tion and management is also being addressed. Firstly, despite increased interest in asthma education only a small proportion of the asthmatic population actually receive it.[6] Secondly, asthma management (compared with published guidelines[7,8]) is worse in socially disadvantaged regions. A community wide intervention in south western Sydney, which includes many socially disadvantaged neighbourhoods, focused on equity in asthma care. This programme consisted of extensive general practitioner education, collaboration with the New South Wales education department, and the delivery of asthma education and optimal clinical care to children with asthma in the community.[8] This intervention has resulted in increased use of peak flow meters and preventive treatment and increased ownership of asthma action plans across the region.

Although there are several well publicised guidelines for care of asthma,[7,9,10] we concur with Dr Burney that our ignorance about asthma has led to an absence of specific quantifiable targets. This remains a problem in the formulation and evaluation of public health interventions. For example, controversy remains about the regular use of β_2 agonist bronchodilators and about the use of theophylline in children. Use of peak flow meters and action plans is strongly recommended, but no population targets are set for their attainment. Thus, although 40% of children with asthma in south western Sydney now have a peak flow meter, we do not know whether this is optimal adoption of the practice in a population.

In the next round of setting health goals and targets, in both Australia and Britain, the collaborative efforts of respiratory and public health researchers are needed to define the knowledge on which to build a sound approach to management of asthma and, if possible, its prevention.

ADRIAN BAUMAN
STEPHEN R LEEDER

Departments of Public Health and Community Medicine,
University of Sydney,
New South Wales,
2006 Australia

LIS YOUNG

Southern Sector (Community Health),
South West Sydney Area Health Service,
Sydney,
Australia

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The "Q" in QALYs

SIR,—In their letter[1] C Gudex and P Kind comment on our article on determining quality adjusted life years.[2] We would like to address three points that they made.

Firstly, we did not and do not "acknowledge the need for a single index measure"; we simply said that "to determine the value of q [in QALY calculations] a method is needed that provides a single value."

Secondly, Gudex and Kind query our focus on the York health measurement questionnaire. The reason is simple: it has been cited as a "standard quality of life questionnaire"[3] without evidence of extensive piloting or of its reliability and validity. Furthermore, the pilot study they refer to was interviewer led, which does not provide evidence of reliability or validity when the questionnaire is used as a self report instrument without such guidance.

Thirdly, Gudex and Kind state, "there have been many new developments and improvements," yet the version of the York health measurement questionnaire used most recently is the original version.[3,4]

We repeat the conclusion of our original article: the use of untested and unvalidated instruments in clinical trials and cost utility analyses must be strongly discouraged. This is a judgment not on the use of QALYs but on how the value of q might be derived.

ROY CARR-HILL
J MORRIS

Centre for Health Economics,
University of York,
York YO1 5DD

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Off the rails

SIR,—In his editorial J Havard lays particular emphasis on the fact that several railway accidents have occurred recently in which it has been proved or suspected that the ability of the drivers concerned was impaired by alcohol or other drugs.[1] At the inquests it was stated that automatic warning signals may easily be bypassed, and a further factual report stated that the driver passed a red signal, going far too fast to stop.

I question the continuing use of red as a danger signal. It has been estimated that one in 12 men, though only one in 200 women, has an inherited failure to distinguish between the colours red and green. Further, as one gets older colour awareness tends to change in previously healthy individuals. I have little doubt that many of your readers find, as I do, increasing difficulty in following a red cricket ball against a green outfield and yet have no difficulty in following the flight of a yellow tennis ball on a grass court. This red-green colour defect also occurs in amblyopia due to tobacco, alcohol, and other drugs, as well as in toxic, metabolic, or inflammatory conditions affecting the optic nerves.

For well over 30 years I have been concerned with the neuro-ophthalmological manifestations of chronic cyanide intoxication, of which tobacco amblyopia and retrobulbar neuritis in pernicious anaemia and other vitamin B-12 deficiency states are but examples.[2] The first symptom is an inability to distinguish between the colours red and green before the vision to white is materially reduced. This failure of colour discrimination may, however, be so gradual that it is unnoticed by the patient. One of our first patients with tobacco amblyopia, whom we treated with intramuscular hydroxocobalamin, was a market gardener who became somewhat disconcerted by the apparent failure of his apples to ripen. It was only after it was pointed out to him that the apples in his orchard were red and not green did he realize that his appreciation of colour was at fault.

The medicolegal aspect of this colour disturbance is important in that if a road user or engine driver suffers from an acquired or inherited failure of colour discrimination, it may not be possible for him to distinguish between green and red traffic lights or railway signals. This loss of colour discrimination was successfully used by defence counsel in a case involving an engine driver who was a heavy pipe smoker and caused an accident, having ignored the red danger signal.[3]

Is it not high time in the interest of safety on the roads and railways that there should be a critical reappraisal of coloured light signals in current use for traffic control?

ANTHONY G FREEMAN

Swindon SN3 1QE

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Camelford revisited

SIR,—In his editorial on the discharge of aluminium sulphate into a reservoir near Camelford in Cornwall David Coggon makes an analogy between the advisory group set up after the pollution incident and a clinician faced with an anxious patient whose symptoms seem after routine investigation to signify nothing sinister.[1] This suggests that the clinician has taken the trouble to perform investigations. In the case of Camelford this has not happened.

Rather, in its first report the advisory group, chaired by Dame Barbara Clayton, specifically declined to investigate further what was at that time the main objective physical finding, a deposition of aluminium in samples of bone in affected people.[2] The investigations were carried out unfunded and outside official scientific and health care channels. It is disingenuous for government scientists to refuse to investigate on their own account and then to criticise the results of work that is performed because it "derives from self selected and highly unrepresentative samples." The position in Camelford is not that the official clinicians have investigated and found no evidence of disease. They have taken the view that the patients' symptoms originate in the psyche and are interpreting all incoming data in that light.

It is vital that the uncertainties in this case are resolved as soon as possible. As Coggon mentioned, both the Clayton reports have been perceived as a whitewash, and those perceptions reflect on to the credibility of all official statements on health matters.

The question of the organic effects of the cocktail at the Lowermoor reservoir could be settled by experimentation under controlled conditions. Volunteers could be asked to ingest water in the same quantity and of the same quality as that taken by the population of Camelford, and any symptoms, signs, and metabolic and serological changes could be closely monitored. Any corrupting anxiety factor could be removed by selecting for the experiment only those who, like Dame Clayton and Coggon, believe that long term toxicity is unlikely. I should add that I believe that the experiment would be so dangerous as to be unethical.

RICHARD LAWSON

Congresbury,
Avon BS19 5DX

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