for the development of any neurotoxicity, and also that hyperbilirubinaemia is a contributory factor.

The controversy over absorption of HCP through the skin and central nervous system toxicology should be placed in proper perspective and, as pointed out in your leading article, "the reasonable conclusion seems to be that . . . the data on treatment and blood level in neonates are so heterogeneous that they should be taken only as a general indicator and that blood concentrations in animals are a poor guide to those in man."

After almost seven years of regulatory restriction one has yet to see any major deleterious effect when HCP is used in the recommended way. It is interesting to note that in Victoria, Australia, the Executive Committee of the National Health and Medical Research Council has recommended that HCP be removed from its restricted status and placed in a freely available category.

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¹ US Center for Disease Control, *Morbidity and Mortality*, vol 21, No 30, 29 July, 1972.

² Hyams, P J, et al, *American Journal of Diseases of Children*, 1975, **129**, 595.

³ Gowdy, J M, and Ulsamer, A G, *American Journal of Diseases of Children*, 1976, **130**, 247.

⁴ Plueckhahn, V D, and Collins, R B, *Medical Journal of Australia*, 1976, **1**, 815.

Brewer's yeast and diabetes

SIR,—With reference to your note about the insulin-potentiating effect of yeast extracts (Any Questions? 4 December, p 1366) I recently noticed an early paper by a Bristol physician, Dr W Bird Herepath, who claimed to have successfully treated a case of diabetes with yeast in 1853.¹

Before treatment the patient had been voiding urine of specific gravity 1.044 and containing 850 grains of sugar per pint. Within two days of employing brewer's yeast (given in a dose of one tablespoonful two to three times daily in milk) the specific gravity of the urine had sunk to 1.020 and the sugar to 300 grains per pint. After a further six weeks there was no glycosuria and the patient rapidly recovered his original weight.

Dr Herepath concluded that yeast worked by fermenting glucose in the stomach, thus preventing excessive absorption into the blood. In the light of modern thinking it seems likely that his patient's diabetes was caused by a depletion of chromium which was corrected by giving yeast.

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¹ Herepath, W B, *Journal of the Provincial Medical and Surgical Society*, 28 April 1854, p 374.

*Our expert writes: "It is interesting that the antidiabetic action of brewer's yeast was noted as long ago as 1853. Brewer's yeast is the richest natural source of "glucose tolerance factor," a trivalent chromium complex. American investigators have produced improvement of glucose tolerance in adult diabetics by adding chromic chloride to their diets.¹⁻³ The chromium complex formed from the inorganic salt exerts its hypoglycaemic action by potentiating the effects of insulin. Brewer's yeast, therefore, has no significant

therapeutic effect on the untreated, insulinopenic juvenile diabetic, although it may facilitate the response to exogenous insulin. Dr Herepath's patient was almost certainly a mild maturity-onset diabetic. The excellent response to yeast supplementation makes one wonder if he simultaneously recommended a reduction of carbohydrate intake. The importance of dietary restrictions in the treatment of diabetes was realised at the end of the 18th century and about the middle of the 19th century Bouchardat introduced the idea of fasting days for diabetics. This advance was well publicised and probably reached the ears of most practising physicians. The role of chromium in human diabetes is by no means resolved. There is no evidence that depletion actually causes diabetes; it may, however, aggravate pre-existent disease."—Ed, *BMJ*.

¹ Glinsmann, W H, and Mertz, W, *Metabolism*, 1966, **15**, 510.

² Levine, R A, Streeten, D H P, and Doisy, R J, *Metabolism*, 1968, **17**, 114.

³ Hopkins, L L, and Price, M G, *Proceedings of Western Hemisphere Nutrition Congress*, Puerto Rico, 1968, vol 2, 40.

Metabolic and cardiotoxic effects of salbutamol

SIR,—We have been following with interest recent publications on this subject but wish to refer specifically to one statement made by Dr A J Johnson and others (19 March, p 772). We fully endorse the view there expressed—namely, that salbutamol should normally be given by *slow* injection when the intravenous route is used. Our literature and packaging are clear on this point. Any suggestion that rapid intravenous injection may be generally acceptable is not of our making and is not supported by evidence available to us.

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Streptococcus mutans endocarditis—a trap for the unwary

SIR,—We would like to draw attention to the difficulties we have recently independently encountered in our respective laboratories in the recognition of *Streptococcus mutans*. In both instances the organism was initially discarded as a diphtheroid.

One patient, a man of 61, was admitted to the Whittington Hospital with a history of a flu-like illness, known congenital aortic valve disease, and features of congenital syphilis. He had been given co-trimoxazole by his general practitioner and four blood cultures on admission were sterile. Ten days later a "diphtheroid" was grown from a further blood culture and was initially dismissed as a contaminant. In view of the strong clinical possibility of endocarditis further blood cultures were then performed, all of which grew the "diphtheroid." Empirical treatment had already been started with penicillin and gentamicin when a culture of the "diphtheroid" in glucose broth revealed it to be a chaining streptococcus subsequently identified by the Streptococcus Reference Laboratory as *Str mutans*.

The second patient, a woman of 81, was admitted to St Thomas's Hospital with a history of increasing breathlessness and malaise and known mitral valve disease. Blood culture on admission grew a "diphtheroid" from one of two blood culture bottles which was dismissed as a contaminant. In view of continuing fevers and anaemia four further blood cultures were performed, from all of which

the "diphtheroid" was isolated. It was soon recognised as a streptococcus when Gram-stained films were made from broth cultures. The Streptococcus Reference Laboratory confirmed that it was *Str mutans*.

This non-haemolytic streptococcus is responsible for 14% of cases of streptococcal endocarditis¹ and is therefore likely to be only rarely encountered in any laboratory. It was originally described in 1924 by J K Clarke,² who recognised its morphological variation dependent on the pH of the medium. For those, like ourselves, previously unaware of this classic description of *Str mutans* we would suggest that no "diphtheroid" should ever be regarded as a contaminant in a blood culture until a Gram-stained film has been made from a broth culture.

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¹ Parker, M T, and Ball, L C, *Journal of Medical Microbiology*, 1976, **9**, 275.

² Clarke, J K, *British Journal of Experimental Pathology*, 1924, **5**, 141.

Cord blood IgM and suspected congenital rubella

SIR,—Professor J E Banatvala and Dr Jennifer M Best (22 January, p 228) and Dr J B Pattison (p 228) comment on our short report on "Importance of IgM determination in cord blood in cases of suspected rubella infection" (1 January, p 23). We concluded that in the second case reported, in which the maternal titre of rubella haemagglutination inhibition antibodies at the 8th week of pregnancy was 1/16 and at the 38th week >1/512, reinfection and viraemia had occurred with infection of the baby, proved by a high titre of rubella antibodies in the IgM fraction of the baby's cord blood. In both samples of this mother's serum we found only IgG rubella antibodies.

Professor Banatvala and Dr Best suggest the possibility of a primary infection in the 8th week of pregnancy. I think that this chance is minimal, for up to now we have always found IgM rubella antibodies in a primary infection; perhaps because we obtain a high concentration of the IgM fraction by using the Amicon filter. As for the rubella antibodies in the IgM fraction of the cord blood, they suggest a possible false-positive reaction by contamination of the IgM fraction through aggregated IgG. I do not think this is possible since heat inactivation was performed after fractionation. Moreover, we examined every fraction by the Mancini method to see whether contamination had occurred; not a trace of IgG could be detected in the IgM fraction with this method. Storage or bacterial contamination as causes of false-positive reactions can be excluded by the fact that fractionation was performed within 8 h after the withdrawal of blood. Contamination during fractionation was avoided by the use of sodium azide in the elution buffer.

Dr Pattison also suggests that a primary infection of the mother was possible, the rubella IgM antibodies having disappeared by the 38th week of gestation, and that the titre of 1/16 found at the 8th week of gestation could have been caused by non-specific inhibitors. As no α - or β -lipoproteins could be