Renal artery stenosis in conjunction with contralateral nephrectomy rapidly and predictably creates a stable hypertension. The renin-angiotensin system is initially activated but renin levels soon return to normal. In many instances body fluids and cardiac output are initially increased but they also return to normal in time. The eventual hemodynamic picture is one of a persistent hypertension with vasoconstriction and normal blood flow. One explanation is that renal artery stenosis initially impairs renal excretion, but that the situation is redressed by fluid retention, increased flows, autoregulatory vasoconstriction, and reestablishment of normal renal perfusion pressure and excretory capability.

Renal artery stenosis with the contralateral kidney left untouched creates a less predictable and less severe hypertension. Plasma renin levels increase markedly after clamping but they then decrease to levels close to, but significantly above, normal in most cases. There is some evidence for fluid retention and the other indicators that imply autoregulatory vasoconstriction, but the data is much less persuasive than in the model with contralateral nephrectomy. The observation that chronic inhibition of angiotensin II formation lowers blood pressure to normal supports an important causal role for the renin-angiotensin system in this model that may be both pressor and antinatriuretic -- involving particularly the intact contralateral kidney.

**ONE-KIDNEY vs TWO-KIDNEY MODELS**

Partial obstruction of one renal artery is seen clinically and it is usually accompanied by moderate to severe hypertension. Stenosis can be diagnosed radiographically and by comparing the function of the suspect kidney to the contralateral kidney. Surgical repair lowers blood pressure -- usually to normal. There is a comparable experimental model wherein renal artery constriction is produced by an external clamp in larger animals and an
external clip in smaller ones. The blood pressure elevation is proportional to the severity of the constriction (571).

Since Goldblatt's initial demonstration in 1934 of the reliability and utility of this method (353), two major variants have been developed. In one case, one renal artery is constricted and the contralateral kidney is removed. This has been called one-kidney Goldblatt hypertension but a recent recommendation is to call it one-kidney, one clip hypertension (735). In the second model, one renal artery is constricted while the contralateral kidney remains untouched. This is called two-kidney Goldblatt hypertension but two-kidney, one clip hypertension is now the recommended name (735) to distinguish this model from the two-kidney, two clip model that is occasionally used. A more suitable name is Wilson-and-Byrom hypertension, recognizing the originators (989). There are striking differences between the one-kidney and two-kidney models and they will be analyzed individually after some preliminary comparisons are made.

Constriction plus nephrectomy is the more predictable and the more popular of the two preparations. Arterial pressure rises rapidly to a value greater than that seen in the two-kidney model and will remain stable at this level for a long time. Pressure will return to normal in the one-kidney model when the constriction is removed but will remain elevated following nephrectomy (302). The one-kidney Goldblatt model has been produced in a wide range of species including cats, dogs, sheep, rabbits and rats.

Blood pressure elevation in the two-kidney model is less predictable, takes longer to obtain, and is not as great as in the one-kidney model. Plasma renin levels are often raised, although the increase in many instances is relatively small. Although the two-kidney model normally shows moderate pressure elevation, it has a tendency to swing into a malignant, accelerated phase of hypertension with very high blood pressure, salt and water loss, high renin levels and poor prognosis. The two-kidney model has been successfully produced in sheep, rabbits and rats but has been only partly successful in the dog.

The two-kidney model is interesting in that total renal function is divided. One kidney is situated beyond a severe constriction and may be underperfused. In contrast, the untouched kidney is perfused at high