Observed average decrease in height from peak stature in women and men by age

Age (years)	Observed decrease (cm)	
	Women	Men
30	0.024	0.127
40	0.410	0.596
50	1.266	1.411
60	2.592	2.572
70	4.388	4.079
80	6.654	5.932

In any future screening for osteoporosis we intend to motivate people to attend by providing data on the rate of decrease in height of people in Busselton, particularly after the age of 40. During 1966-81 serial examinations at three year intervals were conducted on nearly the entire adult population of Busselton; the heights of the 1785 women and 1544 men were measured on three to six occasions. No subject was excluded for health reasons. Random regression analysis of each person's height and age was used to estimate the expected rate of decrease in stature with age.<sup>3</sup> Because the analysis was confined to longitudinal changes in individual people the secular trends were removed.

From the population data women's height was estimated to peak at  $162 \cdot 1 \text{ cm}$  at  $26 \cdot 8$  years; for men the average maximum height was calculated to be  $174 \cdot 9 \text{ cm}$  at  $21 \cdot 4$  years. The table shows the observed decrease in height from peak stature in women and men at ages  $30 \cdot 80$ . It was concluded that men of predominantly northern European background could expect to be about 6 cm shorter than their peak height by the age of 80, and women could expect to be  $6 \cdot 7 \text{ cm}$  shorter. Such information may be a motivation to attend screenings for osteoporosis.

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- I Garton MJ, Torgerson DJ, Donaldson C, Russell IT, Reid DM. Recruitment methods for screening programmes: trial of a new method within a regional osteoporosis study. BMJ 1992;305:82-4. (11 July.)
- 2 Curnow DH, Cullen KJ, McCall MG, Stenhouse NS, Welborn TA. Health and disease in a rural community: a Western Australian study. Australian Journal of Science 1979;31:281-5.
- 3 Chandler PJ, Bock RD. Age changes in adult stature: trend estimation from mixed longitudinal data. Ann Hum Biol 1991;18:433-40.

## Human insulin and unawareness of hypoglycaemia

EDITOR, — The three articles on loss of awareness of hypoglycaemia with human insulin in a recent issue give the general impression that there is not really a problem with human insulin.<sup>1-3</sup> Certainly all the research that has been done has not given a definitive answer, but it does suggest that there may be a problem in certain studies. Nobody, however, seems to have taken any account of what diabetic patients are saying.

I have had insulin dependent diabetes for 23 years and have taken human insulin for two periods of two and a half years each. I found human insulin awful, and my control has been much better since I resumed taking porcine insulin. I know of other diabetic patients who have felt the same, but nobody really listens or thinks that diabetic patients' opinions are of any great value. Doctors ignore this at their peril.

John E Gerich quotes the recent study by Colagiuri *et al* in Australia as being some form of benchmark in research into this problem.<sup>4</sup> This study has two serious flaws in its design. Firstly, being in a study makes diabetic patients control their disease better than they do in normal daily life. This and the fact that the patients were put on to human or porcine insulin for only one month at a time in the study, albeit under double blind conditions, mean that it is not surprising that the patients could not tell which insulin they were receiving. If they had received each type of insulin for six months or a year they might have been able to tell the difference.

This leads me to the second flaw in the study. Colagiuri *et al* say that the patients they took for the study had complained that they had lost their symptoms of hypoglycaemic awareness. If this is so it is common sense to assume that somebody else probably a family member or close work colleague —made the patients aware of this fact; but the questionnaires were completed only by the patients themselves with no input from the family members who would have been the first to notice whether the patients had lost their awareness.

I disagree vehemently with Gerich when he says that a study should be conducted along these lines. If it is it will be worthless as well.

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- Gerich JE. Unawareness of hypoglycaemia and human insulin. BMJ 1992;305:324-5. (8 August.)
  Egger M, Smith G, Teuscher A. Human insulin and hypogly-
- 2 Egger M, Smith G, Leuscher A. Human insulin and hypoglycaemic unawareness: on the need for a large randomised trial. BMJ 1992;305:351-5. (8 August.)
- Williams G, Patrick A. Human insulin and hypoglycaemia: burning issue or hot air? BMJ 1992;305:355-7. (8 August.)
  Colagiuri S, Miller J, Petocz P. Double-blind crossover
- comparing S, Miller J, Feloz F. Doube-onna crossover comparison of human and porcine insulin in patients reporting lack of hypoglycaemia awareness. *Lancet* 1992;339:1432-5.

## Fluid replacement in diabetic ketoacidosis

EDITOR,-If, as Peter Hammond and Simon Wallis suggest, emphasis is to be given to preventing the development of cerebral oedema in diabetic ketoacidosis1 we need a careful analysis of the optimal levels of fluid replacement. My clinical and anecdotal impression is that the pattern of presentation of ketoacidosis is changing. Either because of newer insulins or, more probably, because of the newer regimens of insulin delivery patients are presenting earlier, with severe acidosis yet not necessarily severely dehydrated. I believe that overzealous fluid replacement in these cases may be potentially, and at worst is definitely, harmful. In patients who are haemodynamically stable I adopt an initial rate of fluid replacement of 2-3 ml/kg/h, which is well below that recommended in recently published textbooks.2 Problems have arisen only when doctors have not followed the local protocol.

Experimental proof of this hypothesis is difficult, but support for lower rates of fluid replacement is increasing.<sup>34</sup> Diabetic ketoacidosis carries a significant mortality,<sup>2</sup> much greater than that from hypoglycaemia.<sup>5</sup> Improvements in its management will be achieved only by careful audit of cases. In addition, I think that there is sufficient doubt about the necessity and safety of using the currently recommended initial rates of fluid replacement that a carefully controlled study needs to be performed. This will not be easy and by necessity will need to be a multicentre study, but the cardiologists have shown us the way. It is time for the diabetologists and British Diabetic Association to grasp the nettle.

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 Hammond P, Wallis S. Cerebral oederna in diabetic ketoacidosis. BMJ 1992;305:203-4. (25 July.)

2 Dornan T. Ketoacidosis and hyperosmolar coma. In: Tattersall RB, Gale EAM, eds. Diabetes clinical management. Edinburgh: Churchill Livingstone, 1990:214-27.

- Androgue HJ, Barrero J, Eknoyan G. Salutary effects of modest fluid replacement in the treatment of adults with diabetic ketoacidosis. *JAMA* 1989;262:2108-13.
  Duck SC, Wyatt DT. Factors associated with brain herniation in
- 4 Duck SC, Wyatt DT. Factors associated with brain herniation in the treatment of diabetic ketoacidosis. J Pediatr 1988;113: 10-4.
- 5 Tunbridge WMG. Factors contributing to deaths of diabetics under fifty years of age. Lancet 1981;ü:569-72.

## Poisoning and child resistant containers

EDITOR,—Minerva's extract from the Journal of Epidemiology and Community Health says that the credit for the reduction in childhood poisoning cannot go to the introduction of child resistant containers because they came into general use in 1981 and "the admission rates for poisoning with substances not in safe containers, such as berries, plants, and mushrooms, have fallen more than for poisoning with drugs."<sup>1</sup>

This cannot be allowed to go unchallenged. In drawing a comparison between two methods of prevention it is important to limit methodological differences-in this case the use of child resistant containers. Before these containers were introduced in 1976 for aspirin and paracetamol sold over the counter for children some 7000 children were admitted to hospital with poisoning annually. These drugs sold over the counter for adults were packaged in child resistant containers the next year, and by the end of 1978 the number of children admitted with poisoning had fallen to below 2000. Admission of children from drugs not in child resistant containers had, however, remained unchanged.2 So far as we are aware, the only difference between the two groups was the packaging of the drugs. If the authors of the paper can tell us of any other measures that have reduced the admissions of children to hospital by 5000 a year we would be glad to hear of it.

There is another misleading statement in the paper. Though child resistant containers "came into general use" in 1981, this was by voluntary arrangement, and they were not used anywhere near as widely as had been hoped. It is not surprising, therefore, that the rate of poisoning from drugs did not fall rapidly. The Royal Pharmaceutical Society has therefore now made it a professional requirement that child resistant containers or strip or blister packs must be used unless the recipient specifically requests otherwise.

Of course, several other factors need to be taken into account in considering the numbers of children admitted to hospital with poisoning. Poisoning from berries and so on may be less common than previously as a result of education or the criteria for admission may be stricter than formerly, or both. We remain convinced, however, that use of child resistant containers has been one of the most effective ways of preventing accidents yet introduced.

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1 Minerva. BMJ 1992;305:128. (11 July.)

2 Jackson RH, Craft AW, Lawson GR, Beattie AB, Sibert JR. Changing patterns of poisoning in children. BMJ 1983;287: 1468

## Health service support of breast feeding

EDITOR,—Sally Beeken and Tony Waterston note the wide discrepancy between hospital policy and practice in the establishment and continuation of breast feeding in Newcastle upon Tyne.<sup>1</sup> Such a discrepancy was also evident in a study I carried out in Fife Health Board last year. There the