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 commissurotomy[2]; 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 "meaningless 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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1 Hall R, Kirk R. Balloon dilatation of heart valves. *BMJ* 1992; **305**:487-8. (29 August.)
2 Turi ZG, Reyes VP, Raju BS, Raju AR, Kumar DN, Rajagopal P, *et al.* Percutaneous balloon versus surgical closed commissurotomy for mitral stenosis. A prospective randomized trial. *Circulation* 1991;**83**:1179-85.
3 Reyes VP, Raju BS, Turi ZG, Rajagopal P, Stephenson L, Mehta PM, *et al.* Percutaneous balloon vs open surgical commissurotomy for mitral stenosis: a randomized trial. *Circulation* 1990;**82**(suppl III):545.
4 Patel JJ, Shama D, Mitha AS, Blyth D, Hassen F, Le Roux BT, *et al.* Balloon valvuloplasty versus closed commissurotomy for pliable mitral stenosis: a prospective hemodynamic study. *J Am Coll Cardiol* 1991;**18**:1318-22.

## Cerebral oedema in diabetic ketoacidosis

EDITOR,—In their editorial on cerebral oedema in diabetic ketoacidosis[1] 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 cells[8] 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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1 Hammond P, Wallis S. Cerebral oedema in diabetic ketoacidosis. *BMJ* 1992;**305**:203-4. (25 July.)
2 VanderMeulen JA, Klip A, Grinstein S. Possible mechanism for cerebral oedema in diabetic ketoacidosis. *Lancet* 1987;ii:306-7.
3 Arieff AI, Kleeman CR. Studies on mechanisms of cerebral edema in diabetic comas. *J Clin Invest* 1973;**52**:571-83.
4 Arieff AI, Kleeman CR. Cerebral edema in diabetic comas. II. Effects of hyperosmolality, hyperglycemia and insulin in diabetic rabbits. *J Clin Endocrinol Metab* 1974;**38**:1057-67.
5 Roos A, Boron WF. Intracellular pH. *Physiol Rev* 1981;**61**: 296-434.
6 Grinstein S, Goetz JD, Furuya W, Rothstein A. Gelfand EW. Amiloride sensitive Na+H+ 'xchange in platelets and leukocytes: detection by electronic cell sorting. *Am J Physiol* 1984; **247**:C293-8.
7 Jakubovicz DE, Grinstein S, Klip A. Cell swelling following recovery from acidification in C6 glioma cells: an in vitro model of postischemic brain edema. *Brain Res* 1987;**435**:138-46.
8 Jean T, Frelin C, Vigne P, Barbry P, Lazdunski M. Biochemical properties of the Na+/H+ exchange system in rat brain synaptosomes. *J Biol Chem* 1985;**260**:9678-84.
9 Krane EJ, Rockoff MA, Wallman JK, Wolsdorf JI. Subclinical brain swelling in children during treatment of diabetic ketoacidosis. *N Engl J Med* 1985;**312**:1147-51.

## Code of practice for over the counter products

EDITOR,—A new acaricide—Sargeant'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