It's not over 'til the fat lady sings

Sir,

Dr Cook and Dr O'Higgins' anaesthetic dilemma (Vol 60(5), 1999, p. 387) prompted me to highlight some issues. Dr Cook and his team, no doubt, managed the case safely with their judgment and clinical acumen, but the delay in providing pain relief with regional anaesthesia (RA) to the labouring idiopathic thrombocytopenic mother leaves some room for discussion.

I am not aware of any guideline of the Association of Obstetric Anaesthetists that recommends a safe level of platelets for RA in pregnancy, but the literature suggests the following.

Only 30–50x10^9/litre platelets are needed for normal clotting (Roizen, 1994). Platelet levels have been claimed by some to rise and by others to fall in pregnancy (Crawford, 1984) without obstetric complications.

Bleeding is uncommon with platelet counts above 50x10^9/litre. Platelet numbers between 50 and 100x10^9/litre and bleeding time in the upper normal range are relative contraindications for regional block (Breivik and Bogod, 1996).

Some authors believe that there is little evidence to support the use of bleeding time as a diagnostic test before RA (Anonymous, 1991), although some 'anesthesiologists' suggest that bleeding time should be routinely measured in those with platelet counts below 150x10^9/litre (Shnider and Levinson, 1994).

With the above views in mind, I believe that the epidural could be sited carefully earlier and there was no reason for delaying the epidural analgesia on the basis of the previous platelet counts (73,79 and 72x10^9/litre respectively), especially when the authors' own limit is 80x10^9/litre.

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Anonymous (1991) The bleeding time. *Lancet* **337:** 1447

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Sir,

We wish to thank Dr Palit for his interest in our anaesthetic dilemma, and agree that there is room for discussion. Clinically significant spinal haematoma complicating RA is so rare that its true incidence is unclear but is estimated at less than 1 per 100 000 cases (Stenseth et al, 1994). Wulf (1996) estimated the risk as 1 in 190 000 epidurals with a 95% confidence limit of 1 in 406 000 to 1 in 96 000. There are no randomized studies and no longitudinal studies of anything like the size needed to determine how much this small risk is increased by low platelet numbers or what is a 'safe' lower limit. The available evidence is restricted to case reports, a few small series of patients and the opinions of august bodies. In the ranking of evidence this is weak evidence.

Most cases of epidural haematoma associated with RA have occurred when there is an abnormality of coagulation (Knowles, 1996; Sage, 1990). In one review of 51 case reports, five were associated with thrombocytopenia or abnormal platelet function (Wulf, 1996). Published guidelines vary in the accepted lowest platelet level for safe RA techniques from 50–100x10^9/litre (Stenseth et al, 1994; Tekkok et al, 1991; Owens et al, 1986; Vandermeulen et al, 1993). For instance, the Norwegian guidelines state that regional techniques should not be used when platelet levels are below 40x10^9/litre and only used with provisos between 50–100x10^9/litre (Stenseth et al, 1994). In contrast Sage (1990) reviewed the subject and regarded 100x10^9/litre as the safe lower limit but stated that this is arbitrary.

Uncomplicated RA has been reported in a series of 30 patients with 69–98x10^9/litre platelets (Beilin et al, 1997) and in one patient with almost no platelets (Hew-Wing et al, 1989). Avoiding a rare event in a small series of cases does not provide evidence that that practice is safe. We did not wish to 'get away with it' but to 'do no harm'. The patient's opinion when we discussed these risks mirrored this.

Bleeding time is very operator dependent and like thromboelastography has failed to be widely accepted as sensitive or predictive. We did not feel either would be helpful. With regard to our limit of 80x10^9/litre, while this is arbitrary it is based on consideration of the available evidence. It is our limit, therefore to site the block, in a non-emergency situation, when platelet levels were consistently below this figure would obviate the point of having a limit.

In the absence of strong evidence one must rely on extrapolation of weak evidence, experience and 'clinical judgment'. This is one reason why we considered this case to pose a dilemma. The successful outcome of this one case by no way means that we managed it in the best, safest or only way possible.

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Correction

In the article *Stenting in peripheral vascular disease* (Vol 60(9), 1999, p. 632), *Figure 2b* was reproduced incorrectly as a result of an editorial error. The correct *Figures 2a* and *b* are reproduced below. We apologise for any inconvenience caused.

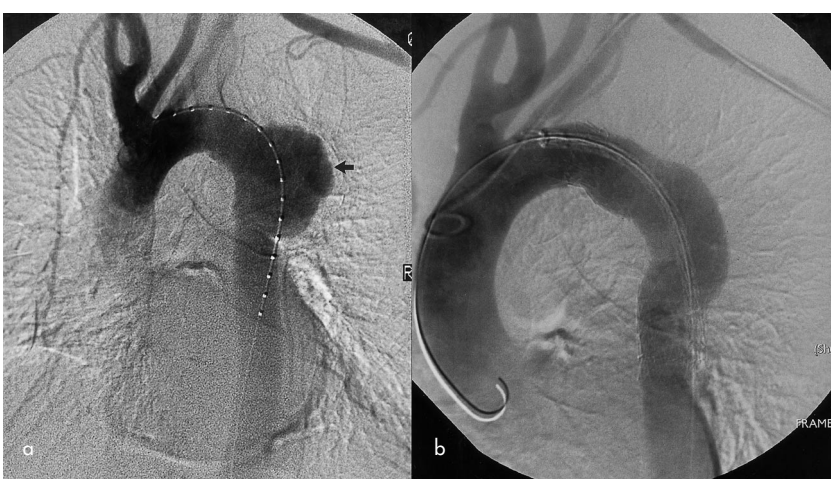

Figure 2. a. Arch aortogram, with a measuring catheter, showing a saccular aneurysm of the descending thoracic aorta (arrow). b. The aneurysm sac has been excluded from the circulation by the placement of a stent-graft.

Evidence-based stroke management

Sir,

I read with interest the article 'Evidence based guidelines for early stroke management' (Vol 60(2), 1999, p. 105) and found it very good. However, I would like to highlight the following points:

1. Despite lack of evidence from controlled studies it is agreed that stroke patients should have early involvement of members of a multidisciplinary team (guideline B/C).
2. Infarction changes may take 1–2 days to appear on a computed tomography (CT) scan and CT is negative in 33–50% patients presenting with clinically established stroke (Foulkes et al, 1988).
3. Contrast enhanced CT is of limited value.
4. There may be benefit in using angiotensin-converting enzyme inhibitors, α-adrenergic blockers or β-blockers which preserve cerebral blood flow in preference to calcium blockers (Powers, 1993).
5. The benefits of anticoagulation are greatest in patients with valvular heart disease, chronic heart failure or previous stroke, and need to be balanced by the risks involved (guideline B/C).
6. Cholesterol lowering studies (4S, CARE) and secondary atrial fibrillation studies (EAFT) have excluded patients above the age of 75 years and there is a lack of data specific to patients over 75 years of age.
7. Available clinical data do not support the use of statins for stroke prevention in subjects with no manifest ischaemic heart disease (Furberg, 1999).
8. Forty patients are needed to prevent one stroke over 2 years treatment with aspirin or dipyridamole alone, but only 18 patients are needed to be treated with combined therapy as the results are additive (Bandolier, 1997).

These are some of useful practical points in 'early' stroke management.

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Bandolier (1997) NNT for stroke prevention. *Bandolier* **38:** 2–3
Foulkes MA, Wolf PA, Price TR, Mohr JP, Hier DB (1988) The Stroke Data Bank: design, methods and baseline characteristics. *Stroke* **19:** 547–54
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Sir,

Dr Gupta makes a number of points, which we would like to address. Our article specifically provided evidence-based guidelines on the management of stroke for the admitting doctor during the first 24 hours or so. There is no evidence that involvement of a multidisciplinary rehabilitation team this early on alters outcome following stroke.

We agree that an early CT scan (<48 hours) may not detect infarction and we state this as a limitation in *Figure 2*. We emphasize that the diagnosis of stroke is primarily clinical and that the value of an early CT scan is to exclude cerebral haemorrhage. A contrast enhanced CT may be useful in excluding non-vascular pathology when the history is equivocal and includes the possibility of a cerebral tumour.

As we point out, the management of hypertension after a stroke is contentious and will remain so until substantive trial evidence becomes available. The choice of antihypertensive agent is even more contentious. The effects of various antihypertensive drugs on cerebral blood flow has only been studied in detail on non-ischaemic animal brains. To extrapolate from these experiments to a clinical guideline would be unwise. The whole issue of lowering blood pressure after stroke may become clearer once the results of the PROGRESS study (Perindopril Protection Against Recurrent Stroke Study) become available.

Our article was not intended to give guidelines on atrial fibrillation (and other risk factors) in primary prevention of stroke. We agree that whenever anticoagulation is used, the benefits need to be balanced against the risks involved.

The evidence for statin use is strongest for patients with symptomatic ischaemic heart disease and this was made explicit in guideline 15.

We agree that the European Stroke Prevention Trial II has been important in raising the issue of synergy between antiplatelet drugs with different mode of action. However, this needs to be placed in the context of other randomized controlled trials (14 direct comparison trials and 34 indirect comparison trials) which investigated the combination of aspirin and dipyridamole, the pooled efficacy estimates of which is no different to aspirin alone (Antiplatelet Trialists' Collaboration).

It is regrettable that so many large trials have recruited few older people. This represents a serious limitation to generalizability in the real clinical world: it is where evidence-based and practice-based medicine overlap and where common sense and clinical judgment are called for. Clinical guidelines are true to their name — best clinical guidelines, not clinical rules.

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Guillain–Barré syndrome and osteoporosis

Sir,

Dr Hatfield and Dr Collin give a very crisp case report of a patient with Guillain–Barré syndrome developing osteoporosis and fracture (Vol 60(3), 1999, p. 216), which is very timely for us. We have had a 2-year-old girl in our paediatric intensive care unit for the last 2 months, admitted with Guillain–Barré syndrome. She has been on ventilatory support since admission, is bed-ridden and has shown a very mild improvement.

We do not have the facility of dual energy X-ray absorptiometry (DEXA). However, her biochemical parameters, including calcium, phosphorus and alkaline phosphatase levels, are normal. In view of her condition, what is the risk of her developing osteoporosis? Can we treat her with prophylactic therapy? Does prophylactic therapy have a role in such cases?

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Sir,

We thank Dr Manzar for highlighting the concerns related to the immobilization of a 2-year-old with Guillain–Barré syndrome. This patient will be at risk of osteoporosis because of her immobilization and we would suggest that use of steroids is kept as low as possible. This case represents the most severe end of the spectrum in Guillain–Barré syndrome. Hopefully there will be some improvement in limb strength and ongoing ventilatory support will not be required. Long-term mobility problems are likely. The severity of the disease in children has been compared with adults and no significant differences found (Kleyweg et al, 1989). Unfractionated heparin should be avoided in favour of low molecular weight heparin.

Optimal intake of calcium should be achieved throughout the course of the illness. Bone densitometry will not be helpful because there are no norms for this age group. There is inadequate experience of bisphosphonate use in children and because complications include hypomineralization and fractures it is not recommended.

We would recommend bone density screening by DEXA during adolescence to allow adequate time for treatment before age of attainment of peak bone mass. Long-term bone density attainment is probably more important and peak bone mass achieved in adolescence after skeletal growth will be a major determinant of osteoporosis and fracture risk in later life. Caution should be exercised in bone density analysis in childhood and adolescence because results are influenced by growth-dependent skeletal changes (Schonau, 1998).

Intensive rehabilitation will be required with proper positioning, gentle strengthening and progressive resistance exercises. The risk of falls needs to be minimized and a standing frame used to improve in bone density by weight bearing. Orthoses may help proper positioning and optimize residual motor function.

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Kleyweg RP, van der Meche FG, Loonen MC, de Jonge J, Knip B (1989) The natural history of the Guillain–Barré syndrome in 18 children and 50 adults. *J Neurol Neurosurg Psychiatry* **52:** 853–6
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