Thyroid dysfunction and affective illness

SIR,—Professor E Szabadi's editorial on thyroid dysfunction and affective illness raised the question of whether subclinical dysfunction of the thyroid has a role in affective illnesses.¹ Lithium is well recognised to affect thyroid hormones; cases of hypothyroidism and hyperthyroidism have been reported.^{2,3} Primary affective disorders may themselves have a role in the development of autoimmune thyroid disease.

A recent study was designed to determine whether the development of autoimmune thyroid disease was due to lithium treatment or depressive illness.⁴ Three main groups were studied: a control group, patients receiving treatment other than lithium for depression, and patients receiving lithium for their manic depressive illness. All patients were clinically and biochemically euthyroid. The serum samples obtained were tested for thyroid microsomal and thyroglobulin antibodies. None of the controls were positive for thyroid antibodies, but 7.5% of the patients receiving treatment other than lithium for depression were positive for the antibodies. This, however, is not significantly high when compared with the incidence in the general population.⁵ The patients receiving lithium for their manic depressive illness showed a greatly increased incidence of thyroid antibodies (20%), in keeping with results of other studies.^{1,6}

The authors concluded that lithium treatment induces the formation of antibodies in susceptible people, which may lead to the development of thyroid disease in these patients. A larger study will be necessary to determine whether affective disorders alone play any part in the development of subclinical hypothyroidism.

JOHN BOLO-DEOKU
HERVEY WILCOX

Department of Chemical Pathology and
Metabolism,
St Helier Hospital,
Carshalton,
Surrey SM5 1AA

- 1 Szabadi E. Thyroid dysfunction and affective illness. *BMJ* 1991;302:923-4. (20 April.)
- 2 Thompson CJ, Bayliss PH. Asymptomatic Graves disease during lithium therapy. *Postgrad Med J* 1968;62:295-6.
- 3 Lazarus JH, Bennie JR, Chalmers RJ, Crockett G. Lithium therapy and thyroid function: a long term study. *Psychol Med* 1981;11:85-92.
- 4 Wilson R, McKillop JH, Crockett GT, *et al*. The effect of lithium therapy on parameters thought to be involved in the development of autoimmune thyroid disease. *Clin Endocrinol* 1991;34:357-61.
- 5 Tunbridge WMG, Evered DC, Hall R, *et al*. The spectrum of thyroid disease in a community. The Wickham survey. *Clin Endocrinol* 1977;7:481-93.
- 6 Emerson CH, Dyson WL, Utiger R. Serum thyrotropin and thyroxine concentrations in patients receiving lithium carbonate. *J Clin Endocrinol Metab* 1973;36:338-46.

SIR,—Further to the editorial by Professor E Szabadi,¹ we would like to draw attention to a link between autoimmune thyroid dysfunction in the postpartum period and depressed mood.

Thyroid dysfunction of a transient nature has been shown by Amino *et al* to be common in women with thyroid microsomal antibodies and thyroglobulin in the postpartum year.^{2,3} About 12% of most populations studied are positive for antibodies, so this constitutes a considerable risk factor.⁴

We have already shown a weak but significant link between thyroid dysfunction and depression at six weeks post partum,⁵ and a recently completed study confirms that postpartum autoimmune thyroid dysfunction is an important contributory factor to postpartum mood disorder.

We administered the 30 item general health questionnaire six weeks post partum to 145 women positive for antibodies and 229 women negative for antibodies. Of these, 110 women positive for

antibodies and 133 controls were given a variety of depression scales at eight, 12, 20, and 28 weeks post partum. Thyroid state was assessed clinically and blood sampled for thyroid antibodies, free triiodothyronine, free thyroxine, and thyroid stimulating hormone. Hyperthyroidism was defined as raised free triiodothyronine or free thyroxine and suppressed thyroid stimulating hormone (<0.2 mU/l) or raised free triiodothyronine and free thyroxine and normal thyroid stimulating hormone; hypothyroidism was defined as raised thyroid stimulating hormone and lowered free triiodothyronine or free thyroxine or raised thyroid stimulating hormone (>10 mU/l) even if free triiodothyronine and free thyroxine were normal.

A greater proportion of women positive for antibodies were rated as depressed on the general health questionnaire at six weeks (62 of 145 compared with 65 of 229 controls, $p<0.001$ with χ^2 test). At this time antibody concentrations are rising rapidly even though plasma concentrations of hormones may be normal.

Women positive for antibodies and with thyroid dysfunction had significantly higher scores on the 17 item Hamilton depression rating scale ($p<0.001$), the Edinburgh postnatal depression scale ($p<0.01$), and the depression subscale of the hospital anxiety and depression scale ($p<0.001$) compared with the women negative for antibodies. According to research diagnostic criteria there was no excess of major depression in women positive for antibodies, but overall there was an excess of depression ranging from probably minor to definite major ($p<0.01$).

Such women may need thyroxine for hypothyroid states and β blockers for symptomatic hyperthyroidism. Most will return to a normal thyroid state by the end of the first postpartum year, though hypothyroidism may be permanent in a minority.

BRIAN HARRIS
DAVID W PHILLIPS
SEKINAH OTHMAN
REGINALD H HALL
JOHN H LAZARUS

Departments of Psychological Medicine and
Medicine,
University of Wales College of Medicine,
Cardiff CF4 4XN

COLIN J RICHARDS

Department of Obstetrics and Gynaecology,
Caerphilly District Miner's Hospital,
Caerphilly,
Mid Glamorgan CF8 2WW

- 1 Szabadi E. Thyroid dysfunction and affective illness. *BMJ* 1991;302:923-4.
- 2 Amino N, Miyai K, Onishi T, *et al*. Transient hypothyroidism after delivery in autoimmune thyroiditis. *J Clin Endocrinol Metab* 1976;42:296-301.
- 3 Amino N, Mori H, Iwatani Y, *et al*. High prevalence of transient post partum thyrotoxicosis and hypothyroidism. *N Engl J Med* 1982;306:849-52.
- 4 Prentice LM, Phillips DIW, Sarsero D, Beever K, McLachlan SM, Smith BR. Geographical distribution of subclinical autoimmune thyroid disease in Britain: a study using highly sensitive direct assays for autoantibodies to thyroglobulin and thyroid peroxidase. *Acta Endocrinol* 1990;123:493-8.
- 5 Harris B, Fung H, Johns S, *et al*. Transient post partum thyroid dysfunction and post natal depression. *J Affect Disord* 1989;17:243-9.
- 6 Goldberg DM. *The detection of psychiatric illness by questionnaire*. Oxford: Oxford University Press, 1972. (Maudsleys monographs, 21.)

Lack of cyanosis in cyanotic congenital heart disease

SIR,—We were impressed by the improved accuracy of diagnosis of congenital heart disease when the algorithm presented in the paper by Dr Rodney C G Franklin and colleagues was used.¹ We question, however, the use of cyanosis alone without the hyperoxia test as part of the early decision making in the algorithm as cyanosis is a variable feature in neonates with cyanotic heart disease.²

We have reviewed 24 babies with proved cyanotic

heart disease from our district maternity units. Seven were thought not to be cyanosed on the first day of life, and four remained acyanotic on the fourth day, by which time most babies would have been discharged home. Many people use a threshold of at least 20 kPa in the hyperoxia test to exclude cyanotic lesions.¹ Of our 24 babies, 17 had a hyperoxia test and seven had an oxygen tension >12 kPa. One of five cyanosed babies with an inferior axis in whom Ebstein's anomaly had been excluded had an oxygen tension of 17 kPa: he had critical pulmonary stenosis and a patent ductus arteriosus.

We wonder whether the algorithm could be improved by using the hyperoxia test with a higher threshold to detect cyanotic heart disease early in the algorithm.

DAVID LACY
MICHAEL WATKINSON

Solihull Hospital,
Solihull,
West Midlands B91 2JL

- 1 Franklin RCG, Spiegelhalter DJ, Macartney F, Bull K. Evaluation of a diagnostic algorithm for heart disease in neonates. *BMJ* 1991;302:935-9. (20 April.)
- 2 Shinebourne EA, Anderson RH, Bowyer JJ. Variations in clinical presentation of Fallot's tetralogy in infancy. *Arch Dis Child* 1975;37:946-55.
- 3 Warburton D, Rehan M, Shinebourne EA. Selective criteria for differential diagnosis of infants with symptoms of congenital heart disease. *Arch Dis Child* 1981;56:94-100.

AUTHORS' REPLY,—We agree with Drs Lacy and Watkinson that many neonates with cyanotic congenital disease do not look blue; accordingly, several types of cyanotic congenital heart disease are diagnosed by our algorithm without the patient passing the "main problem=cyanosis" branch point.

For those who did go down the "main problem=cyanosis" branch we experimented with different thresholds for upper body oxygen tension (8-20 kPa) reached when the patients were given 100% oxygen to breathe. The value that brought the greatest number of patients to the correct diagnosis was 12 kPa. We do not think it surprising that this is lower than the value used to discriminate cardiac from pulmonary disease in a more general neonatal population, where the "costs" of false positive or false negative results are different.

As we emphasised, the algorithm has evolved and been validated as a diagnostic tool to aid a paediatric cardiologist with an over the telephone referral of a neonate with suspected congenital heart disease, not for a neonatologist trying to discriminate cardiac from pulmonary disease—the more usual context in which the hyperoxic test is used.

RODNEY FRANKLIN
FERGUS MACARTNEY
KATE BULL

Hospital for Sick Children,
Great Ormond Street,
London WC1N 3JH

DAVID SPIEGELHALTER

MRC Biostatistics Unit,
Cambridge CB2 2BW

Postoperative urinary retention in men

SIR,—Mr P H O'Reilly's editorial¹ and Dr R Wight and colleagues' subsequent letter on postoperative urinary retention in men² cannot be allowed to go unchallenged. Although we agree with Mr O'Reilly that better management of retention may follow a better understanding of the physiology and pathophysiology of micturition, a better understanding of the pharmacology is also essential. Atropine and other reversal agents may not be the culprits as often as is suggested. Virtanen *et al* found that atropine has a half life of 10 hours with a standard deviation of over seven