patibility. The temporal relation in the fall in PEF and arterial oxygen tension during haemodialysis, confirmed by Dr Wu and his colleagues, supports our initial contention that changes in PEF may be due to the activation of inflammatory mediators consequent on the activation of complement, neutrophils, monocytes, and platelets after the blood-dialyser interaction, resulting in an increase in pulmonary arteriolar tone and ventilation-perfusion mismatch and a reduction in tissue oxygen delivery.1 This is supported by data obtained during the use of cuprophan dialysers, when the expected fall in PEF2 and arterial oxygen tension and increase in platelet activation were much less than when the dialysers was used the first time.3

AJ WILLIAMS
A DAVENPORT
Department of Renal Medicine,
St James’s University Hospital,
Leeds LS9 7TF

We were interested to read the article by Dr AR Webb and others (August 1989;44:674-5) showing patients’ preference for lignocaine gel over lignocaine aerosol for topical nasal anaesthesia preceding fibroptic bronchoscopy. Seven years ago we reported the same preference for lignocaine gel over patients’ and normal subjects’ nasal anaesthesia.4 Nasal anaesthesia was equally effective with these two different methods, but the use of the aerosol was often associated with considerable nasal discomfort, an unpleasant taste, and epiphora, which did not occur with the gel. The additional advantage of the lubricating effect of the gel in passing the bronchoscope noted by Dr Webb and colleagues was also reported in our study. Furthermore, in our study plasma lignocaine concentrations were lower after the same dose of lignocaine gel by comparison with the aerosol, suggesting that the gel might also be safer in terms of lignocaine toxicity.

Topical nasal anaesthesia for fibroptic bronchoscopy

We would strongly support the conclusion of Dr AR Webb and others (August 1989;44:674-5) that lignocaine gel is preferable to lignocaine spray as a topical nasal anaesthetic for fibroptic bronchoscopy. We suspect that any physician who has applied both agents to his own nostrils would agree with this suggestion as the spray preparation tends to cause an unpleasant stinging sensation when it comes in contact with the nasal mucosa. We have used lignocaine gel for many hundreds of bronchosopic procedures with few complaints of discomfort from patients.

The technique which the authors used to apply the gel to the nose does, however, seem somewhat laborious. Although the revised technique described in the discussion section of the paper is more convenient than that used in the trial, we can recommend an alternative technique for gel application which we have found to be both convenient and effective. We use a 12.5 cm hollow plastic applicator (Everett, Kwill) to draw up 10 ml of lignocaine gel from its tube into a syringe. The same applicator is then used to inject gel into each nostril. The 4 mm diameter applicator can easily be advanced to any desired depth within the nasal cavity, whereas the conical applicator on the tube of lignocaine will barely enter the anterior nares. If we ask the patient to sniff, while occluding the opposite nostril, as the gel is applied, and we find that some of the gel is drawn into the pharynx, where it seems to provide useful preliminary topical anaesthesia before the introduction of the bronchoscope.

JOHN EFTHIIMIOU
Oster Chest Unit,
Churchill Hospital,
Oxford OX3 7LJ

We agree with Drs O’Driscoll and Webb that users of lignocaine gel for topical nasal anaesthesia may develop their own techniques for applying the gel. Indeed, some bronchoscopists in our unit use a syringe based method similar to the one they describe. The technique documented in both the “Methods” and the “Discussion” sections; it is the detail which is different in the two sections. We can assure readers that it is no more laborious to inject the gel from tube to nostril and massage it posteriorly than it is to open a syringe and Everett Kwill, draw the gel from the tube to the syringe, and then inject. It is also a little cheaper and, as our data show, provides effective topical anaesthesia.

We are grateful to Drs Eftimiou and Higenbottam for bringing their paper to our attention. The peak plasma lignocaine concentration in the nine patients given gel was reported to be not significantly less than the concentration in the 32 patients given lignocaine spray, though a lower peak plasma concentration was noted in volunteers given lower dosages of spray and gel. Thus lignocaine gel is at least as safe as lignocaine spray. These authors suggested a preference for lignocaine gel over patients for the gel in the discussion of their paper, and we have now measured the preference with a randomised study focusing on acceptability to patients.


The disturbance of respiratory mechanics in infants with bronchiolitis

I have read the report by Dr S Seidenberg and others (August 1989;44:660-7) on lung function in infants with bronchiolitis with considerable interest given our own studies in this field.1 Whereas their results relating to forced and passive expiratory flow are certainly in line with what we expect in this obstructive lung disease, it appears that they, like us, are in fact finding surprisingly low values for thoracic gas volume (TGV). It is true that in the acute phase their TGV was 130% of predicted and in the chronic phase 126%, but the scatter was wide (see their SEM values) and several infants must have had values in or below their normal range. In our study in the chronic phase we noted many infants with TGV values below our normal range,2 which is somewhat higher than the normal range used by Dr Seidenberg and his colleagues.

Given the differences in normal range I suspect that the two studies contain an appreciable number of bronchiolitic infants with surprisingly low TGV values. They do not really come to grips with the thoracic problem of whether or not TGV measurements are reliable in bronchiolitis. How, for example, do they know that all their values (both the high and the low) in the acute and chronic phases are not underestimated? I was delighted to see their results, which seem to confirm our own anxieties and suggest that our results were not simply an artefact. I should most interested in their further thoughts on this issue.


Adverse effect of additional weight on exercise capacity in patients with chronic obstructive airways disease

The conclusions of Dr C R Swinburn and others (September 1989;44:716-20) can be derived from common sense and an elementary knowledge of physics.

Acceleration or deceleration of a mass requires a force. If the mass is increased, a greater force is needed for the same acceleration. Alternatively, if the force is unchanged, less acceleration is produced (force = mass x acceleration), so the man the force is produced by muscle contraction, which is proportional to the energy stored in the force produced. When one walks at a steady pace, the legs alternately accelerate and decelerate but the body does not. Therefore the wearing of lead aprons will not substantially increase energy requirements, unless they are worn on the legs, not the thorax. Clearly, in step testing the whole body accelerates and decelerates in a vertical plane against gravity. The removal of the wearing of lead aprons will make a difference to energy expenditure and hence oxygen consumption during this form of exercise.