discriminate according to age in providing care to people who are chronically ill. However, as long as age is viewed to some extent as a relevant factor the unit of measurement (the SAVE) needs to be specified in terms of age.

The specification of "a young life" would depend on how strongly society wishes to discriminate between young people. This is not for me to decide. The point is that the SAVE procedure is not inherently agist. By deciding equivalence numbers (or the inverse, priority weights) for different age groups in specific contexts, and by explicitly deciding to what degree the social value of an outcome increases with its duration, society can make the SAVE procedure precisely as agist or non-agist as it wishes.

The choice of "saving a young life" as a unit of measurement is based on consideration of medical benefit (quantity of well life produced) only. It has nothing to do with contribution to the economy. The SAVE approach consists in valuing outcomes—that is, movements from different levels of dysfunction to higher levels. These valuations should be elicited independently of diagnoses and procedures. They need to be combined with doctors' assessments of the effects of procedures to obtain the value of the procedures. The SAVE approach does not include scaling of personal preferences for health care. It is concerned with equivalence of numbers of patients.

I don't believe that any numerical measure of benefit can be free of limitations. Hence I suggest the SAVE procedure only as an aid to priority setting.

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1 Lewis PA, Charny M. Which of two individuals do you treat when only their ages are different and you can't treat both? *J Med Ethics* 1989;15:28-32.

Balloon dilatation of heart valves

EDITOR,—We agree with Roger Hall and Richard Kirk's conclusions regarding the poor overall results of balloon dilatation in aortic valve stenosis and share their preference for aortic valve replacement in most cases.¹ With regard to mitral valvuloplasty, however, the authors' statement that "no randomised comparisons of valvuloplasty and surgery have been made" is incorrect. At least three published studies have compared percutaneous balloon mitral valvuloplasty with surgery for mitral stenosis: two performed by our group working with fellow investigators in Hyderabad, India, and one from South Africa.²⁻⁴

Our two studies were randomised prospective trials, with the data analysed by investigators blinded to the treatment and the time of evaluation. The first study compared percutaneous balloon mitral valvuloplasty with closed mitral commissurotomy²; the second compared it with open mitral commissurotomy.³ Both studies included cardiac catheterisation before the study and one week, six months, and three years after the procedure as well as echocardiography and stress testing. The results to six months have been reported, and we found no significant differences in efficacy or safety between the treatment groups.

These trials enrolled patients with pliable valve leaflets, fairly modest calcification, and an absence of factors that would favour surgery, such as appreciable mitral regurgitation or severe subvalvular disease. They did, however, include some patients with severe pulmonary hypertension, who might benefit from avoiding thoracotomy. Thus, although we share Hall and Kirk's concerns that the advantages of balloon valvuloplasty are "mean-

ingless unless the safety and efficacy of dilatation compare favourably with those of conventional surgery," we believe that as linear follow up of these patients is published there will be ample data to support the use of balloon valvuloplasty for well selected patients with mitral valve stenosis.

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Cerebral oedema in diabetic ketoacidosis

EDITOR,—In their editorial on cerebral oedema in diabetic ketoacidosis¹ Peter Hammond and Simon Wallis refer to the sodium ion-hydrogen ion exchanger hypothesis.² They state that in models of diabetic ketoacidosis "cerebral pH is not greatly decreased, so weak organic acids probably do not enter brain cells to cause cytoplasmic acidification." This statement contains two errors.

Firstly, no studies that I am aware of have examined cerebral pH in diabetic ketoacidosis. If the authors are referring to the animal studies of Arieff and Kleeman,^{3,4} these have already been dismissed as more relevant to the non-ketotic hyperosmolar state and of questionable relevance to diabetic ketoacidosis with its prominent organic acidosis. Of greater concern is the authors' implication that weak organic acids would not enter "brain cells." Cellular acidification by weak organic acids does occur at clinically relevant pH values (pH 6.9-7.4). This is because the process depends not on a tissue specific membrane carrier mechanism but, rather, on simple diffusion of the protonated form of the weak organic acid across the lipid bilayer.^{5,6} Cultured glial cell lines undergo cytoplasmic acidification on exposure to weak organic acids and then swell in a manner totally consistent with activation of the sodium ion-hydrogen ion exchanger.⁷ Sodium ion-hydrogen ion exchanger activity is well documented in mammalian neuronal cells⁸ and can be expected to behave in a qualitatively similar manner.

Once basic properties of the sodium ion-hydrogen ion exchanger are appreciated it is difficult to see how the exchanger could be prevented from producing cerebral oedema in the metabolic setting of diabetic ketoacidosis. Indeed, evidence suggests that cerebral oedema may occur routinely in uncomplicated diabetic ketoacidosis.⁹ What is not explained is why only a minority of children develop the extreme, catastrophic form of cerebral oedema. One explanation would be variations in those details of treatment that could act through the sodium ion-hydrogen ion exchanger mechanism (for example, rapid correction of extracellular pH or excessive delivery of insulin or sodium). It is equally plausible, however, that the basis of the observed clinical variation will be found in a functional polymorphism of either the exchanger itself or factors controlling its activity.

The sodium ion-hydrogen ion exchanger model for cerebral oedema in diabetic ketoacidosis remains remarkably consistent with the available evidence. It should be of interest to those wishing

to explore this clinical condition through use of an animal model.

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Code of practice for over the counter products

EDITOR,—A new acaricide—Sergeant's House Dust Mite Patrol—that is claimed to reduce levels of house dust mites has recently been launched. The manufacturers provide data on its ability to reduce house dust mite levels but no data to show that it helps patients with asthma. Correspondence from the manufacturers indicates that they are able to provide reasonable data on toxicity to humans but have no data on clinical efficacy. One of their promotional posters contains this misleading statement: "As an allergy sufferer you can benefit from a reduced frequency and severity of attacks by establishing a preventative programme to significantly reduce the effects of House Dust Mite population in your home."

I am concerned that the manufacturers have done no clinical trials to show that this particular formulation has any clinical benefit. Many previous acaricides have failed to result in clinical benefit despite reducing house dust mite levels (and therefore airborne allergen levels) substantially.

It seems to me that a code of practice needs to be applied to such substances, particularly as they are sold without prescription. I am aware that this substance is not ingested by patients, but it seems inappropriate for any product to be launched so aggressively without any evidence of clinical benefit. Pharmaceutical companies have to go to enormous lengths to produce data on efficacy before their products are released on to the market; when an over the counter preparation claims to reduce asthma some assessment of the regulation of such products is surely needed.

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*We sent this letter to the manufacturers, who replied as follows.

EDITOR,—It is difficult to understand why Jon G Ayres refers to Sergeant's Dust Mite Patrol specifically and not to any other product. If his argument is that acaricides designed for use against clinically important mites should be restricted in availability then he should lobby the Medicines Control Agency over its licensing of the product. On the other hand, he acknowledges that the

product is effective against house dust mite. It also conforms with all the criteria required by a recent position paper of which he is a coauthor¹: because it is a heavy powder, thorough vacuuming (part of integrated allergen avoidance) is necessary unlike with other acaricidal products except for liquid nitrogen. In addition, Sergeant's Dust Mite Patrol has the least residual action and safest toxicity profile of all acaricides.

Of all the clinical trials quoted by Colloff *et al*, only six resulted in significant clinical improvement in patients, and three of those used some form of acaricide.²⁻⁴ As Sergeant's Dust Mite Patrol kills mites effectively and the necessary vacuuming will remove allergen by removing the dead bodies and mite faeces, the product must offer the possibility of relief of symptoms for people sensitive to dust mites when used as part of integrated allergen avoidance, which we have always advocated.

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Monitoring digoxin treatment

EDITOR,—J K Aronson and M Hardman's article on monitoring treatment with digoxin¹ may cause delay in stopping treatment in cases in which the plasma digoxin concentration is not available rapidly. Newly qualified house physicians tend to send off for measurement of plasma concentrations and wait for the results before stopping or decreasing the dose of digoxin.

If digoxin toxicity is suspected clinically it is much safer to stop the drug at once whether a blood test is done or not. By the next day the patient's nausea, anorexia, or vague ill health is generally much better if digoxin is the culprit. Measurement of the digoxin concentration is often not needed under these circumstances, although it is helpful in more complicated cases, as the article shows.

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Breast feeding and HIV infection

EDITOR,—In the past few years in Nairobi no single issue related to HIV has caused more debate than whether HIV can be transmitted by breast feeding. Should the possibility be tested scientifically in Kenya? Is it valid to carry out research in communities that are unlikely, as William A M Cutting points out,¹ to be able to respond by changing feeding practices if transmission by breast milk is shown? The operational implications seem of clear benefit only to the affluent communities of the industrialised world, where bottle

feeding can easily be instigated. Or is it more important to find out what the level of risk is and then decide how to respond to the problem with hard data?

There are no easy answers to these complex issues, which are common to much of the clinical research carried out in the developing world; it is just that the issue of breast feeding and transmission of HIV is highly emotive and highlights the dilemmas clearly.

Perhaps we are being too academic about the problem. Even if a high risk of transmission is shown and Kenya then decides to recommend and implement a policy of formula feeding for HIV infected mothers, who will actually take it up? It will be almost impossible for most mothers to conceal the fact that they are bottle, not breast, feeding—which will be tantamount to advertising their HIV status. I do not think that many women will be prepared to do this. As Cutting says, poor women will continue to feed their babies in the most convenient, economic, and familiar way. More importantly, they will continue to breast feed because this maintains the confidentiality and privacy of their own diagnosis.

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Role of breast feeding in paralytic poliomyelitis

EDITOR,—Much evidence shows that breast feeding protects infants from infection, but no data are available about its protective effect against poliomyelitis.^{1,2} Human milk can neutralise poliovirus, and limitation of intestinal shedding of vaccine poliovirus by breast feeding has been shown under certain conditions.^{3,4} We investigated the association between breast feeding and poliomyelitis during the last epidemic in Campania region, Italy, which occurred in 1958, by a population based case-control study.

In 1958 there were 114 000 live births, and 942 infants with paralytic poliomyelitis were reported⁵: of these, 634 were admitted to the Regional Hospital for Infectious Diseases Domenico Cotugno. The incidence was 8.3/1000 and the case-fatality rate 15%. We studied all 103 infants aged 0 to 6 months admitted with a diagnosis of paralytic poliomyelitis between 1 January and 31 December 1958. Ten patients, for whom information on feeding was not available, were excluded. One control for each case was selected by systematic sampling of infants admitted to the same hospital with an acute illness during the same period. The controls were matched to the cases by age (within 15 days) and gender; their main diagnoses were seizures, anaemia, and fever of unknown origin. Information on feeding was not available for 14 patients, who were replaced by the next patients in the list for whom information was available from the records.

Breast feeding was classified as a dichotomous variable: never breast fed and at least partially breast fed. Data on background variables and feeding were collected from the clinical charts by a person blinded to the objectives of the study. Information on breast feeding at admission and parental smoking was not available from the records. Confidence intervals for odds ratios were calculated with Cornfield's method.⁶ The number of infants who had ever been breast fed was 75 among the cases and 88 among the controls ($p < 0.01$). Compared with the infants who had received some human milk, those who had never been breast fed had a higher risk of paralytic

poliomyelitis (odds ratio 4.2, 95% confidence interval 1.4 to 14). When we stratified by social class and the number of other children at home the odds ratio did not show any relevant modification.

Because of lack of information in the records our study has some limitations: breast feeding was available only as a dichotomous variable, and the method of feeding at the time of admission was unknown. This may have decreased the strength of the observed association. Moreover, because the data were obtained 34 years ago we could not check their quality (for example, by interviewing the parents). On the other hand, the epidemic of 1958 was the last that occurred in Italy and our study probably has the interest and the limits of a historical study; epidemiology was a young discipline then, and no other studies of dietary risk factors for poliomyelitis have been published.⁷ Our data suggest a protective role of breast feeding against paralytic poliomyelitis during the first six months of life.

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Nedocromil sodium may substitute for steroids in asthma

EDITOR,—J R Hughes and colleagues report a case in which a moderate dosage of inhaled beclomethasone—presumably 500 µg twice daily, not 500 mg as published—caused severe acne in a man aged 77.¹ They were unable to control the asthma without steroid treatment but fail to say whether other inhaled anti-inflammatory treatment had been used.

In a recent study I and colleagues looked at the steroid sparing effect of nedocromil sodium in moderate to severe asthma and found that it was effective in replacing a mean of 330 µg of inhaled steroids yet allowing optimal control of asthma.² That figure hides the fact that 65% of those patients taking high doses (> 600 µg, mean 1100 µg) were able to halve their dosage and 16% to maintain optimal control of asthma on a quarter of the original dosage. High doses of inhaled steroids are extremely valuable in controlling chronic asthma and may have contributed to the fall in deaths from asthma in New Zealand,³ but systemic side effects may occur; if they do, nedocromil sodium may well be useful.

I have recently seen three patients (two men and one woman) who developed skin thinning and purpura while taking 2000 µg of inhaled steroids. The woman developed bilateral rupture of the Achilles tendon, which was then complicated by pulmonary embolus. These patients were given nedocromil sodium, which substituted completely for the steroids with resolution of the skin changes and continuing optimal control of the asthma.