia rheumatica

Editor – Dasgupta, writing on behalf of the polymyalgia rheumatica (PMR) guideline development group, presents a welcome and thorough overview of this common condition (*Clin Med* June 2010 pp 270-4). I have concerns with the recommended three-monthly 'lab monitoring' of full blood count, erythrocyte sedimentation rate/C-reactive protein (ESR/CRP), urea and electrolytes and glucose. The management of straightforward PMR is to relieve symptoms (and not to treat inflammation) until the condition runs its natural course. Steroid withdrawal should be based on the clinical picture and not on the level of ESR and this is alluded to in the article 'raised ESR/CRP without clinical symptoms is not an indication to continue corticosteroids'.

It is my belief, based on reviewing many patients with PMR and iatrogenic Cushing's/osteoporosis, that the main reason for the continuation of higher dose steroids is the regular checking of an ESR to follow disease activity. The secret to the successful management of straightforward PMR is, once the diagnosis has been made, never to check an ESR/CRP unless there is a clinical indication.

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In response

Editor – We thank Dr Morris for highlighting an important issue – the objectives of steroid treatment for polymyalgia rheumatica (PMR). Steroids are prescribed for their important effect on pain, disability and stiffness and the quality of life in untreated PMR is lower than in most other comparable conditions. On the other hand steroids also have many side effects and over-treatment based on raised inflammatory markers alone may prolong duration of treatment and induce treatment comorbidities such as fractures, diabetes, hypertension, weight gain and cataracts.

However, we now know that the PMR constitutes only one of many conditions that can present with bilateral shoulder pain and stiffness. Such conditions include late onset rheumatoid arthritis, other arthropathies, spondyloarthropathies and connective tissue diseases. Large vessel vasculitis may also present with polymyalgia, constitutional symptoms and raised inflammatory markers. Other serious pathology, such as systemic infection, disseminated cancer and so on, may also be mistaken as PMR and may have an initial response to high dose steroids.

We therefore stand by our recommendation of inflammatory marker testing in the context of a clinical review – especially in the first year of disease. Transient elevations of CRP/ESR may be due to common causes such as urinary or chest infections and urinalysis and chest radiographs may be considered. Persistent elevation in the presence of definite symptoms suggests partial or

non-response to steroids, search for alternative pathology or adjuvant immunosuppressives and a specialist referral. Persistent symptoms in the absence of elevated markers suggests evaluation of co-existing non-inflammatory conditions such as osteoarthritis, rotator cuff and other local shoulder conditions, fibromyalgia, etc. These conditions should be dealt with by explanation, reassurance and local treatments such as physiotherapy, injections and exercises; while the steroid dose is tapered.

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On behalf of the PMR guideline development group

A complicated hyperglycaemic emergency

Editor – I read with interest the article by Vidyarthi and Chowdhury describing a hyperosmolar non-ketotic diabetic emergency complicated by diabetes insipidus (*Clin Med* June 2010 pp 264-5).

I agree that these complex cases are best managed in a critical care environment where point of care testing is available to guide therapy. I feel a number of other features merit clarification, however. Firstly, the authors fail to emphasise that hyperglycaemia causes water shift from intracellular fluid (ICF) to extracellular fluid (ECF). Correction of hyperglycaemia thus causes an influx of water back into the ICF causing a rise in serum sodium despite reduced free water losses. As this rise is accompanied by an influx of water into the brain, osmotic demyelination syndrome (central pontine myelinolysis) should not arise, as long as serum osmolarity is falling. Conversely cerebral oedema can be a risk if serum osmolarity falls very rapidly with volume expansion. However, this danger may have been over-emphasised in this case where serum osmolarity paradoxically rose with therapy, attributed by the authors to diabetes insipidus of uncertain aetiology. I feel administration of large volumes of 0.9% saline may have contributed to this outcome. The patient described was in early shock with a mean

arterial blood pressure of 75 mmHg. Six litres of 0.9% saline were given for volume correction, certainly an over estimate of any ECF deficit – and potentially risking ECF volume overload and hyperchloraemic metabolic acidosis. Interestingly, the patient subsequently passed very dilute urine and became hypernatraemic beyond the rise expected by fall in serum glucose – so that they then were at risk of osmotic demyelination, a state that corrected with use of DDAVP. A similar scenario is well described in the correction of hyponatraemia associated with a volume deficit¹ where appropriate suppression of a hypovolaemic ADH signal results in a water diuresis (with low urinary osmolarity) and over-shoot hypernatraemia. I feel the case described is exactly analogous, with removal of hypovolaemia leading to water diuresis and significant over-elevation of serum sodium. This response is transient and physiological, although short duration treatment with DDAVP is appropriate.²

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References

- 1 Mohmand HK, Issa D, Ahmad Z *et al*. Hypertonic saline for hyponatremia: risk of inadvertent overcorrection. *Clin J Am Soc Nephrol* 2007;2:1110–17.
- 2 Perianayagam A, Sterns RH, Silver SM *et al*. DDAVP is effective in preventing and reversing inadvertent overcorrection of hyponatremia. *Clin J Am Soc Nephrol* 2008;3:331–6.

Clinical & Scientific letters

Letters not directly related to articles published in *Clinical Medicine* and presenting unpublished original data should be submitted for publication in this section. Clinical and scientific letters should not exceed 500 words and may include one table and up to five references.

A survey of acute neurology at a general hospital in the UK

The Association of British Neurologists (ABN) in their publication *Acute neurological emergencies in adults* (2002) recommend that all patients with acute neurological disease should be seen by a neurologist within 24 hours. We therefore undertook a study to consider the proportion of patients admitted with neurological symptoms to a district general hospital covering a population of 350,000 to determine what level of access they have to a neurologist.

During a two-week period (22 September 2008 and 5 October 2008) all the accepted medical admissions were reviewed. Patients

were grouped under a neurological category if the first differential diagnosis at the most senior review was a neurological disorder, or if the differential was yet unclear at the time of clerking but the presentation was predominantly neurological.

A total of 358 patients were admitted in this period, neurology had the highest proportion of admitted patients (93 patients or 26%). This was followed closely by cardiology (23%) and respiratory (22%). The other specialties made up the remainder (104 patients or 29%) (Fig 1).

Of the 93 neurological patients, most (21) were admitted for cognitive disorders, followed by ‘blackouts/seizures’ (19), falls (15), weakness (15), headache (11), dizziness/vertigo (10) and movement disorders (2). The mean age of these neurological patients was 70 (range 21–100); 40% were under 70 years of age; 55 patients were female and 38 were male. Only 10 patients (11%) were referred to the neurology team for specialist advice.

Our findings are similar to previous studies that have reported 19% of inpatients in general hospitals to have neurological symptoms. We were surprised by the low number of patients who were referred to a neurologist. The input of a neurologist has been shown to lead to a change in management of patients in approximately a third of

Fig 1. Primary reason for urgent medical admission by specialty.

