Considering advances in technology

Promising new solutions, such as haemoglobin based oxygen carriers, may help to resolve the nihilistic dilemma now faced by many clinicians.11 Perhaps by providing limited slow infusions of a solution that can be stored without special refrigeration but has augmented capacity to carry oxygen might safely provide earlier treatment to patients with polytrauma.

Recent technological developments may also better delineate patients with true hypoperfusion. In contrast to traditional crude parameters such as blood pressure, new monitoring devices (for example, sublingual CO₂ monitoring) may help better to titrate the rapeutic interventions and their timing. $^{\!\scriptscriptstyle 12}$ We may be able to obviate some of the current controversies revolving around the management of shock in polytrauma by better determining a situation in which the relative benefits of delaying treatment is outweighed by a more precise titration and better timed infusion of an oxygen carrying solution.2 We may also define shock more precisely. The all too common assumption that injured people with hypotension are in shock warrants re-evaluation.

For now it is still the experience and judgment of the discerning knowledgeable clinician that best guides the treatment of the polytrauma patient. Victims of polytrauma will be benefited if that clinician pays attention to the differences in various mechanisms of injury, their anatomical involvement, and the staging of those processes and also recognises that, in some circumstances, less treatment may be better.² ⁴ ⁶ ¹⁰

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Intensive education for lifestyle change in diabetes

Ongoing input is required to effect and maintain change in behaviour

'n the past 10 years the diabetes control and complications trial and the UK prospective diabetes study (UKPDS) have shown that tight control of diabetes reduces the risk of complications in type 1 and type 2 diabetes.^{1 2} As a result of these studies we have set our patients demanding targets, which often require important changes in their lifestyle. But we have failed to provide the education and self management training needed to help them meet these targets. In this context, intensive modifications to lifestyle means structured education designed to facilitate change in behaviour. Such education programmes are used in type 1 and type 2 diabetes and in prevention of diabetes in people with impaired glucose tolerance.

Traditional education for diabetes treats the patient as a receptacle for knowledge or a pot to be filled with information by doctors, nurses, and dieticians. To achieve change in behaviour education must encourage self motivation and self determination,3 and a professional who simply tells patients to make a change "for their own good" invites a negative response.

Helping people to change their lifestyle is never easy and can be done only by approaching the problem from the patients' point of view.4 In type 1

diabetes this approach was developed and refined in Germany by Ingrid Mühlhauser and the late Michael Berger.⁵ Centres in other countries have adapted the German programme, which has recently been transplanted to the United Kingdom as the dose adjustment for normal eating (DAFNE) project. A randomised controlled trial including three centres showed that this programme leads to improvements in glycosylated haemoglobin A_{1c} test, dietary freedom, and quality of life. DAFNE has been successfully rolled out to other centres in the United Kingdom, but the cost of the programme has led other units to modify it. These programmes with reduced professional input are cheaper but require evaluation.

The epidemic of type 2 diabetes, projected to reach 333 million cases worldwide by 2025, is causing alarm in both medical and political circles. Since increasing obesity and decreasing physical activity are responsible, modifications of lifestyle, focusing on diet and exercise, is the logical way of stemming the tide.

Several studies have shown that programmes designed to bring about lifestyle changes can slow the progression of impaired glucose tolerance to diabetes. The United States diabetes prevention programme randomised 3234 subjects with impaired glucose toler-

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ance to placebo, metformin, or an intensive programme of diet and exercise.7 New cases of diabetes were reduced by 58% in the diet and exercise group compared with 31% in patients randomised to metformin. The lifestyle modification group received intensive education and support with care managers delivering a personal 16 lesson curriculum and subsequent monthly follow up sessions to reinforce behavioural change. Subjects were advised to make a 7% reduction in body weight by a low fat, low calorie diet and to take moderate physical activity such as brisk walking for 150 minutes per week.

Twenty four weeks into the study, half the subjects achieved the weight reduction target, but despite continuing support only 38% maintained this over the three year study period. Prevention studies using trained educators to deliver intensive education achieved equally encouraging results in Finland and China.89 However, a pilot study in Oxford that employed less intensive dietary and exercise advice did not achieve a fall in either body weight or blood glucose and showed that lifestyle modifications were not sustained once educational input had been withdrawn.10 The message is clear-the onset of diabetes can be delayed by lifestyle modification, but intensive ongoing input is required to effect and maintain the change.

When type 2 diabetes is diagnosed, patients are often distressed, anxious, and confused about the implications of this disease. In an ideal world they would receive information and emotional support at diagnosis followed by a structured education and self management programme. The effectiveness of such group education programmes is widely acknowledged,11 but availability is patchy, and there is a need to ensure that high quality diabetes education is universally available, irrespective of social status. Some countries are addressing this need. For example, the US government has supported a national diabetes education programme, with the aims of increasing awareness of diabetes and ensuring that diabetes education programmes are validated and delivered by accredited educators, who now number more than 11 000. In spite of this, some deprived communities with a high prevalence of diabetes may be unable to access these programmes.

The United Kingdom lags far behind, although the national service framework for diabetes has recognised that the provision of information, education, and psychological support that facilitates self management is the cornerstone of diabetes care and has set primary care groups the target of providing empowering education by March 2006.12 Only a handful of UK centres have established intensive education programmes, and these are now part of a national group that is working to develop a coordinated system of educational care. Considerable energy and resources are required to set up and maintain educational programmes, but the cost per individual is small compared with that of treating the consequences of uncontrolled diabetes.

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Genomic imprinting as a cause of disease

Is increasingly recognised, especially after assisted reproduction

enomic imprinting, defined as gene expression dependent on the parent of origin, has been increasingly recognised over the past decade as a mechanism contributing to human disease. The topic now features as a core part of any genetics curriculum, appears in postgraduate medical examinations, and is a term familiar to many clinicians. Recently abnormalities of genomic imprinting have been discussed in the context of assisted reproductive technologies. So what exactly is genomic imprinting, and how does it occur?

For many years a gene was assumed to have the same function, whether it was inherited from the mother or the father. We now know this is not the case, as the DNA of some genes is modified during gametogenesis and as a result may have altered expression, becoming either inactivated or activated. Genes that are susceptible to parent specific modification in this way (termed epigenetic, because the modification does not entail mutation of the DNA code) are referred to as imprinted genes.2 The mechanism of imprinting is still not entirely clear but in most cases

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