diets low in fat and the Eskimos have diets very high, but in each case the ratio of essential fatty acids to saturated fatty acids is high. On an Eskimo diet (marine food and water only for 100 days) my plasma fibrinogen concentration started and remained at a normal level.

The Japanese and Eskimos also have very low prevalences of bronchial carcinoma despite their heavy smoking; in Eskimos these were documented by Stefansson.[6] In an analysis of 43 countries Wynder *et al* showed a possible relation between high dietary fat, smoking, and lung cancer.[7] But Eskimos on their traditional diet have the highest dietary fat intake in the world and the Japanese among the lowest, which with other considerations led me to suggest more than 30 years ago that there was a relation between a relative deficiency of essential fatty acids (which is a low ratio of essential fatty acids to long chain saturated fatty acids and *trans* isomers), smoking, and lung cancer[8]; this ratio is about 0·2-0·3 in British diets but 1·0 or more in Japanese and traditional Eskimo diets. Keys, at a time when he thought that the quantity and not quality of fat was the important factor in coronary heart disease, stated that the American diet was higher in fat than that of Eskimos[9] and quoted me in support, but my paper on Eskimo diets showed just the opposite[10]; the Eskimo diet is, of course, rich in the long chain *n*-3 essential fatty acids of marine oils,[11] as is the Japanese diet.[12]

So we have a plausible explanation of the low prevalence of coronary heart disease and lung cancer despite heavy smoking in those who have a high ratio of these essential fatty acids to long chain saturated fatty acids and *trans* isomers, whatever the actual amount of fat in the diet.

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Botulinum toxin treatment of spasmodic torticollis

Dr A P Moore rightly emphasises the effectiveness of botulinum toxin in treating spasmodic torticollis (16 July, p 201). Our experience confirms its beneficial effect in relieving pain in more than 90% of patients and in improving head posture in 70%.[1] Unwanted effects, present in 30%, were confined to mild to moderate dysphagia for solids only, deepening of the voice, and occasionally localised pain at the injection site, all of which resolved within two to three weeks. In the hope that others will develop this treatment we describe here the technique we use.

Using the Porton Down toxin, we have found that a total of 1000 mouse units (MU), equivalent to 25 ng of toxin, is the optimum dose for most patients; it minimises side effects without the loss of efficacy. One ampoule of toxin, containing 2000 MU of freeze dried toxin, is diluted into 10 ml of normal saline, and 5 ml of this solution is then used: 2·5 ml is injected into each of the two most active muscles, selected clinically or by electromyography. For pure rotational torticollis the muscles injected are the splenius capitus ipsilateral to the shoulder to which the chin points and the contralateral sternomastoid. For retrocollis both splenius capitus muscles are injected, and for laterocollis we usually inject the ipsilateral splenius capitus and trapezius. It is not necessary to locate the motor point as this does not significantly improve the response to treatment. The sternomastoid muscle is injected in its anterior border at the junction between the upper one third and lower two thirds to a depth of 15 mm. This keeps the toxin well away from the pharyngeal muscles, which lie deep to its lower third, and reduces the incidence and severity of dysphagia. Splenius capitus lies on the floor of a triangle bordered anteriorly by the posterior fibres of the sternomastoid and posteriorly by the lateral fibres of the trapezius muscle. The injection is made at right angles to the neck to a depth of 12-15 mm, and the operator must be careful to draw back the syringe before injecting, as a venous plexus lies deep to this muscle.

Patients with more complex forms of torticollis are harder to treat and require careful study with surface and needle electromyography as several muscles may be implicated. In this condition it is best simply to inject two muscles at a time and have the patient return every three weeks for injections of other muscles if required.

Improvement in head posture and control and in pain usually appears within a week after injection, reaching a maximum over 24 hours. Benefit lasts about 9 to 15 weeks. Reinjection, exactly as before, is required whenever pain or the original pulling sensation returns. So far we have repeated injections up to five times over periods of up to 18 months, with no loss of efficacy.

With this technique we have successfully treated over 60 patients, without serious side effects or systemic toxicity. If patients with disabling spasmodic torticollis do not remit spontaneously and fail to respond to treatment with an anticholinergic drug such as benzhexol toxin injections are the treatment of choice. Since spasmodic torticollis is a lifelong disability in most patients, however, careful monitoring of the long term use of this new treatment is necessary. Theoretical problems that might emerge include loss of efficacy due to the development of antibodies to botulinum toxin, although this has not happened yet.

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Continuous arteriovenous haemodialysis

Drs E A Brown and W Kox provided views on a recent addition to the range of techniques available for the management of uraemia and advocate its use in any intensive care unit without the need for nephrological skills (23 July, p 242). Knowing the range of treatments available for use in uraemia is, however, essential in providing appropriate care. There is general agreement that the convection techniques, intermittent haemofiltration, continuous arteriovenous haemofiltration, and continuous arteriovenous haemodialysis are associated with improved cardiovascular stability in some patients with failure of several organs. There are, however, reservations.

Continuous arteriovenous haemofiltration and haemodialysis may not be effective in all patients in a hypercatabolic state. In those with haemorrhagic complications or requiring emergency surgical procedures continuous anticoagulation can be problematic unless the staff are familiar with the use of epoprostenol infusions. The additional work required of intensive care nurses in monitoring continuous arteriovenous haemodialysis should not be underestimated, and errors in balancing fluids can occur. The skill needed to deal with complications arising in patients being treated with extracorporeal circulatory devices cannot be acquired by occasionally using such devices in an intensive care unit.

Skill in creating arteriovenous access also requires frequent practice. Vessels that may have to serve for up to three months in a patient with failure of several organs can be damaged by those who are inexperienced. Femoral cannulation and the continuous immobilisation that follows are also hazardous and can pose problems in movement for diagnostic or other procedures.

There is no evidence that continuous arteriovenous haemodialysis can offer advantages over current intermittent haemodialysis or haemofiltration techniques in acute renal failure caused by intrinsic renal disease or when the kidney is the sole organ failing after obstetric, nephrotoxic, or transient ischaemic insults.

Stevens *et al* quoted an overall survival in their patients of 31%.[1] This result is similar to the experience of most renal units over the past two decades, highlighting the fact that we have probably achieved the best techniques for managing uraemia. Further improvement in survival in those with failure of several organs will depend on advances in managing cardiovascular support and the acute respiratory distress syndrome. Perception of these improvements, however, will be difficult unless we categorise and compare patients more specifically in terms of precipitating causes and what systems are failing.

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1 Stevens PE, Davies SP, Brown EA, Riley B, Gower PE, Kox W. Continuous arteriovenous haemodialysis in critically ill patients. *Lancet* 1988;ii:150-2.

The editorial by Drs E A Brown and W Kox (23 July, p 242) on continuous arteriovenous haemodialysis was important for all doctors concerned with intensive care. The mortality in ventilated patients with acute renal failure is still distressingly high at 75-100%.

In 1987 we published our observations on 25 such patients treated by continuous haemodialysis and ultrafiltration.[1] We had already found serious limitations in the use of continuous haemofiltration alone in intensively fed and hypercatabolic patients. The clearances achieved were inadequate, the patients continued to have high concentrations of urea, and intermittent haemodialysis was still needed. Our technique of continuous arteriovenous ultrafiltration and haemodialysis allows smooth and gentle control of fluid and electrolyte concentrations. There are three points that we wish to add to your editorial, based on our continued experience with this technique.

Firstly, the authors failed to consider the most important question in the use of continuous dialysis: Does maintenance of a stable, normal internal environment improve mortality in patients