cardiac massage. The apparent resistance to large doses of isoprenaline noted in previous reports^{1 2} was observed. In two reported cases^{1 3} glucagon has been used successfully in the treatment of myocardial depression following beta blockade.

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¹ Kosinski, E J, *et al*, *New England Journal of Medicine*, 1971, **285**, 1325.

² Lagerfelt, J, and Matell, G, *Acta Medica Scandinavica*, 1976, **199**, 517.

³ Ward, D E, and Jones, B, *British Medical Journal*, 1976, **2**, 151.

Limulus test and meningitis

SIR,—The difficulties encountered in the diagnosis of partially treated pyogenic meningitis are stressed in your recent leading article (5 February, p 340). However, we do not agree with the statement that "the limulus test seems unlikely to be generally applicable."

A commercialised form of the limulus test (Mallinckrodt) has been applied to 43 patients with meningitis in our general hospital. The test remained negative (no false-positives) in Gram-positive meningitis (seven cases), tuberculous meningitis (three cases), and viral meningitis (eight cases). In contrast it appeared very helpful in the early diagnosis of Gram-negative meningitis. Twenty-five patients had Gram-negative meningitis, proved by culture or countercurrent immunoelectrophoresis, of whom 24 (97%) gave a positive response in the limulus test, although only 11 (42%) had a positive Gram stain. More particularly, four patients who had received antibiotics before admission had a negative Gram stain in contrast to the positive result of the limulus test.

Finally, from a practical point of view the limulus test appears easy to apply—the rapid procedure (about 15 min), early response (1–4 h), good sensitivity of the commercialised test, and relatively low cost are arguments in favour of its general application.

The practical advantages and the high efficiency of this method in the bacteriological diagnosis of pyogenic meningitis^{1 2} permit early institution of appropriate treatment.

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¹ Nachum, R, Lipsey, A, and Siegel, S E, *New England Journal of Medicine*, 1973, **289**, 931.

² Ross, S, *et al*, *Journal of the American Medical Association*, 1975, **233**, 1366.

Effect of thyrotrophin-releasing factor on lactation

SIR,—Thyrotrophin-releasing factor (TRF) is a potent stimulus for the release of prolactin in lactating women.¹ The rise in circulating prolactin induced by TRF has been said to be associated with breast engorgement and, by inference, with increased milk production.^{1 2} It therefore seemed that TRF might be useful to stimulate lactation in women with inadequate milk for their babies.

We studied six women to assess the rise in milk volume caused by TRF. All were healthy Black women living in Soweto, near Johannes-

burg, and were between two and six weeks post partum. Three were lactating successfully, while three were producing inadequate milk for the infant's needs. The study was approved by the university ethical committee and informed consent was obtained. The production of milk was measured by test-weighing before and after suckling, followed by complete expression. Measurements were carried out over a 24-h control period before the administration of TRF and for two feeds after TRF (that is, 6 h). The administration of TRF and the collection of blood samples for prolactin estimation were timed and carried out as described by Tyson *et al*¹ except that the dose of TRF was 200 µg. Prolactin was measured by radioimmunoassay.

There was a wide variation in basal plasma prolactin levels and in the response to suckling, but the prolactin level rose significantly in five of the six women after administration of TRF. The sixth was lactating very successfully and had high basal levels. There was no correlation between resting or stimulated prolactin levels and the success or failure of lactation. Milk production during the control period fluctuated very widely, as found by Hytten.³ In no case did the milk output rise in the two feeds following TRF administration. In five of the six women the post-TRF feed actually produced less milk than the feed immediately before TRF.

These results suggest that, while lactation is dependent on prolactin, milk production is not proportional to the plasma prolactin level. Other factors must be responsible for lactation failure. An effective galactagogue would be invaluable; however, our results suggest that evaluation of such agents must be based on actual measurements rather than subjective impressions such as breast engorgement.

We thank Professors Wayburne and Hansen for advice and Roche Ltd (South Africa) for supplies of TRF.

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¹ Tyson, J E, *et al*, *Journal of Clinical Endocrinology and Metabolism*, 1975, **40**, 764.

² Tyson, J E, Friesen, H G, and Anderson, M S, *Science*, 1972, **177**, 879.

³ Hytten, F E, *British Medical Journal*, 1954, **1**, 175.

IgA deficiency during penicillamine treatment

SIR,—The report by Dr O Hjalmarson and others (26 February, p 549) of a case of penicillamine-induced IgA deficiency appears to confirm the report of a case from Belgium.¹

After reading the Belgian report we decided to check the serum IgA levels in our own group of patients receiving penicillamine for Wilson's disease. We have currently three adult women with this diagnosis under treatment. Two of them are sisters (now aged 31 and 28 respectively) in whom the diagnosis was made in the autumn of 1975. The elder received penicillamine from February 1976 until she developed the nephrotic syndrome in October 1976. Her IgA level was normal (1.1 g/l (normal range 0.70–3.8 g/l) despite a urinary loss of around 3000 mg/24 h) when penicillamine was withdrawn. Her sister, who tolerated the drug for only six months before developing the nephrotic syndrome, also had

normal IgA levels when the penicillamine treatment was withdrawn (1.0 g/l; urinary albumin 4000 mg/24 h).

Our third patient, a woman now 28 years old whose Wilson's disease was diagnosed in 1970, has been treated with penicillamine and prednisolone (2.5 mg/24 h) since April 1970. IgA levels were followed during the first years of treatment and were on no occasion found to be low, ranging from 2.0 g/l in February 1971 to 1.6 g/l in September 1974. She remains well on 1.5 g of penicillamine daily (steroids were withdrawn early in 1976) and denies any increase in frequency of upper respiratory tract infections. Further, she successfully completed a normal pregnancy in January 1975, giving birth to a normal, healthy baby girl weighing 2780 g. We plan to report this case in full at a later date.

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¹ Proesmans, W, Jaeken, J, and Elckels, R, *Lancet*, 1976, **2**, 804.

Lingual polyp as cause of birth asphyxia

SIR,—Tumours of the mouth and neck occasionally cause birth asphyxia, but we have not been able to find any previous record of a lingual polyp causing respiratory obstruction at birth.

The baby, the third child of a healthy Nigerian mother, was born at term following a pregnancy complicated by moderate polyhydramnios from the 27th week onwards. He was delivered by forceps because of fetal distress, weighing 4020 g. He cried at birth but within a few seconds became cyanosed and appeared to be having respiratory difficulty. Inspection of the upper airways showed a pedunculated tumour arising from the base of the tongue in the midline and obstructing the pharynx like a ball-valve. The pedicle was ligated and the tumour excised immediately. The baby had no further difficulties. The tumour (see figure)

Section through lingual polyp ($\times 4.3$).

measured $18 \times 13 \times 13$ mm and consisted of smooth muscle bundles and fibrous tissue containing nerve bundles, blood vessels, lymphatics, adipose tissue, and glands and ducts similar to mucus-producing salivary glands. The nodule was covered with keratinising stratified squamous epithelium containing pilosebaceous follicles and one definite hair. There was no evidence of neoplasia.

The features of this tumour were similar to those described in hairy polyps arising on the