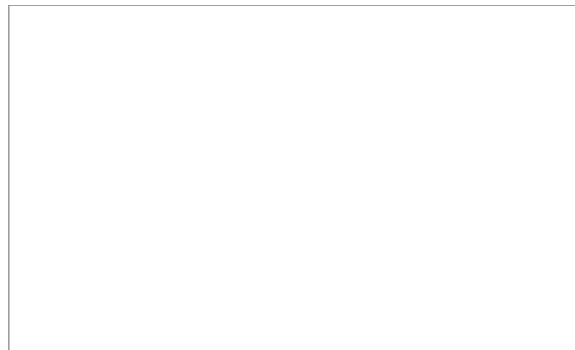


# Renal Artery Stenosis



Partial obstruction of a renal artery -- renal artery stenosis -- produces hypertension. Clinically, the cause is often atherosclerosis or comparable forms of vascular damage. Experimentally, renal artery stenosis is produced by a clamp.

Before the work of Harry Goldblatt, there was no reliable way to produce hypertension in experimental animals. Then Dr. Goldblatt demonstrated that partial constriction of the renal arteries produces a sustained blood pressure increase (shown below, data from Goldblatt) that is proportional to the severity of the constriction. This technique was then simplified to partial constriction of one renal artery with surgical removal of the other kidney.



Clinically, hypertension due to renal artery stenosis is relatively rare and potentially very serious. It is often surgically correctable.

Renal artery stenosis decreases pressure in the renal vasculature beyond the obstruction. Decreased pressure immediately decreases salt and water excretion. Renin secretion is stimulated. First angiotensin II and then retained sodium work to elevate systemic arterial pressure. An increase in arterial pressure elevates renal artery pressure beyond the stenosis and sodium balance is reestablished. The price paid for reestablishing sodium balance is chronic arterial hypertension (shown below, data from Murphy).





### ***The Renal Artery Stenosis Protocol***

Begin by clicking Restart to reset the model's variables to their initial values. Record control data in the table below. Then go to  Circulation. Slide the renal artery stenosis slider over to severe and record data for the immediate response (0+). Advance time and record data as hypertension develops.

Arterial Pressure (mmHg)

Arcuate Artery Pressure (mmHg)

Plasma Renin Activity



Sympathetic Nerve Activity

Plasma Volume (mL)  
Extracellular Fluid Volume (L)

Na<sup>+</sup> Excretion (mEq/Min)

Time	0 Min	0+ Min	1 Hr	1 Day	1 Week
Arterial Pressure					
Arcuate Pressure					
PRA					
Sympathetics					
Plasma Volume					
ECFV					
Na <sup>+</sup> Excretion					

## *References*

Goldblatt, H., J. Lynch, R. F. Hanzal, & W. W. Summerville. Studies on experimental hypertension. I. The production of persistent elevation of systolic blood pressure by means of renal ischemia. *J. Exp. Med.* 59:347-379, 1934.

Murphy, W. R., T. G. Coleman, T. L. Smith, & K. A. Stanek. Effects of graded renal artery constriction on blood pressure, renal artery pressure, and plasma renin activity in Goldblatt hypertension. *Hypertension* 6:68-74, 1984.

