members need time to read the applications. In fact, 50 days is a much more realistic figure.

The major issue raised in the discussion is the risk that commercial practice might be jeopardised. This is not a matter of the greatest importance for patients taking part in research projects.

I submit that there is no evidence in the article that a central ethics committee would protect the interests of research subjects better than LRECs.

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The medical management of terminal illness

Sir—Lest there is any misunderstanding following the helpful statement on 'The medical management of terminal illness' from the Committee on Ethical Issues in Medicine (prepared by Sir Douglas Black, October 1993, page 397), clinicians should note that the Law Lords made clear in the case of Airedale NHS Trust vs Tony Bland that their ruling on treatment withdrawal in persistent vegetative state applied only to that case. Any decisions to withdraw treatment in PVS will require individual application to the High Court.

To make such an application, clinicians must have the opinion of two independent neurologists that the victim is indeed in a persistent vegetative state and must demonstrate that at least six months' active rehabilitation has been delivered and that the patient has been under close observation for at least one year without any sign of improvement. The opinions of any family or carers should also be given great weight.

Clinicians should not take the Bland case as a precedent which will enable them to withdraw treatment in PVS without recourse to the law.

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Research and training

Sir—It is encouraging to see the place of research in the training of NHS physicians being questioned (October 1993, pages 403–404) but several points need scrutiny. First, various claims are made for the present system including training people to design and appraise research, and the teaching of presentational skills. But how effective are present schedules in achieving these aims? I suspect that current methods are quite poor and based on fallacious beliefs that ideas will 'rub off' on participants, but would welcome rather more factual information.

Second, how efficient is the present system? Could a formal short course of maybe 2–3 months, specifically designed to teach certain points, achieve the stated goals as well or better? This should be formally tested before making *ex cathedra* statements on research training.

My third point is more a matter of opinion but no less important. How much time is it desirable and sensible to set aside for topics related to research in an already crowded training programme which is under further pressure to be shortened? I imagine most people would support the concept of continuing to teach a scientific approach to medicine, and this is clearly bound up with an appreciation of research methods and results. However, many would point to other aspects of training that are possibly inadequately covered at present, such as better structured approaches to improving communication skills with patients and better training in audit procedures.

It will prove easier to sustain a case for training in research if this can be shown to be carried out effectively and efficiently and in a shorter time span than at present.

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Decompression sickness on mountains

Sir—James in his excellent article about decompression sickness (October 1993, pages 367–74) intriguingly suggests that mountain sickness may be caused by gas formation [1]. This suggestion is not new [2] but there is little evidence to implicate gas bubble formation in benign acute mountain sickness or high altitude cerebral or pulmonary oedema during mountaineering excursions to altitude. We believe that the rapid decompression experienced by divers and aviators is unlike the slow decompression of mountaineers.

In favour of James' speculation, Gray (1983) wondered if rapid ascent might cause microembolisation of the lung with air bubbles leading to high altitude pulmonary oedema [2]. If this were true, a higher incidence of high altitude pulmonary oedema would be expected during rapid simulated ascent in hypobaric chamber studies, but this does not occur [3].

Aviators decompress rapidly, a situation analogous to diving but James cites the occurrence of cerebrospinal fluid bubbles in aviators at 10,000 feet as evidence to support his suggestion of decompression sickness in mountaineers without mentioning rate of ascent. Conkin and Van Liew (1992) have found that by extrapolating to hypobaria the straight line which describes the lowest pressure to which a diver can ascend without developing decompression sickness after becoming equilibrated at some higher pressure, there was an excess of symptoms of decompression sickness in hypobaric chamber studies when compared with the expected from the extrapolated line in hyperbaric studies [4]. These findings demonstrate the risks of rapid decompression for aviators (or subjects in an hypobaric chamber) but do not parallel decompression schedules for climbers.

Acute mountain sickness is a common syndrome of

headache, nausea, anorexia, dizziness, dyspnoea and insomnia. The common symptoms of type I decompression sickness (joint pain and skin rashes) and type II decompression sickness (spinal cord symptoms) are not associated with acute mountain sickness or high altitude cerebral oedema, suggesting a different mechanism.

It is recommended that divers do not ascend faster than 60 feet per minute if they are to avoid decompression sickness (National Hyperbaric Centre, personal communication). To exceed this ascent rate would be a remarkable achievement for a mountaineer and it thus seems more likely that it is the lack of environmental oxygen at altitude which causes mountain sickness rather than decompression.

References

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