

letters

TO THE EDITOR

Please submit letters for the Editor's consideration within three weeks of receipt of the Journal. Letters should ideally be limited to 350 words, and sent by e-mail to: Clinicalmedicine@rcplondon.ac.uk

Rationing of medical care by age

Editor – I welcome your recent editorial for encouraging debate on this important issue (Robert Allan, *Clin Med* July/August pp 329–30). However, the concept of using age as the criteria for rationing medical care is primarily flawed. There is the temptation for using age as a surrogate for ageing but in this debate, age is not the issue; the issue is ageing and frailty. It is also important to recognise that ageing is not disease and disease is not ageing, but that in old age the disease may present as ageing.

There is no denying the fact that after the age of 60, the spectrum of pathology changes and disability rises steeply. Combinations of chronic pathology make predicting the course of illness far more complex. New patterns of disease emerge with altered response to treatment. Therefore, basic pathology, frailty and disability should be the main focus when managing patients. It will be a sad day for medicine if age alone is used for rationing healthcare and the prerogative of treating patients on clinical grounds is taken away from the attending physician.

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Current clinical uses of intravenous immunoglobulin

Editor – In their review article describing the clinical use of intravenous immunoglobulin (IVIg), El-Shanawany *et al* (*Clin Med* July/August 2006 pp 356–9) warn of anaphylaxis as a rare but severe adverse

effect. Previous reports suggest that anaphylaxis is associated with IgA deficiency and the presence of anti-IgA antibodies.¹ Population studies suggest the prevalence of IgA deficiency may be as high as 1:400 in healthy blood donors.² Although some guidelines do not recommend essential determination of IgA prior to IVIG, others advise testing.^{3,4} Fear of this potentially fatal complication may direct local hospital protocol.

In our experience the decision to test is often based on local availability and laboratory turnaround time. It is often junior doctors who are asked to arrange testing and prescribe therapy and it could therefore be argued that clear guidelines are needed to minimise patient risk. Although the clinical significance of IgA deficiency and anti-IgA antibodies is debated,⁵ we would welcome the thoughts of the authors on this issue.

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- 2 Cunningham-Rundles C. Physiology of IgA and IgA deficiency. *J Clin Immunol* 2001;21: 303–9.
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muscular diseases: present status and practical therapeutic guidelines. *Muscle Nerve* 1999;22:1479–97.

- 4 Brannagan TH, 3rd. Intravenous gamma-globulin (IVIg) for treatment of CIDP and related immune-mediated neuropathies. *Neurology* 2002;59(12 Suppl 6):S33–40.
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In response to Hull and Hull

Hull and Hull raise a number of important points in response to our article regarding IgA deficiency (IgAD), its diagnosis, testing for anti-IgA antibodies, and guidelines for the assessment of patient prior to the use of IVIG.

The reported incidence of selective IgA deficiency in the healthy population varies from 1:223 to 1:3000.¹ IgA deficiency can occur in isolation, where it may have little effect on health (though is associated with an increase in autoimmune conditions), or in association with deficiency of other immunoglobulin subclasses and classes as in common variable immunodeficiency.

Anti-IgA antibodies should only be tested for in individuals with confirmed absent IgA using a sensitive assay which measures levels down to 0.05 g/l. If IgA levels are found to be <0.05 g/l, then, as far as we are able to determine, the patient has a complete deficiency.² Patients with low but detectable IgA (partial IgA deficiency) are not at increased risk of reactions to IVIG.

Current assays measure IgG anti-IgA, but the correlation between the presence of these antibodies and reactions to IVIG is no longer as certain as was once thought. High titre anti-IgA have the best association with adverse reactions. Patients with IgM anti-IgA have been successfully treated with IVIG.³ It has also been argued that anaphylactic reactions to the small quantities of IgA in IVIG are likely to be due to IgE anti-IgA antibodies. However, this remains controversial and there is not a reliable and validated assay currently in use in diagnostic laboratories.

The urgency of the clinical setting will also affect the workup performed. In the acute setting it may be difficult to delay the administration of high dose IVIG (hdIVIg) while awaiting these results. The