detected by the Ouchterlony technique in the IgG fraction of the first serum sample of the mother with anti- α - and β -lipoprotein serum we still believe that this was a case of reinfection.

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Patient acceptability and drug effectiveness

SIR,—In your leading article (12 March, p 668) you state that the dangers of dextropropoxyphene overdosage have to be set against "its popularity based, presumably, on doctors' and patients' clinical impression of its effectiveness."

It is my impression, based on patients' remarks—and intuition—that the popularity of the most widely prescribed proprietary preparation containing the drug—namely, Distalgesic—is based primarily on its shape, size, and coating, which render it easy to swallow and tasteless. Many other analgesics, such as the once popular compound codeine tablets, are large, bitter, and granular when crushed and taken with water—that is, bulky, offensive, and troublesome in comparison with the small, smooth, rounded, oblong tablet with a non-powdery surface, easily slipped into the pocket or handbag as well as the mouth.

On two occasions I have been presented with two compound codeine tablets with the remark: "These have passed right through my body without disintegrating." This of course is due to overcompression in the manufacturing process by small, inexperienced firms and is probably rare. Such observations, however, highlight possibilities which do not occur to the average medical mind.

Patient acceptability in itself will often enhance the effectiveness of any drug or preparation and thus increase patient demand for it, especially when price does not concern them. It would be interesting to issue Distalgesic tablets without dextropropoxyphene.

T H H GREEN

Wallasey

Alcohol-induced Cushingoid syndrome: a false diagnosis

SIR,—Following the reports by Dr A G Smals and others (27 November, p 1298) and Dr A Paton (18 December, p 1504) a similar case presented to this hospital recently. Biochemical evidence of Cushing's syndrome was observed in a plethoric, obese alcoholic who in addition was mildly hypertensive and had glucose intolerance. The tests were carried out, however, while the patient was receiving spironolactone and this led to a false diagnosis. Several weeks later the tests were repeated with the patient no longer receiving spironolactone and were within the normal range.

A 57-year-old man with a long history of alcohol abuse presented with atrial fibrillation and congestive cardiac failure. In addition he was noted to be plethoric and moon-faced, with mild hypertension and glucose intolerance. He had evidence of mild hepatic dysfunction (raised prothrombin time and plasma bilirubin and gamma-glutamyl transpeptidase levels) although a liver biopsy was reported as being within normal limits. His congestive cardiac failure was treated with frusemide

40 mg, digoxin 0.2 mg, and spironolactone 50–100 mg daily. Two weeks after admission the plasma cortisol concentration at 8 am after an oral dose of 1 mg of dexamethasone given at midnight was $1.03 \mu\text{mol/l}$ ($37.3 \mu\text{g}/100 \text{ ml}$) (normal level after dexamethasone suppression $<0.10 \mu\text{mol/l}$ ($<3.6 \mu\text{g}/100 \text{ ml}$)). With the improvement of his heart failure spironolactone was discontinued and he was discharged. Four weeks later after a midnight dose of 1 mg of dexamethasone his plasma cortisol level at 8 am was $0.09 \mu\text{mol/l}$ ($3.3 \mu\text{g}/100 \text{ ml}$).

The fluorescence emission spectrum of spironolactone is identical with that of cortisol and this explains the abnormal results obtained while the patient was taking the drug.¹ In addition, all the previously reported cases of alcohol-induced Cushingoid syndrome improved after a short period of abstinence from alcohol, whereas in the above case the (false) diagnosis was made two weeks after admission to hospital. This case illustrates how important it is to check that concurrent therapy does not interfere with planned investigation. If this had been done in the above case time, money, and effort would have been saved.

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¹ Lunde, A O, *Journal of the American Medical Association*, 1970, **211**, 1851.

Metabolic effects of liquorice

SIR,—Dr M T Epstein and his colleagues speculate that small quantities of liquorice might cause hypokalaemia in certain susceptible subjects (19 February, p 488). They demonstrate the effects of liquorice ingestion in normal subjects and they warn against its use in patients at special risk—that is, those with hypertension and other circulatory disorders. They also state that a serious metabolic crisis is a possibility in some people.

I have previously described a patient who was admitted to this unit moribund, severely dehydrated, and areflexic, with flaccid paralysis.¹ Her plasma electrolytes at this time were: sodium 148 mmol (mEq)/l , potassium 1.2 mmol (mEq)/l , and bicarbonate 23 mmol (mEq)/l , and her blood urea concentration was 12 mmol/l ($72 \text{ mg}/100 \text{ ml}$). She had previously been given spironolactone and potassium supplements. Despite the very low plasma potassium concentration, she was not conserving urinary potassium. This woman also showed myoglobinuria, which has been previously reported in patients with severe hypokalaemia due to treatment with carbenoxolone.²

It was found that before admission she had been taking liquorice in the form of a mixture containing liquid extract of liquorice BP, liquid paraffin, and cascara in a dose of 5 ml three times weekly. This had not induced diarrhoea. She made a complete recovery following potassium repletion. She has subsequently been readmitted to the unit for detailed studies of the renin/angiotensin/aldosterone axis under controlled conditions. Measurements were made of the above substances and of plasma electrolytes before and after administration of a similar preparation of liquorice when on a fixed normal intake of sodium and potassium throughout. During the 10 days that she was given liquorice her plasma potassium level fell from 4.1 to 2.7 mmol (mEq)/l , while total body sodium

rose and total body potassium fell. Full details of this study will be reported at a later date. The findings clearly indicated that this woman was abnormally sensitive to liquorice.

Dr Epstein and his colleagues also found marked oedema in some of their subjects. This was not a feature in the case reported by me, either when she was acutely ill or during the later controlled study.

I am grateful to Dr K Boddy for the estimations of total body sodium and potassium.

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¹ Cumming, A M M, *Nursing Times*, 1976, **72**, 367.

² Mohamed, S D, Chapman, R S, and Crooks, J, *British Medical Journal*, 1966, **1**, 1581.

Suppressing lactation

SIR,—As a red-haired mother of one, who has had at least six months' experience of obstetrics, I can only agree with Drs D B Jelliffe and E F Patrice Jelliffe (12 March, p 714) that in your leading article (22 January, p 189) you were talking of an unrepresentative group of patients (mothers).

In my recent pregnancy I attended antenatal classes in which the participants were from mixed social backgrounds. Of a dozen or so, only one mother expressed a firm wish to bottle-feed; all others opted to breast-feed. Attitudes varied from extreme keenness to breast-feed to a willingness to give it a fair trial. No pressure was brought to bear on us by the instructors, although the advantages of each were outlined. It was emphasised that one should not feel a failure if breast-feeding was unsuccessful. Those mothers with whom I have since made contact are still breast-feeding.

I also had a chance to talk to the midwives while in hospital. They have noticed a tremendous upsurge in the popularity of breast-feeding, over recent months especially. One even suggested that economic factors are becoming increasingly important, as inflation affects the price of artificial milks as everything else.

I can only suggest that the opening paragraph of your article was written about five years ago, when I was doing obstetrics and bottle-feeding was more popular.

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Alcohol and the motorist

SIR,—Your leading article on alcohol and road accidents (12 March, p 665) is timely and informative, yet your conclusions and suggestions incorrect.

The facts are well known and need no further investigation. Alcohol reduces the mental and physical ability of every motorist and there can be no safe level. Surely it is time the medical profession came out clearly and unequivocally in favour of the Blennerhassett Committee's advice "Don't drink and drive" and supported politicians to bring in the only logical law—no level of alcohol in the motorist.

This, if enforced, would solve most of the problems in connection with alcohol and