uneventful pregnancies giving rise to unaffected children had the screening programme been offered to them: a tricky concept. (It is not easy to see how that estimate of cost is obtained from their abbreviated table III.) The cost of permanent care of people born with Down's syndrome relative to that of an assumed normal birth cohort is so low partly because the estimated cost of permanent care of 45-year-olds (the age at which 100% of Down's syndrome births are assumed to be in permanent care) is low (about £250 per person per annum) but principally because, when discounted at 10% per annum for 45 years, it gives a "present value" of only £3 per person per annum.

Next they estimate the total (relative) "present value" cost (of caring for people born with Down's syndrome) as £4150 per person per annum. Yet in their summarising paragraph they call this (8·1 affected births) a benefit and it is discounted a second time at 10% in the calculation of a total programme benefit. This notwithstanding that their calculations suggest for West Scotland a "replacement" benefit of some £350 000 and a "no replacement" benefit of £800 000; in other words society will benefit to the tune of nearly £½m ("present value") by preventing each year eight uneventful pregnancies resulting in eight normal births (after amniocentesis and terminations for the eight affected).

This illustrates how a high discount rate makes distant future earnings of the newborn negligible compared with the present costs of infant care and the near future costs of schooling, even in the healthy. One of the benefits that has to be taken into account (even if it is not given a cash value) is the value of life per se.[1] The logical conclusion derivable from their calculation is not to screen the over-40s for Down's syndrome but to prevent all births (whether "replacement" or not).

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[1] Buxton, M J, and West, R R, *British Medical Journal*, 1975, **2**, 376.

Effect of skin-cleaning agents on Dextrostix readings

SIR,—With the increasing use of reagent strip tests for biochemical screening we would like to report a potential cause of misleading results which we have encountered in the use of Dextrostix and which is illustrated by the following case.

A 79-year-old insulin-dependent diabetic was brought to the accident and emergency department unconscious. Apart from increased muscle tone and bilateral extensor plantar responses there were no abnormal physical signs. An initial assessment of blood glucose was made with Dextrostix reagent strips by two independent observers from samples of capillary blood obtained by finger prick. Visual readings were recorded as falling between 9·7 and 13·9 mmol/l (175 and 250 mg/100 ml). However, formal laboratory estimation on a venous blood sample taken at the same time gave a value of 1·67 mmol/l (30 mg/100 ml) and as a consequence 50% glucose was then given intravenously, with rapid improvement in the patient's clinical condition.

The Dextrostix reagent strips used were from a freshly opened bottle and when tested on venous blood were found to give accurate and reproducible results, thus indicating that the discrepancy was in the technique used rather than in the Dextrostix reagent strips themselves. Further inquiry revealed that before the finger stab the skin had been liberally swabbed with 70% isopropyl alcohol (Sterets swab).

Ethanol or 70% isopropyl alcohol when added to blood produces a sticky brown precipitate of denatured haemoglobin that will adhere to the surface of a Dextrostix reagent strip. Possibly residual alcohol on the skin after swabbing may mix with blood to produce this brown precipitate, which can impart a brown colour to the test surface. This may be interpreted by an uncritical observer as indicating a falsely high blood glucose level, particularly in poor lighting and when the true blood glucose is low. More significantly, Dextrostix reagent strips discoloured by the brown precipitate also give falsely high readings when used with a reflectance meter (Eyetone, Ames Co).

These misleading results may occur when rapid determinations are being made in emergencies with appreciable quantities of alcohol remaining on the skin surface. This appears to have been the explanation in this case and this phenomenon has not been reported previously. We would therefore recommend that any alcohol-based skin cleansing preparation be allowed to evaporate completely before skin puncture is performed and that any brown colour on the test surface should be treated with extreme suspicion. This will avoid the potential danger of misdiagnosing hypoglycaemia when using Dextrostix reagent strips.

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Treatment of diarrhoea in undernourished children

SIR,—Dr G Hatcher (6 March, p 571) advises adamant resistance to any milk feeds being given to infants and children under 3 years of age for 24 hours after commencing treatment of a mild diarrhoea because of the risk of hyperosmolar dehydration.

It is essential to stress that this practice is extremely hazardous in infants and children in whom there is any degree of undernutrition. The *BMJ* is read widely in many parts of the world and it behoves those who review problems of childhood to take into account all the different conditions in which they may occur and which may modify management considerably.

A recent WHO guide[1] to the management of diarrhoea for primary health auxiliaries has stressed the importance of early feeding, starting not more than six hours after hydration has been instituted. Hyperosmolar dehydration presents usually in infants on highly concentrated feeds before the start of treatment but should never develop during treatment, in which adequate hydration is as imperative as early calorie-protein intake. Furthermore, it is not clear whether Dr Hatcher would include breast-feeding in "milk feeds". I cannot believe he would. Twenty-four hours' withdrawal of breast-feeding would expose infants in many parts of the world to further risk of the diarrhoea-malnutrition circle, with the likelihood of total termination of breast-feeding. In addition, breast milk is highly unlikely to produce a hyperosmolar state.

The further suggestion that milk should not be recommended for the much longer period of up to three weeks in severe diarrhoea on account of the risk of temporary lactose intolerance serves only to increase my anxiety concerning the adoption of the practice in undernourished infants in the first year of life, and particularly in areas of the world where low-lactose alternative feeds are not available. Diarrhoea may increase slightly when milk feeding is recommenced, but as long as hydration is maintained this is of little consequence compared with the necessity for maintaining and improving the nutritional state of the infant.

Finally, there is strong evidence[2] that the use of glucose-electrolyte solutions given orally in adequate amounts restores appetite and allows feeding to be started earlier, consequently with less weight loss and more rapid nutritional recovery. Water on its own is less effective in this respect, especially if it is unaccompanied by early feeding.

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[1] World Health Organisation, *Treatment and Prevention of Dehydration due to Diarrhoea—Guide for Workers at Primary Level*. Geneva, WHO. In press.
[2] Hirschhorn, N, *et al*, *Journal of Pediatrics*, 1973, **83**, 562.

Glucose absorption and diabetes

SIR,—Professor G C Cook (20 March, p 688) reports that an unselected group of Arabs in Riyadh absorbed glucose more rapidly than a group of Africans studied in Lusaka and comments that the prevalence of diabetes differs in the inhabitants of these two areas. He suggests that increased sucrose consumption may result in portal hyperglycaemia which stresses the pancreas and thus impairs glucose tolerance. However, as he also points out, both Arabs and Africans eat sucrose, the Africans adding it to their staple carbohydrate diet. Consequently sucrose alone cannot be responsible for this phenomenon. He suggests that subclinical or inadequately treated gut infections may be the agent responsible for slower absorption in Africans.

While accepting these arguments we suggest that the form in which food is taken may be another factor. Trapping of otherwise available carbohydrate within the intact cell walls[1] may reduce the amount of available carbohydrates. Dietary fibre or other unavailable carbohydrates (storage polysaccharides) which may modify the physicochemical properties of the intestinal contents may also be important. Some of the undigestible polysaccharides form gels which reduce postprandial glycaemia,[2 3] possibly by transient trapping of glucose molecules within the gel, thus delaying their absorption.

If β-cell stress of the pancreas is a factor in the development of diabetes, then any agent which modifies postprandial glycaemia may be a factor which alters predisposition to diabetes. The mode of action by which such substances may operate to modify glucose tolerance in the long term has been mentioned previously.[4] We have studied the effect of two unavailable carbohydrates, Guar gum (a storage polysaccharide of a cluster bean, *Cyamopsis tetragonoloba*) and pectin, on non-insulin requiring diabetics.[5] When 26 g of these