VASODEPRESSIVE EFFECT OF PARATHYROID HORMONE IN POST-TRANSPLANT BLOOD PRESSURE REGULATION

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Persistent hyperparathyroidism (HPTH) and its attendant hypercalcaemia has been implicated as an aetiologic factor in post-transplant hypertension [1]. These studies further define the role of PTH and hypercalcaemia in blood pressure regulation following successful transplantation.

Methods
Blood pressure, ionised calcium, PTH and PRA were measured during a 4-hour

Figure 1. Correlation of $\% \Delta$ parathormone and $\% \Delta$ systolic BP during calcium infusion in transplant recipients
Ca\textsuperscript{++} infusion (15mg/kg) in seven normal subjects and thirteen successful transplant recipients with hyperparathyroidism.

**Results**

For both the transplant recipients (Figure 1) and the normals (Figure 2) there was a significant negative correlation of the relative change (\%Δ) in PTH with \%Δ systolic blood pressure at all sampling intervals during the Ca\textsuperscript{++} infusion. In the transplant subjects, the \%Δ Ca\textsuperscript{++} was also correlated (p < 0.01) with a \%Δ SBP. No such relationship existed in the normal subjects. No relationship existed between PRA and either Ca\textsuperscript{++} or systolic blood pressure.

**Discussion**

We conclude that (1) endogenous PTH contributes to blood pressure regulation in both hyperparathyroid transplant recipients and normal subjects, acting as a vasodepressor, (2) HPTH alone does not increase blood pressure, and (3) the blood pressure response of normal and HPTH subjects to hypercalcaemia is dependent, in part, upon the degree of endogenous PTH suppression and not necessarily the increase in serum Ca\textsuperscript{++}.

**Reference**