Injuries sustained on "bouncy castles"

SIR,—We wish to report the dangers of playing on "bouncy castles." A bouncy castle is an inflatable children's playground, consisting essentially of a rubber mattress inflated with air. Three walls generally surround the castle, with the fourth side open to allow entry and exit, but some castles are open on all four sides. They are popular in fairgrounds and recreational halls. During last summer five children and one adult were treated at Northwick Park Hospital for injuries sustained while they were playing on bouncy castles. All injuries were to the arm or neck.

Three supracondylar fractures of the humerus occurred. Two of these required open reduction and internal fixation with Kirschner wires. The third was managed with a collar and cuff support. A girl aged 6 sustained a fracture of the mid-shaft of the humerus, which was managed with a hanging cast support. The neck injuries occurred in a 52 year old man and a 12 year old boy. These were both soft tissue injuries caused by flexion and were managed conservatively.

The hazards of playgrounds to young children are well recognised, and, among other measures, absorbent surfaces have been recommended to help reduce the severity of injuries.¹ The injuries that we report are due to the unpredictable nature and power of the bounce of these castles—part of their fun. Most injuries occur when those playing on the mattress fall off it; the surrounding surface is firm rather than cushioned. The weight of the body on an outstretched hand accounts for the frequency of supracondylar fractures.² To our knowledge injuries from playing on bouncy castles have not been reported previously, though many injuries, particularly to the arm, have been reported in people playing on giant inflated cushions without surrounding walls.³

The risk of sustaining serious injury, particularly to the arm, while playing on bouncy castles could be reduced by providing a cushioned landing around the open side of the castle. Avoiding overcrowding and setting an age limit may also help, and supervision by a responsible adult should be mandatory.

GIAN SINGER
LAWRENCE S FREEDMAN

Northwick Park Hospital,
Harrow,
Middlesex HA1 3UJ

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Self poisoning by adolescents

SIR,—L McGibben and colleagues' short report on the relation between school attendance and deliberate self poisoning by adolescents¹ prompted me to examine our records of deliberate self poisoning by adolescents in Bradford. A preliminary study confirmed the authors' finding that the rate of self poisoning is lower during school holidays than during school terms.

In Bradford 321 adolescents aged 12-16 were

referred for a child psychiatric assessment after admission for deliberate self poisoning in the four years 1988-91. Bradford's education department supplied me with the dates of school terms and numbers of schoolchildren aged 12-16 for each of the four years studied. The average number of schoolchildren in schools covered by Bradford Health District during the four years was 22 950.

The rate of admission per 1000 schoolchildren aged 12-16 was 4.00 a year during school terms (95% confidence interval 3.37 to 4.64) and 2.12 a year during school holidays (1.68 to 2.56). I was not able to show any significant difference between rates for different holidays.

Clearly, school attendance as a factor in deliberate self poisoning by adolescents warrants further attention.

E A PROCTER

Child and Family Unit,
Bradford NHS Hospitals Trust,
Bradford BD5 0HT

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The cholesterol controversy

SIR,—Michael F Oliver's editorial on the failure of intervention programmes to prevent heart disease highlights the need to change the emphasis of prevention away from screening for so called risk factors towards promoting the positive, healthy aspects of every situation and making change relevant to people's priorities.¹

Only one such intervention programme has ever shown a beneficial effect in terms of years of life saved when the intervention group was compared with a control group. Other studies show that men may feel worse three months after a well man check than they did before;² people labelled as hypertensive may develop symptoms of depression and have an increased rate of absenteeism from work;^{3,4} patients are more concerned with having a listening doctor than with a clinic's facilities;⁵ and "at least half (of the working class women under study) hold fatalistic views about illness causation."⁶ The message is clear: our present health promotion strategies may well change behaviour, but not necessarily in a healthy direction.

The exception to these intervention programmes has been one in North Karelia, where between 1974 and 1979 age standardised mortality fell by 22% in the intervention group compared with 12% in the control group.⁷ The programme was also unique for one other reason: it was the only one that happened because it was demanded by the public.

If strategies to prevent heart disease are under scrutiny so must be our understanding of what causes the disease. Except with smoking, no cause and effect has ever been proved between the variables that people like to call risk factors and the disease itself. We have to reconcile our understanding of the causes of heart disease with the results of research done in Roseto, Pennsylvania, exposed by the *Horizon* programme "Sudden Death" (7 January 1991). A group of fat, smoking Italians in America were investigated because of their unusually low incidence of heart disease. The younger generation developed the American

lifestyle of not smoking, taking a low fat diet, holding fast track jobs, and breaking from community ties; their incidence of heart disease increased.

Health promotion means promoting health. To most people health means believing that they are worth while and have enough control over their lives to be able to achieve things and be creative. This must be our goal. Health promotion strategies must decrease their emphasis on the individual and be "owned" by communities and families acting on their health priorities. Professionals must be used as a resource—a partnership—to enable realistic action plans to develop the positive aspects of people's lives and their society. In this way we can be sure of catching what motivation to change there is and work towards the real goal, health. With this approach we should also reduce the levels of heart disease.

PAUL THOMAS

Primary Health Care Facilitation Project,
Vauxhall Health Centre,
Liverpool L5 8XR

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SIR,—George Davey Smith and Juha Pekkanen take meta-analysis of primary prevention trials for coronary heart disease a stage further¹ by adding three trials to the six considered by Muldoon *et al*² and then grouping dietary trials separately from drug trials. The disturbing excess of non-coronary mortality is then confined to the drug trials. In addition to these single factor trials, total mortality was lower in the intervention groups in each of three major multiple risk factor primary prevention trials in which diet lowered plasma cholesterol concentration but drugs were not used.^{3,5} Thirdly, in the three largest trials of secondary prevention which included effective diet but not drugs mortality was also lower in the intervention groups.^{6,8} When these nine trials (with 72 000 participants) are put together the total number of deaths was 1581 in the combined intervention groups—that is, 93% of the 1693 deaths in the control groups. Total mortality was lower in the intervention groups in eight of the nine trials.

The prevention trials for coronary heart disease were designed to show a significant reduction in coronary events, not mortality. Many more subjects, a longer duration, and greater expense would be needed to test this.

There is a growing body of information that Michael F Oliver did not consider: the changing patterns of mortality in different countries. In the past 30 years the number of age standardised deaths from coronary heart disease has fallen considerably in the US, Australia, and Canada and

by smaller amounts in New Zealand, Switzerland, Finland, Belgium, and Israel.¹⁰ All cause mortality has fallen in these countries at the same time. By contrast, the countries of eastern Europe—for example, Hungary, Poland, and Czechoslovakia—have suffered large increases in coronary deaths accompanied by rises in total mortality. In an intermediate group of countries—for example, Sweden, Denmark, Ireland, England and Wales, Scotland, and Northern Ireland—changes in coronary mortality have been small or late.

Those who have examined these changes in contrasting countries conclude that dietary change seems to have been the most consistent variable in lifestyle—notably an increase in ω -6 polyunsaturated fatty acids in countries with falling mortality¹¹ and an increase in animal fat in countries with rising coronary and total mortality.¹² In England and Wales coronary mortality started to move downwards in about 1977¹³; this coincided with the start of an increase in the ratio of polyunsaturated to saturated fatty acids in the average household's diet.¹⁴

Oliver's group in Edinburgh found significant inverse and progressive relations between the linoleic acid content of adipose tissue and the risk of angina and myocardial infarction in a population based case-control study.¹⁵ Four other case-control studies have yielded similar findings. Other components of the dietary package first recommended for preventing coronary disease at the end of the 1950s may play important parts as well—namely, reduced total fat, reduced saturated and hydrogenated fats, and increased vegetable, fruit, and fibre intakes.

A STEWART TRUSWELL

Human Nutrition Unit,
University of Sydney,
New South Wales 2006,
Australia

- 1 Davey Smith G, Pekkanen J. Should there be a moratorium on the use of cholesterol lowering drugs? *BMJ* 1992;304:431-4. (15 February.)
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SIR,—One of the hazards of writing an editorial is that some do not trouble to read it with care or without prejudice. Thus M F Ryan and colleagues have decided¹ that my appraisal of the Helsinki trial is unsound because I suggested that some of the adverse effects 10 years after the end of the trial might be related to the use of hypolipidaemic drugs

during the trial.² But neither I nor the authors of the trial made any such suggestion. I did not even refer to these drugs in relation to the Helsinki trial.

Other correspondents have focused entirely on the Helsinki trial and have ignored the equally or more important negative results of other major trials.¹ The doubts about preventing coronary heart disease stem from the overview of all these studies. In short, multifactorial primary prevention of the degree generally recommended during the past two decades has failed to prevent coronary heart disease in middle aged men.

The more stringent approach to hypercholesterolaemic men used in the Oslo trial did lead to a reduction in the incidence of coronary heart disease: cigarette smoking was reduced by 45% and serum cholesterol concentration was 13% lower in the intervention group.³ A diet enriched in ω -6 polyunsaturated fat was used (polyunsaturated: saturated ratio ≥ 1.0), supporting A Stewart Truswell's emphasis on their importance. I fully agree with him.

Unfortunately, there is no simple explanation for the decrease in mortality from coronary heart disease in the United States, Australia, Canada, England, Wales and even Scotland as the downturns in the incidence of both coronary heart disease and stroke began before public health advice on prevention was widely put into practice.

I agree with Tony Winder and J P D Reckless that people at high risk must be identified and should receive aggressive treatment for their specific risk factors.¹ But we must now move on, and a renewed research effort is needed to study causes of coronary heart disease other than smoking, cholesterol, and blood pressure so that new initiatives in prevention can be established.

M F OLIVER

Wynn Institute for Metabolic Research,
National Heart and Lung Institute,
London NW8 9SQ

- 1 Correspondence. The cholesterol controversy. *BMJ* 1992;304: 711-3. (14 March.)
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SIR,—J P D Reckless and J A Tobert¹ disagree with the inclusion criteria for the studies in our review of cholesterol lowering and mortality.² Reckless suggests that we “look at only a third of relevant studies.” He may have missed the fact that our quantitative analysis was solely of the primary prevention studies. It is in primary prevention, as we made clear, that we suggest caution should be exercised before the use of cholesterol lowering drugs expands rapidly.

Tobert suggests that the expanded clinical evaluation of lovastatin (EXCEL) trial should not have been included as a primary prevention trial as 28% of the participants suffered from pre-existing ischaemic heart disease.³ Our definition of primary prevention trials was those studies for which previous ischaemic heart disease was not an entry criterion. Thus, for example, in the colestipol-Upjohn study 31% of participants had evidence of ischaemic heart disease at entry.⁴ The classification of studies we used follows that of other reviewers.⁵⁻⁷

We agree with Tony Winder that inappropriate use of cholesterol lowering drugs is the danger.¹ Our worry is that the current exponential increase in prescription rates, together with widespread promotion of guidelines for treatment which suggest that a substantial proportion of the British population is eligible, makes such inappropriate use likely. Current evidence of the effects of cholesterol lowering drugs on mortality cannot be viewed as favourable. Lovastatin is now the most frequently prescribed cholesterol lowering drug in

the United States,⁸ where its use was already widespread and rapidly increasing by 1988.⁹

Tobert suggests that in the EXCEL trial differences in mortality “did not approach significance ($p>0.2$ by the Kaplan-Meier method)”; we assume a log rank test is meant. With the small number of deaths in this study, survival analysis closely approximates to a simple χ^2 test. A global comparison across the four lovastatin groups and the placebo group produces a similar statistic ($\chi^2=5.21$, 4 df, $p=0.27$). Combining the four lovastatin groups and making the comparison of active treatment versus placebo, however, gives a different picture ($\chi^2=3.15$, 1 df, $p=0.076$). Perhaps the more than twofold increase in mortality among the patients treated with lovastatin should not be dismissed so lightly. Certainly, more powerful data should be obtained before doubts about the wisdom of widespread promotion of these drugs are dismissed.

On the principle of first do no harm we find it difficult to see how expanding the use of cholesterol lowering drugs in the primary prevention of ischaemic heart disease in patients other than those with severe familial hyperlipidaemias can be justified, even though these drugs apparently protect against non-fatal ischaemic heart disease. As Rose pointed out,¹⁰ in primary prevention of disease a small adverse effect can often outweigh any benefit; thus there should be stringent requirements for the acceptable level of evidence regarding safety. A large enough randomised clinical trial should be carried out to examine the effects of pharmaceutical lowering of cholesterol concentrations on all cause mortality, and therapeutic restraint should be maintained until this is completed.

GEORGE DAVEY SMITH

Department of Public Health,
University of Glasgow,
Glasgow G12 8RZ

JUHA PEKKANEN

Department of Epidemiology,
National Public Health Institute,
Helsinki,
Finland

- 1 Correspondence. The cholesterol controversy. *BMJ* 1992;304: 711-3. (14 March.)
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Age associated memory impairment

SIR,—Thomas H Crook and Steven H Ferris accuse us of arguing against the investigation of possible pharmacological treatment of age associated memory impairment.¹ We do nothing of the sort. Our editorial was subtitled “Too broad an entity to justify drug treatment yet” and emphasised the