associated—as is now becoming apparent. Indeed, the situation with regard to both B27 and B5 associations may vary in different parts of the world. In our patients with Behçet's triad, two out of four are B5 positive and one out of four is B27 positive. Of those with "definite" Behçet's disease (by the criteria of Mason and Barnes²), two out of 11 are B5 positive and only one out of 11 is B27 positive. The connection with the B5 antigen seems less strong than in Turkey but further work is necessary to clarify the position.

The criteria of Mason and Barnes are valuable in indicating some measure of diagnostic certainty. Patients with the triad of symptoms undoubtedly have Behçet's syndrome. But some patients satisfy their criteria at the "definite" level without the concurrence of mouth and genital ulceration which I would now regard as obligatory. Nor would I attach much importance to the presence or absence of a family history.

Thus the stated prevalence rate of 0.064 per 10 000 can be only an estimate of the disorder's prevalence: it may be a slight overestimate. In any event it contrasts markedly with the Japanese finding of 1 in 10 000.⁶

M ANNE CHAMBERLAIN

Rheumatism Research Unit,
School of Medicine,
University of Leeds

¹ Chamberlain, M A, *Annals of the Rheumatic Diseases*, 1977, **36**, 491.

² Macrae, I, and Wright, V, *Annals of the Rheumatic Diseases*, 1973, **32**, 16.

³ Haslock, I, *The Arthritis Associated with Crohn's Disease: A Family Study*. MD thesis, University of Edinburgh.

⁴ Moll, J M H, *A Family Study of Psoriatic Arthritis*. DM thesis, University of Oxford.

⁵ Mason, R M, and Barnes, C G, *Annals of the Rheumatic Diseases*, 1969, **28**, 95.

⁶ Aoki, K, Fujioka, K, and Katsumata, H, *Japanese Journal of Clinical Ophthalmology*, 1971, **25**, 2239.

Burn hazard with cement

SIR,—The article by Mr M W Flowers (13 May, p 1250) contains some inaccurate and misleading statements about the lime content and caustic properties of Portland cement and concrete that require correction.

Mortar containing lime or hydraulic cement has been used by humans since ancient times and its characteristic of being an alkaline or caustic material has always been present. Some persons seem to be particularly sensitive to skin reactions with caustics while others have more tolerance to contact. Lime is a product of universal distribution, used for many purposes, and knowledge of its properties is widespread.

Portland cement is a hydraulic product, which means that the constituents will react with water and in so doing it becomes a hydrated material. Water reacts with the silicates, aluminates, and any free lime which is sometimes present in small quantities. As a result of this reaction with water calcium hydroxide (hydrated lime) is formed. This is responsible for giving a cement mortar or concrete its alkaline or caustic property. Hydrated quicklime is the same material as this reaction product.

There is no "free calcium oxide" in hydrated Portland cement concrete. When the concrete is fresh and plastic, calcium hydroxide is present and is the reason for "burns" when proper protective measures are not followed. It is only necessary for the worker to avoid contact on exposed skin for any prolonged length of time. The use of watertight boots, gloves, and clothing will prevent this contact, while any accidental splash landing on exposed skin must be promptly washed away.

After concrete loses its plasticity and becomes

hard any calcium hydroxide residing on the surface rapidly reacts with carbon dioxide from the atmosphere and becomes a relatively inert material that is no longer caustic. Hardened concrete will probably contain only 15% Portland cement and the remainder will be combined water, sand, and coarse aggregates (possibly limestone in this instance). Of the 15% cement in the concrete, about 25% of calcium hydroxide will ultimately be generated; this means that the maximum content of calcium hydroxide will be 15% × 25% = 3.75%, which is 10% of the value quoted in Mr Flowers's report. With reference to the statement in the article that hardened concrete contains "37% by weight of free calcium oxide," it must be assumed that this refers to the total calcium oxide in the concrete as reported in the customary manner for chemical constituents and therefore includes the calcium in the aggregate. Thus it does not mean that free calcium oxide actually exists in the concrete, but is simply the quantity of calcium, expressed as the oxide, in the entire mass.

Since some persons will use this material without regard to warnings or without knowledge of the product it would be most helpful if medical personnel could easily recognise the symptoms and provide the proper attention. It is common practice in the United States for manufacturers of Portland cement to place warning notices on sacked cement.

N R GREENING

Director,
Chemical Physical Research Department

J R TONRY

Principal Research Engineer,
Environmental Portland Cement Association

Skokie, Illinois

Thyroid disease and pregnancy

SIR,—A paper on "Myxoedema in young women" that I published in 1963¹ would appear to be relevant to your leading article (7 October p 977). That paper described four women between 25 and 37 years old who showed good evidence of subhypothyroidism when seen 3–20 months after normal confinements.

Their thyroid glands could not be felt or were very small. They all returned to full health on thyroid treatment by mouth and became pregnant again between three months and five years later and had healthy babies. One developed lupus erythematosus, which responded well to treatment, but she died from a florid systemic form of it in 1976. The others have remained well on permanent oral thyroxine.

In April 1968 two other striking cases were seen within 10 days. Women 23 and 22 years old were referred by their general practitioners for investigation of suspected myxoedema. The clinical picture was frank but not florid, their thyroid glands were very small or impalpable, and test results confirmed the diagnosis—namely, low basal metabolic rate, high blood cholesterol concentration, delayed ankle and knee jerks, and low-voltage electrocardiograms. The first gave negative tests for thyroid antibodies. She had a son nearly 3 years old, the other had sons of 22 months and 8 months. Both returned to normal on oral thyroid and one had a baby girl after 14 months, the other a baby girl after two years.

In May 1977 Ginsberg and Walfish² reported from Toronto on "postpartum transient thyrotoxicosis with painless thyroiditis." Of their five patients (whose thyrotoxicosis resolved within four months), four later developed transient hypothyroidism and one of these developed permanent myxoedema. They

thought that painless subacute thyroiditis was probably the underlying condition in most of their patients. They referred to the 1976 paper by Amino *et al*³ from Osaka, which also described transient hypothyroidism occurring 3–6 months after delivery. In 1963 the BMA librarian could trace no references to myxoedema following pregnancy; and another search completed in August 1977 revealed only the three papers that I have mentioned.

The observation of this group of cases in Hartlepool has always left a suspicion that they represent the tip of an iceberg, as proved to be the case with Hashimoto's disease after 1956.⁴ To find other cases all women should be briefly seen six months after delivery to determine if there are any symptoms of subhypothyroidism (especially puffiness of the face) and to feel whether the thyroid gland is abnormally small, abnormally large, tender, or firm. In my opinion this condition is due to postpregnancy hyperinvolvement of the thyroid.

R T COOKE

Hartlepool, Cleveland

¹ Cooke, R T, *Journal of the College of General Practitioners*, 1963, **6**, 626.

² Ginsberg, J, and Walfish, P G, *Lancet*, 1977, **1**, 1125.

³ Amino, N, *et al*, *Journal of Clinical Endocrinology and Metabolism*, 1976, **42**, 296.

⁴ Luxton, R W, and Cooke, R T, *Lancet*, 1956, **1**, 105.

Clinical information on x-ray and pathologist requests

SIR,—Dr B Golberg's Personal View on x-ray examinations (7 October, p 1017) includes a *cri de coeur* common to pathologists as well as radiologists. His solution is to include "imaging" as a subject in the undergraduate and postgraduate curriculum. May I suggest another?

Personal contact between radiologist and clinicians is an unrivalled opportunity for exchange of information. How often does Dr Golberg share his coffee break and lunch with junior clinical medical staff? Informal social exchange at this level may be worth more than many periods of formal education.

L J H ARTHUR

Derbyshire Children's Hospital,
Derby

Beta-blockade in subarachnoid haemorrhage

SIR,—In their study of patients with subarachnoid haemorrhage (SAH), Mr G Neil-Dwyer and others (7 October, p 990) have demonstrated that treatment with propranolol and phentolamine has a highly significant cardioprotective effect. The cardiac lesions seem to be related to excess catecholamines and beta-blockade therefore appears to be the crucial therapeutic factor. We have recently had a patient with SAH in whom treatment with oxprenolol failed to prevent ECG changes, which were so grossly abnormal that we were concerned that the patient might have additionally sustained a silent myocardial infarction.

The patient, a fit 62-year-old man, had been maintained on oxprenolol 80 mg thrice daily for hypertension, which was thus well controlled. However, shortly after his admission with SAH (confirmed by lumbar puncture) his blood pressure rose to levels of about 180/105 mm Hg and oxprenolol was increased to 80

mg four times a day, and then to 160 mg four times a day. His ECG initially showed widespread T-wave inversion, but this abnormality was most marked two days later, with gross T-wave inversion in most leads (figure). Serial enzyme studies failed to confirm myocardial damage.

ECG of patient with subarachnoid haemorrhage showing T-wave inversion.

Thus this patient was already taking a beta-blocker, in a dose comparable to 240 mg propranolol, but despite a further increase in dosage and apparently clinically adequate beta-blockade (resting pulse rate about 60 beats/min) he proceeded to develop grossly abnormal ECG appearances, of a severity which we have not previously observed in association with subarachnoid haemorrhage.

HENRY L ELLIOTT
BRIAN C CAMPBELL
IMOGEN MORGAN

Department of Materia Medica,
Stobhill General Hospital,
Glasgow

Pertussis and the risk of non-accidental injury

SIR,—Pertussis is described in your leading article (22 April, p 1007) as "often a frightening and sometimes terrifying illness," but its social implications have not been amplified. The troublesome paroxysms of coughing can impose such a strain on parents that child abuse becomes a real danger, as highlighted recently by the following case.

A mother of a 15-month-old boy had lost much sleep over her son's whooping cough, which he had been suffering from for four weeks. She had become increasingly agitated by the paroxysms of coughing and was witnessed to have shaken the boy violently during one of these paroxysms. The incident took place in the family doctor's surgery and it was felt that the boy was at risk. A Place of Safety Order was necessary, so that the child could be admitted to hospital. After admission of the child his mother seemed relieved and later divulged that she had become so irritated by her son's coughing that she probably would have harmed him. At a subsequent court hearing, the child was returned home with the provision of a supervision order for three years. The child had by then recovered from whooping cough.

This child may be one of many children with pertussis whose symptoms are sufficiently distressing for their parents to subject them to the real danger of physical harm. Medical complications of pertussis are well recognised,¹

but social complications are much less apparent. As we are in the midst of an epidemic of pertussis² (the worst since 1960), which will probably last until mid-1979, it is pertinent to bear in mind the distressing nature of the disease for parents and consider hospitalisation, even for mild cases, where a risk of non-accidental injury exists.

We would like to thank Dr W C Marshall, who drew our attention to this aspect of pertussis.

D S C LEE
G MCENERY

Whipps Cross Hospital,
London E11

G F NORRIS

The Health Centre,
London E4

¹ Lloyd, C Olson, *Medicine*, 1975, **54**, 427.
² *British Medical Journal*, 1978, **1**, 1007.

Borderline substances

SIR,—The bureaucratic treatment of the question whether Optimax drinking chocolate is a food or a drug is rivalled only by the situation in which the Department of Health, whose inspectors stamp on the slightest degree of cross-contamination in pharmaceutical production, insists that only food containing traces of gluten may be prescribed under the National Health Service for the treatment of coeliacs. The present policy of the Department's Borderline Substances Committee is to permit only non-luxury food items to be prescribed, and with hidebound logic they maintain that to allow naturally gluten-free flours on prescription would mean that any other essential food that contained no gluten—for example, meat and vegetables—would have to be allowed too. Such an argument is so ridiculous as to be almost beyond belief.

Practically all the "gluten-free" products that are allowable for the 100 000 coeliacs in the United Kingdom are based on gluten-containing flours such as wheat. It is impossible to remove all traces of gluten from such flours and a draft FAO standard states that "gluten-free" food may contain "wheat, rye, barley or oat flour from which all gluten has, so far as is practicable, been extracted." Australian recommendations allow a maximum of about 0.3% gluten.¹ Treatment of coeliac disease is by complete exclusion of gluten from the diet² and lack of response or suboptimal response is commonly due to failure to eliminate gluten completely.³ As an example, the case has been reported of a patient with confirmed coeliac disease who failed to improve until it was discovered that she was receiving Communion wafers containing wheat flour daily.¹ Baker *et al*³ estimated that 65% of coeliacs continue to ingest gluten even when on a supposedly gluten-free diet. Some of this inadvertently ingested gluten is undoubtedly derived from so-called "gluten-free" foods which contain enough residual protein to provoke symptomatic exacerbations in at least some coeliacs and dermatitis herpetiformis patients.⁴

Alternative foods that naturally contain no gluten, such as rice, maize, and potato flours, are available and are generally cheaper than the prescribable "gluten-free" foods. All are just as capable as the gluten-reduced flours of producing palatable and nutritious bread, cakes, and biscuits. The cheapest of the

naturally gluten-free flours (maize) costs 72p per kg less than the dearer of the two most commonly prescribed proprietary brands based on wheat flour, while the most expensive natural flour (rice) costs 11.6p per kg less than the cheaper proprietary brand.

R J WOODWARD
R BAXTER

Larkhall Laboratories,
London SW15

¹ McCausland, J, and Wrigley, C W, *Journal of the Science of Food and Agriculture*, 1976, **27**, 1203.

² Davidson, S, *et al* (editors), in *Human Nutrition and Dietetics*, p 483. Edinburgh, Churchill Livingstone, 1975.

³ Townley, R R W, and Anderson, C M, *Ergebnisse der Inneren Medizin*, 1967, **26**, 1.

⁴ Price, H, Zownir, J, and Prokipchuk, E, *Lancet*, 1975, **2**, 920.

⁵ Baker, P G, Barry, R E, and Read, A E, *British Medical Journal*, 1975, **1**, 486.

⁶ Monro, J, *Lancet*, 1975, **2**, 920.

Immunisation of adults against diphtheria

SIR,—In his letter (30 September, p 962) Dr S E Ellison details the schedule used for adults at the Central Middlesex Hospital: two doses of 0.2 ml (10 Lf) of adsorbed diphtheria vaccine for those with no history of previous immunisation, and one dose of 0.5 ml (25 Lf) for those previously immunised. We are surprised to learn that no severe reactions have been reported over a period of 10 years, particularly as Schick testing was not done before immunisation.

Severe local and general reactions have been reported¹ in adults following the use of as little as 5 Lf of purified diphtheria toxoid, mainly in those with an allergic reaction (pseudo-reaction) to the Schick control. During an outbreak of diphtheria in Manchester in 1971 severe reactions resulting in time off work were common in adult health workers given 10 Lf of diphtheria vaccine.² For this reason in adults we have been using a preparation containing 1.5 Lf of adsorbed diphtheria toxoid (similar to that described by Edsall *et al*³).

J D ABBOTT

Public Health Laboratory,
Withington Hospital,
Manchester

A G IRONSIDE

Regional Department of Infectious
Diseases,
Monsall Hospital,
Manchester

F W SHEFFIELD

National Institute for Biological
Standards and Control,
London NW3

¹ Pappenheimer, A M, *et al*, *American Journal of Hygiene*, 1950, **52**, 353.

² Butterworth, A, *et al*, *Lancet*, 1974, **2**, 1558.

³ Edsall, G, *et al*, *American Journal of Public Health*, 1954, **44**, 1537.

SIR,—Dr F W Sheffield and others (22 July, p 249, and 30 September, p 962) outline methods for the immunisation of selected adults against diphtheria. Protective antitoxin levels were found in 41 out of 60 adults before immunisation and 56 out of 60 after immunisation. We have been examining, similarly, antitoxin levels in random blood donors by a modified immunoelectro-osmophoresis and passive haemagglutination method,¹ using commercial, British Standard, and fourth British reference preparation for diphtheria antitoxin. Only 3% of adult donors