plasma cholesterol levels exceeding 8 mmol/l, whereas seven were shown in the Table. There was an incidence of 12% impaired thyroid function for subjects with a plasma cholesterol over 8 mmol/l. These impressive figures compare with no cases of hypothyroidism amongst 90 matched controls with plasma cholesterol levels under 4.0 mmol/l and no cases of hypothyroidism amongst 25 controls of which one had sub-clinical hypothyroidism with a serum cholesterol of 6.8 mmol/l. The authors use the unsatisfactory term 'asymptomatic' hypothyroidism in their title, as have other workers[1]. The presence or absence of symptoms in both hypothyroidism and sub-clinical hypothyroidism depends more on the clinician than the patient.

The effect of thyroxine on the lipid profile differs in various studies[1,2]. Although some workers[3] have not found a significant increase of serum cholesterol in sub-clinical hypothyroidism and some have not found a significant fall of serum cholesterol on L-thyroxine in sub-clinical hypothyroidism[4], a meta-analysis might show significant changes.

Since a correlation between serum TSH and cholesterol was first found within the normal range of TSH[5], Elder and his colleagues[6] have shown that 10% of subjects with a TSH between 1.1 and 3.0 mU/l have a plasma cholesterol >7.5 mmol/l, whereas 20% of those with a TSH between 3.1 and 5.0 mU/l have a plasma cholesterol >7.5 mmol/l.

At the top end of the reference range for plasma cholesterol levels, there is about five times the risk of getting a myocardial infarction compared with those at the lower end. If L-thyroxine can shift the whole distribution curve of plasma cholesterol to the left, this could be an important preventive strategy for coronary artery disease.

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References

1 O'Kane MJ, Neely RDG, Trimble ER, Nicholls DP. The incidence of asymptomatic hypothyroidism in new referrals to a hospital lipid clinic. *Ann Clin Biochem* 1991;**28**:509-11
2 Powell JT, Wiseman SA, Carter G, *et al.* Can thyroxine halt the progression of peripheral arterial disease? *Eur J Vasc Surg* 1989;**3**:85-7
3 Nilsson G, Nordlander S, Levin K. Studies on subclinical hypothyroidism with special reference to the serum lipid pattern. *Acta Med Scand* 1976;**200**:63-7
4 Cooper DS, Halpern R, Wood LC, *et al.* L-thyroxine therapy in subclinical hypothyroidism. *Ann Int Med* 1984;**101**:18-24
5 Powell J, Alaghband Zadeh J, Carter G, *et al.* Raised serum thyrotrophin in women with peripheral arterial disease. *Br J Surg* 1987;**74**:1139-41
6 Elder J, McLelland A, O'Reilly DStJ, *et al.* The relationship between serum cholesterol and serum thyrotropin, thyroxine and tri-iodothyronine concentrations in suspected hypothyroidism. *Ann Clin Biochem* 1990; **27**:110-13

Dr Henry Harington

Dr Sakula has commented kindly (November 1991 *JRSM*, p 698) on my recent paper on Dr Henry Harington[1] and on my suggestion that he may have looked after John Hunter on his *early* visits to Bath. There has never been any doubt about Parry looking after Hunter, but many writers have been confused.

When Hunter first came to Bath Parry was still a student! However, in 1785 when Hunter made his fourth and last visit to Bath, he was looked after by the young, 29 year old, as yet unknown, Caleb Parry. This remarkable fact can only be explained by Jenner's close friendship with both Hunter and Parry, and his high opinion of his young friend.

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Reference

1 In: Rolls R, Guy J, Guy JR, eds. *A Pox on the Provinces.* Bath: Bath University Press, 1990

Successful treatment of neonatal purpura fulminans with epoprostenol

We read with interest the experience of Stewart and colleagues reversing neonatal purpura complicating septicaemia with epoprostenol (prostacyclin) (October 1991 *JRSM*, p 623). Perhaps this will help to validate the use of epoprostenol, despite its cost, in neonatal septicaemia particularly when its other beneficial effects are considered. We report another infant successfully treated with epoprostenol. We have treated a 36 week gestation infant receiving intensive care, including ventilatory support, for Group B haemolytic streptococcal septicaemia with epoprostenol. As a result of the septicaemia, the infant suffered refractory hypoxia due to pulmonary hypertension, marked acidosis and purpura with abnormal coagulation profiles. Epoprostenol was commenced at 5 ng/kg/min increasing to 15 ng/kg/min which was maintained for the next 48 h. There was a rapid improvement in oxygenation and acid-base balance. This improvement was prostacyclin-dependent as reversible deterioration occurred when the infusion was accidentally interrupted.

Coincidentally the coalescing purpura began to regress. Neither hypotension nor haemorrhage was encountered. The infant made a full recovery.

Pulmonary hypertension associated with septicaemia is believed to be partially mediated by the cyclo oxygenase product thromboxane A_2[1] whose actions are antagonized by prostacyclin. Some studies have suggested some benefit from prostacyclin therapy[2]. Whilst epoprostenol needs careful comparison with tolazoline, the usual anti-pulmonary hypertension therapy, other features favouring epoprostenol action in septicaemia include the prevention of thromboxane-induced platelet aggregation which is implicated in the pathophysiology of septic purpura and shock. Whilst financial constraints must be considered, the multiple actions of epoprostenol suggest a role in neonatal septicaemia particularly complicated by pulmonary hypertension or purpura and underline the need for controlled trials in comparison with the cheaper and more widely-used tolazoline.

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References

1 Abu-Osba YK. Treatment of persistent pulmonary hypertension of the neonate: an update. *Arch Dis Child* 1991;**66**:74-7
2 Kaapa P, Koivisto M, Ylikorkak O, Konvalainen K. Prostacyclin in the treatment of neonatal pulmonary hypertension. *J Pediatr* 1985;**107**:951-3