Perturbations in M_A were caused more readily by changes in K_{AA} than in PR_A. In five, M_A rose 35, 50, 105, 154 and 426%; corresponding falls in K_{AA} were 11, 30, 43, 55 and 67% while only one showed a rise in PR_A of over 20%. In two, M_A fell 20 and 14% (despite rise in PR_A in the latter of 336%) because K_{AA} rose 42 and 116%. We conclude that while hypertriglyceridaemia in general reflects both raised production and lowered removal, acute changes in pool size (VLDL) are more closely related to removal.

For the whole group removal rate was inversely related to body weight.

The practical implications are that increased production (e.g. from excess calories, ethanol etc.) need not cause HTG if removal rates also increase. Factors that improve removal are therefore potentially beneficial and include lowering body weight (as shown above) and clofibrate which stimulates VLDL catabolism.

CORTISOL AS A DETERMINANT OF CARDIAC GLUCOSE METABOLISM IN VIVO 17

S.P. LIM, M.L. WAHLQVIST, E.A. SHANAHAN and K. OUTCH

Departments of Medicine & Surgery, Monash University, Prince Henry's Hospital, Melbourne and Section of Human Nutrition, Deakin University, Geelong

There is current interest in the possibility that administration of glucocorticoid may limit infarct size. Such an approach could well have important implications for cardiac metabolism. Although the metabolic effects of glucorticoid have been examined in the isolated heart, little is known about how physiological concentrations of glucocorticoid relate to cardiac metabolism in vivo.

Healthy conscious mongrel dogs with catheters implanted in the aorta and the coronary sinus for assessment of arterial-coronary sinus differences (a-cs) and in a central vein for infusion of glucose were studied.

Arterial glucose concentration (Ca glucose) was not significantly correlated with Ca-cs glucose ($r=0.23$, $n=50$). The range of blood glucose values was 3.7 to 74.4 mmol/l (mean ± sem, 13.9 ± 2.2). Cortisol immunoreactivity in arterial plasma (Ia cortisol) was significantly and negatively correlated with Ca-cs glucose ($r=0.37$, $n=50$, $P<0.01$). The range of plasma cortisol values was 13 to 438 nmol/l (mean ± sem, 93 ± 13). A multiple regression equation to predict Ca-cs glucose from both Ca glucose and Ia cortisol as independent variables had an F-ratio of 6.85 significant at the 1% level.

Ca-cs glucose (μmol/l)	=	294+0.78	Ca glucose (μmol/l)	+	1.52 Ia cortisol (nmol/l)

Of particular interest was the finding that the heart took up and released cortisol in relation to Ia cortisol ($r=0.75$, $n=50$, $P<0.001$). The range of Ia-cs cortisol was -31 to 138 nmol/l (mean ± sem, 12 ± 4). If it is assumed that dog coronary blood flow is 150 ml/min, for the mean Ia-cs, the heart would take up about 1 nmol cortisol/min. The Ca-cs glucose was negatively correlated with Ia-cs cortisol ($r=0.43$, $n=50$, $P<0.01$). The question therefore arises as to whether Ia cortisol or Ia-cs cortisol, or both, are determinants of Ca-cs glucose. By multiple regression analysis.

Ca-cs glucose (μmol)	=	331	−0.2	Ia cortisol (nmol/l)	−4.3	Ia-cs cortisol (nmol/l)

(F ratio = 5.05, P = 0.01)

The predictive value of this equation is actually less, judged by the F ratio, than for either of the univariate regression analyses, Ca-cs glucose/Ia cortisol or Ca-cs glucose/Ia-cs cortisol.

Thus it may be that Ia cortisol and Ia-cs cortisol reflect different aspects of the regulation of cardiac glucose metabolism. Specific radioimmunoassay for cortisol in these studies may account for the recognition of direct relationships between Ca-cs glucose and Ia cortisol or Ia-cs cortisol, not recognized in previous human studies in which a fluorometric method for glucocorticoid was used.

EFFECT OF HAEMODIALYSIS ON LEFT VENTRICULAR FUNCTION IN END-STAGE RENAL FAILURE 18

J. HUNG, P. HARRIS, R. UREN, D.T. KELLY and D. TILLER

Hallstrom Institute of Cardiology, Nuclear Medicine and Nephrology Department, Royal Prince Alfred Hospital, Sydney

End stage renal failure is often associated with clinical evidence of left Ventricular dysfunction and improvement following haemodialysis is uncertain. Left ventricular function was measured before and after dialysis in patients with chronic renal failure using gated cardiac blood pool imaging (GBPS).

In 19 patients (8 male, 11 female) aged 17-63 years (mean 42) left ventricular ejection fraction (EF) derived from GBPS was measured one day before dialysis (pre-EF) and within 2 hours following routine dialysis (post-EF).

Patients were divided into two groups — Group A: pre-EF>50% (15 patients) and Group B: preEF<50% (4 patients). All 4 patients in Group B and 6 patients in Group A had cardiomegaly and episodes of circulatory congestion. Mean pre-EF in Group B was significantly lower than in Group A (30% vs 6).

After dialysis there was a mean weight loss of 2.2Kg, an average mean BP fall of 19mmHg and heart rate did not change significantly. No difference was noted between Groups A and B.

	No. = 15	
GROUP A	Pre-EF (%) 63 (52-71) 62	Post-EF (%) (53-79) NS

	No. = 4	
GROUP B	Pre-EF (%) 30 (26-36)	Post-EF (%) 44 (31-35) p < 0.01

In Group A there was no significant change in EF with dialysis. In Group B all 4 patients showed a rise in