

parathyroidism.<sup>2</sup> But iPTH (carboxy-terminal) concentrations are normal in the patients.

The data leads us to propose that bone in these patients is more sensitive to PTH as are kidneys as shown by Heath's demonstration of raised urinary cyclic AMP/iPTH ratios.<sup>3</sup> Increased sensitivity in more than one PTH target organ can be accounted for by proposing an abnormality of either PTH or its receptors. There is no evidence for the former in familial hypocalcaemic hypercalcaemia.<sup>4</sup> Adenylate cyclase is involved in the control of PTH secretion by parathyroid glands<sup>5</sup> and is part of the complex regulating its action on target cells.<sup>6</sup> Our hypothesis is that genetically altered adenylate cyclase modifies the bone and kidney response but does not suppress PTH secretion in response to hypercalcaemia. In such a way familial hypocalcaemic hypercalcaemia would be a true pseudo-hyperparathyroidism, the opposite pattern of Albright's syndrome.<sup>7</sup> More studies are needed to investigate this hypothesis.

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#### Mean platelet volume changes in infection

Van der Lelie and van dem Borne report an increase in mean platelet volume (MPV) during episodes of septicaemia.<sup>1</sup> We believe that another pattern of change in platelet size may occur during infection. In a recent survey of 372 cases of thrombocytosis<sup>2</sup> we studied the temporal relationship between MPV and platelet count in 10 patients with marked episodes of thrombocytosis due to bacterial infection. All measurements were made on a Coulter Counter Model S Plus. In all 10 patients MPV fell as the platelet count rose. The peak platelet count was  $212 \pm 63\%$  (mean  $\pm$  SD) of baseline platelet count and the minimum MPV was  $80.2 \pm 5.1\%$  (mean  $\pm$  SD) of baseline MPV. On average the peak platelet count occurred 10 days after the onset of infection and the lowest MPV was seen one day after the peak platelet count. We conclude that at least two patterns of platelet size changes are possible in response to infection: an early rise in MPV seen in severe infection such as septicaemia (this may be associated with thrombocytopenia) and a later fall in MPV seen in patients with sustained bacterial infection and associated with thrombocytosis. It is possible that the early rise in MPV is due to rapid release of large platelets from the spleen<sup>3</sup> rather than shape changes occurring in the circulation. The cause of the fall in MPV in reactive thrombocytosis remains enigmatic, but does not relate to platelet age.

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#### Bacteriuria in patients undergoing prostatectomy

Dr Williams and Dr Hole<sup>1</sup> state that a knowledge of the prevalence of different bacteria helps in the choice of a suitable chemoprophylactic agent. We agree, but believe that there are more important considerations.

Septicaemia, much the most dangerous complication of infection in these patients, is almost entirely confined to patients with preoperative bacteriuria and to those who develop bacteriuria before removal of the catheter after operation.<sup>2-4</sup> Because of the variability of the infecting bacteria, and of their sensitivity, no predetermined drug can be relied upon to give certain protection against septicaemia—as can be seen from Table 3 of Dr William's and Dr Hole's paper.<sup>1</sup> The only reliable method is to select an appropriate systemic antibiotic from the results of a urine culture and direct sensitivity test put up one or two days before operation, so that the results are available in the ward before the patient goes for operation. If the urine is infected, an appropriate systemic antibiotic is administered at or before premedication. Occasionally, two drugs are required in some patients with mixed infections. Since septicaemia is sometimes provoked by catheter removal, antibiotic administration should be extended, or repeated, to cover this event.<sup>4</sup> Experience in Bristol and in Dublin shows that this system is quite practicable and gives reliable protection against septicaemia.<sup>4</sup>

If the preoperative urine is not infected, we believe it is, on balance, better to avoid using systemic antibiotics prophylactically, because of the risk of, sooner or later, promoting infection by resistant organisms,<sup>5-7</sup> and because postoperative infection is less dangerous than preoperative, and is usually easy to treat after a successful operation. However, we believe that prophylaxis by means of nitrofurantoin may be justified in these patients, since it is not systematically active. In a recent pros-