been slowly inserted, expelling the air gradually. When the time came for removal it was fitting the meatus closely and removal exerted considerable negative pressure. Perhaps "the champagne cork syndrome" would be a suitable name for the injury.

D McCracken

University Health Service,
University of Leeds

Direct translation

SIR,—In your leading article entitled "Beyond Calais" (11 September, p 606) you state that direct translation is often impossible and different nations often claim the same syndrome for their own compatriots. You give as an example hyperthyroidism, which is known in German as "Glutzaugenkrankheit." I have not met with this designation in my long years of medical practice. On the contrary the designation "Basedow-Erkrankung" is used by us, because C A von Basedow was a German doctor and had worked in Merseburg (1799-1854). Therefore we speak also of the "Merseburg triad"—namely, exophthalmos, struma, tachycardia.

H-D PACHE

Münchener Medizinische
Wochenschrift,
Munich

Oculo-mucocutaneous reactions to β -adrenoceptor antagonists

SIR,—Dr R G Finch (16 October, p 946) questions my statement (31 July, p 289) that "a validated case of oculo-mucocutaneous syndrome associated with practolol treatment has not yet been reported with any other beta-blocker," and quotes two supporting references.

No one denies that skin problems or eye symptoms may develop in patients receiving treatment with beta-blockers other than practolol.¹ However, proof of a "true-bill" oculo-mucocutaneous syndrome² similar to that associated with practolol has not been validated, so far as I am aware, in any patient on oxprenolol, or propranolol, at the time of writing.

J C PETRIE

Department of Therapeutics and
Clinical Pharmacology,
University of Aberdeen

¹ Petrie, J C, *et al*, *Postgraduate Medical Journal*, 1976, **52** (suppl 4), 63.

² Wright, P, *British Medical Journal*, 1975, **1**, 595.

Misuse of tubular elasticated bandages

SIR,—I read with interest Mr C V Ruckley's condemnation of the increasing use of untapered tubular elasticated bandages for everything from dressings to fractures (16 October, p 940).

This summer I injured my left little finger playing cricket, and subsequent radiography showed a fracture of the terminal phalanx. I was immediately expertly cared for by a colleague who applied a tubular elasticated bandage to hold a dressing in place. In spite of keeping my hand up to help reduce swelling an intense throbbing pain developed over the next three hours, and I could feel the dressing becoming tighter and tighter on my little

finger. Great relief was obtained by removing the tubular elasticated bandage and re-bandaging using the left ring finger as a splint.

Perhaps the use of tubular elasticated bandages with their inherent property of radial compression in the immediate management of finger injuries should be questioned and investigated further.

J B BOURKE

Department of Surgery,
University of Nottingham

In vitro lymphocyte reactivity during pregnancy

SIR,—Many in vitro studies on lymphocyte reactivity during pregnancy have been reported in your journal.¹⁻⁴ There is still some controversy about the part played by maternal serum blocking factors, because conflicting results have been obtained when lymphocytes were cultured in the presence or absence of autologous sera. We would like to report that even thoroughly washed lymphocytes have demonstrable serum proteins on their surfaces. It is possible that these adsorbed surface proteins, if they contain blocking factors, could modify in vitro lymphocyte reactivity in the absence of additional autologous sera.

Lymphocytes were obtained from defibrinated venous blood of pregnant and non-pregnant women as previously described.⁵ Crossed immunoelectrophoresis⁶ using an LKB Multiphor system was performed on: (1) freshly isolated lymphocytes; (2) lymphocytes which had been kept overnight in autologous serum at 37°C, room temperature, and 4°C; and (3) lymphocytes which had been kept in liquid nitrogen in a DMSO/autologous serum mixture. The cells were washed three times and six times before immunoelectrophoresis. The first dimension was performed with 20 microlitres of a 10⁷/ml concentration of lymphocytes for 35 minutes at 8-10 V/cm. Following this, viability of the cells was checked by removing the cells from the wells and staining with trypan blue. The second dimension was run overnight at 2 V/cm against 1% anti-whole human serum incorporated in the gel.

Precipitin arcs could be seen in all cases but were particularly numerous with lymphocytes which had been kept in autologous sera at 37°C, room temperature, 4°C, and in liquid nitrogen. Serum proteins were present in substantial quantities after three washes, which is the usual number employed in most experimental procedures, and traces were still demonstrable after six washes. This occurred with pregnant and nonpregnant lymphocytes. Most lymphocytes (average of 76%) were alive after the first dimension electrophoresis, as judged by trypan blue exclusion. This high viability count, together with the gradual disappearance of serum proteins with increasing number of washes, indicate that these proteins were from the lymphocyte surface rather than liberated from disrupted cells.

Inhibitory factors in pregnancy serum appear to be effective in very low concentrations⁷ so a small amount bound to the lymphocyte surface could be sufficient to alter its in vitro reactivity. It has also been suggested that the immunosuppressive activity of pregnancy serum globulins acts upon the earliest events of lymphocyte response at the time of antigen or mitogen recognition, possibly by competition for receptor sites,^{8,9} so the presence of these globulins on the

lymphocyte surface at the beginning of culture could bring about a depressed reactivity. Thus it would seem that serum blocking factors cannot entirely be excluded even in those experiments where lymphocytes were cultured without autologous sera, and the conflicting results reported could be influenced by the amount of serum proteins left on the lymphocyte surface.

Y W LOKE

S S BROOK

Department of Pathology,
University of Cambridge

G E ALLEN

Gresham Road Surgery,
Cambridge

¹ Curzon, P, *et al*, *British Medical Journal*, 1972, **4**, 49.

² Finn, R, *et al*, *British Medical Journal*, 1972, **3**, 150.

³ Walker, J S, *et al*, *British Medical Journal*, 1972, **3**, 469.

⁴ St Hill, C A, *et al*, *British Medical Journal*, 1973, **3**, 513.

⁵ Loke, Y W, *et al*, *American Journal of Obstetrics and Gynecology*, 1975, **122**, 561.

⁶ Weeke, B, *Scandinavian Journal of Immunology*, 1973, **2**, suppl 1, 47.

⁷ Gatti, R A, *et al*, *Clinical and Experimental Immunology*, 1973, **13**, 427.

⁸ Cooperband, S R, *et al*, *Transplantation Proceedings*, 1969, **1**, 516.

⁹ Yachnin, S, *Journal of Immunology*, 1972, **108**, 845.

Intravenous and aerosol salbutamol

SIR,—Drs M R Hetzel and T J H Clark (16 October, p 919), comparing intravenous and aerosol salbutamol, showed that aerosol salbutamol had a longer bronchodilatory effect. They suggest that this discrepancy may be due to the slow removal of salbutamol from the bronchial mucosa or its inhibition of mediator release. This difference may also be due to the differing metabolism of salbutamol when administered via these routes.

Studies by Evans *et al*¹ on the metabolism of salbutamol showed that salbutamol is conjugated in the liver and that this metabolite had negligible beta-adrenoreceptor stimulant activity. Following intravenous salbutamol 27% of the drug excreted in the urine over 24 h had been metabolised. However, when salbutamol was introduced directly into the lungs through a bronchoscope most of the dose in the plasma and urine was present as free salbutamol, suggesting that it is not metabolised in the lung. Their study also showed a more rapid urinary excretion rate for intravenous salbutamol, with 50% dose excretion by four h compared with only 25% dose excretion by four h for aerosol salbutamol. The longer bronchodilatory effect of aerosol salbutamol therefore reflects the higher proportion of active drug in the lungs and its slow removal from the lungs.

S P DEACON

London Road Hospital,
Boston, Lincs

¹ Evans, M E, *et al*, *Xenobiotica*, 1973, **3**, 113.

Can hepatitis B be transmitted sexually?

SIR,—If your expert seriously considers sexual contact to be an example of "non-parenteral" infection (28 August, p 517) he must surely be preoccupied with "unnatural practices." The opposite of "parenteral" is more concisely expressed as "enteral" than by the clumsy and ambiguous term "non-parenteral."^{1,2} Perhaps your expert was really thinking of inapparent parenteral transmission, for which, of course,