differences show that delivery by lower segment caesarean section is more dangerous when the fetus presents by the breech, even in the best possible circumstances. The greater likelihood of a low Apgar score might be due to an inherent abnormality which in itself had caused the breech presentation but perhaps is more likely to be due to respiratory efforts before delivery of the head. A mature, healthy fetus is probably well able to withstand this extra insult, but for a breech-presenting fetus which becomes distressed during preterm labour the added hazard might be important. If the hypothesis that delay in delivery of the fetal head causes the depression is correct then this is another reason to consider delivery by classical caesarean section in cases of breech presentation where the fetus is already compromised.

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Effects of oral propranolol on parathyroid hormone secretion in chronic renal failure

Parathyroid hormone secretion is stimulated by beta-adrenergic agents¹ and this effect is blocked by propranolol.² Acute infusion of propranolol in uraemic patients has been shown to inhibit secretion of parathyroid hormone,³ and a retrospective study has suggested that treatment with beta-blockers may protect against secondary hyperparathyroidism in haemodialysis patients.⁴ We report a prospective study designed to assess the long-term effects of oral propranolol on plasma parathyroid hormone concentrations in haemodialysis patients.

Patients, methods, and results

We studied four male and five female haemodialysis patients from 12 to 68 years. None of them were taking vitamin D compounds and all were taking aluminium hydroxide preparations. Their fasting plasma concentrations of calcium, phosphate, and immunoreactive parathyroid hormone were measured and they were then treated with propranolol by mouth. Seven received propranolol 40 mg twice daily and the remaining two (both weighing under 45 kg) 20 mg twice daily. Isoprenaline infusion before treatment (0.75-1.0 µg/min) caused a significant increase in mean (±SD) pulse rate of from 79±10 to 96±15 beats/min ($p<0.01$). Repeat isoprenaline infusion during treatment caused little change in pulse rate (68±13 to 70±14). Fasting concentrations were again measured after six and 12 weeks' treatment. All blood samples were taken 48 hours after dialysis. Plasma calcium and phosphate were measured by standard methods and plasma immunoreactive parathyroid hormone by a previously described method.⁵ Statistical analysis was by the Wilcoxon matched pairs rank sum test.

Before treatment one patient was hypocalcaemic, four were normocalcaemic, and four were hypercalcaemic (range 1.68-3.1 mmol/l (6.72-12.4 mg/100 ml), normal range 2.2-2.63 mmol/l (8.8-10.52 mg/100 ml)). Eight patients were hyperphosphataemic (range 1.23-2.36 mmol/l (3.91-7.5 mg/100 ml), normal range 0.8-1.4 mmol/l (2.5-4.5 mg/100 ml)). Seven patients had raised concentrations of immunoreactive parathyroid hormone (range 410-4000 ng/l, normal range 250-950 ng/l). The figure shows the effects of treatment on plasma calcium, phosphate, and immunoreactive

parathyroid hormone concentrations expressed as a percentage of the initial value. There was no significant change in mean plasma calcium throughout treatment. The mean plasma phosphate concentration rose and was significantly higher than baseline values at six weeks ($p<0.05$). Plasma concentrations of immunoreactive parathyroid hormone were below baseline values in seven out of nine patients at six weeks and in five out of six patients at 12 weeks. There was a mean fall in plasma immunoreactive parathyroid hormone at six weeks of 7.8% ($p<0.05$) and at 12 weeks of 14.7%. There was no correlation between changes in plasma calcium or phosphate and changes in plasma immunoreactive parathyroid hormone.

Changes in plasma concentrations of calcium, phosphate, and immunoreactive parathyroid hormone in response to treatment with oral propranolol. Vertical bars indicate SEM. Asterisk indicates $p<0.05$.

Comment

The beta-adrenergic-blocking activity achieved by the doses of propranolol given was significant as judged by the abolition of isoprenaline-induced rises in pulse rate. Plasma concentrations of immunoreactive parathyroid hormone fell during treatment, and this may indicate a direct effect of propranolol on beta-adrenergic receptors in parathyroid tissue.^{1,2} The degree of parathyroid gland suppression during acute propranolol infusion in uraemic patients was much greater³ than that observed in this study. The differences may be accounted for by the significant rise in plasma phosphate concentrations which also occurred during this study. In normal circumstances this would lower plasma ionised calcium concentrations, which would tend to stimulate parathyroid hormone secretion. Hence the low plasma concentrations of immunoreactive parathyroid hormone seen may underestimate the actual degree of parathyroid hormone suppression achieved by propranolol. There were, however, only small reductions in concentrations of immunoreactive parathyroid hormone after three months' treatment, and we doubt whether propranolol has a role in the treatment of established hyperparathyroid bone disease in chronic renal failure.

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³ Coevoet B, Desplan C, Sebert JL, et al. Effect of propranolol and metoprolol on parathyroid hormone and calcitonin secretions in uraemic patients. *Br Med J* 1980;280:1344-6.

⁴ Caro JF, Besarab A, Burke JF, Glemon JA. A possible role for propranolol in the treatment of renal osteodystrophy. *Lancet* 1978;ii:451-3.

⁵ Farrington K, Varghese Z, Moorhead JF. Human calcitonin in the treatment of renal osteodystrophy. *J Lab Clin Med* 1980;96:399-406.

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