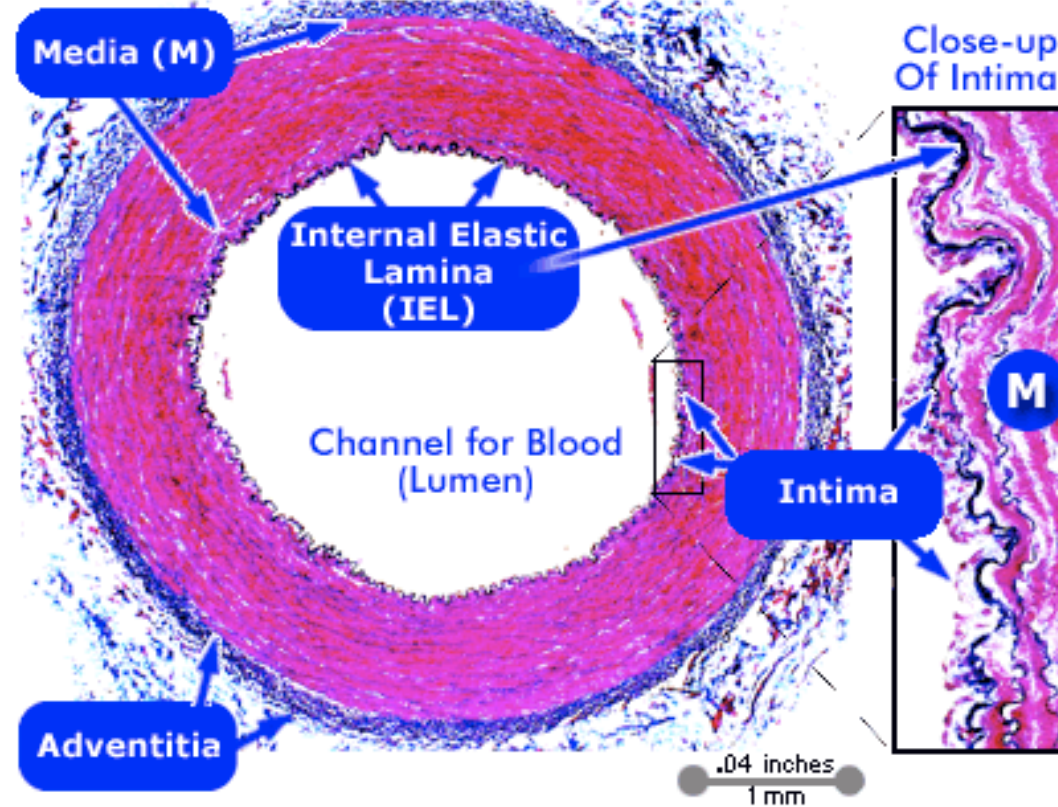


Pathophysiology of Hypertension

Robert C. Stanton, MD
Associate Professor of Medicine
Harvard Medical School
Chief of Nephrology
Joslin Diabetes Center

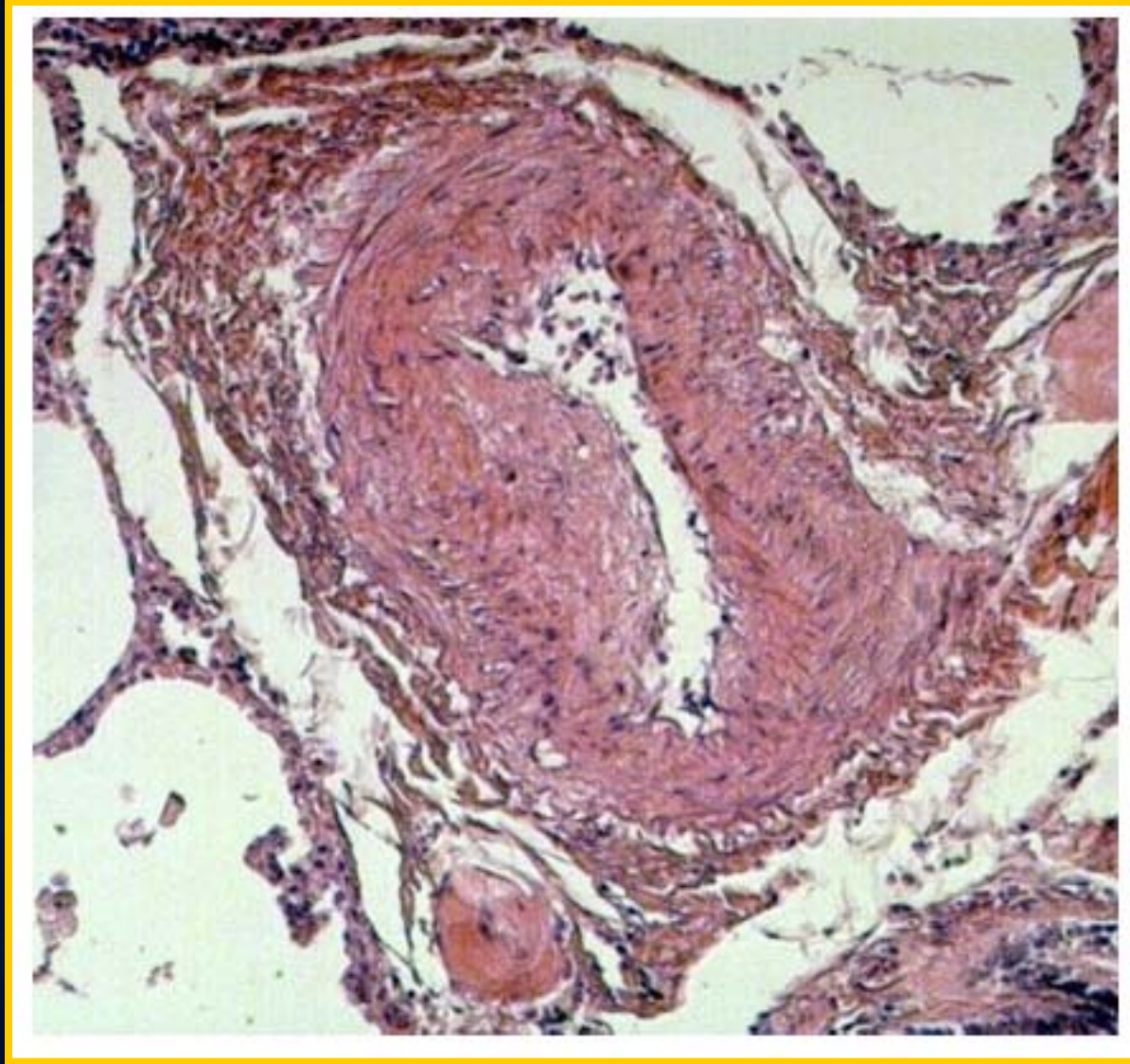
Normal Artery

**Figure 1: Normal Coronary Artery
Cross-sectional Microscopic View**
(Move your mouse over the words for highlights.)



In **Figure 1**, smooth muscle is red, and connective (supporting) tissue is black (elastic) or blue (collagen).

Hypertensive Artery



mediswww.meds.cwru.edu/.../vascular.html

Blood Pressure

$$\text{BP} = \text{Cardiac Output} \times \text{Total Peripheral Resistance}$$

$$\text{Heart Rate} \times \text{Stroke Volume}$$

β blockers

Salt restriction

Diuretics (e.g., thiazides)

Natriuretics (e.g., ANP, BNP)

Aquaretics (AVP rec. antagonist)

Aldosterone antagonist

Calcium channel blockers

Dihydropyridines

Nondihydropyridines

Angiotensin II inhibitors

Renin inhibitor

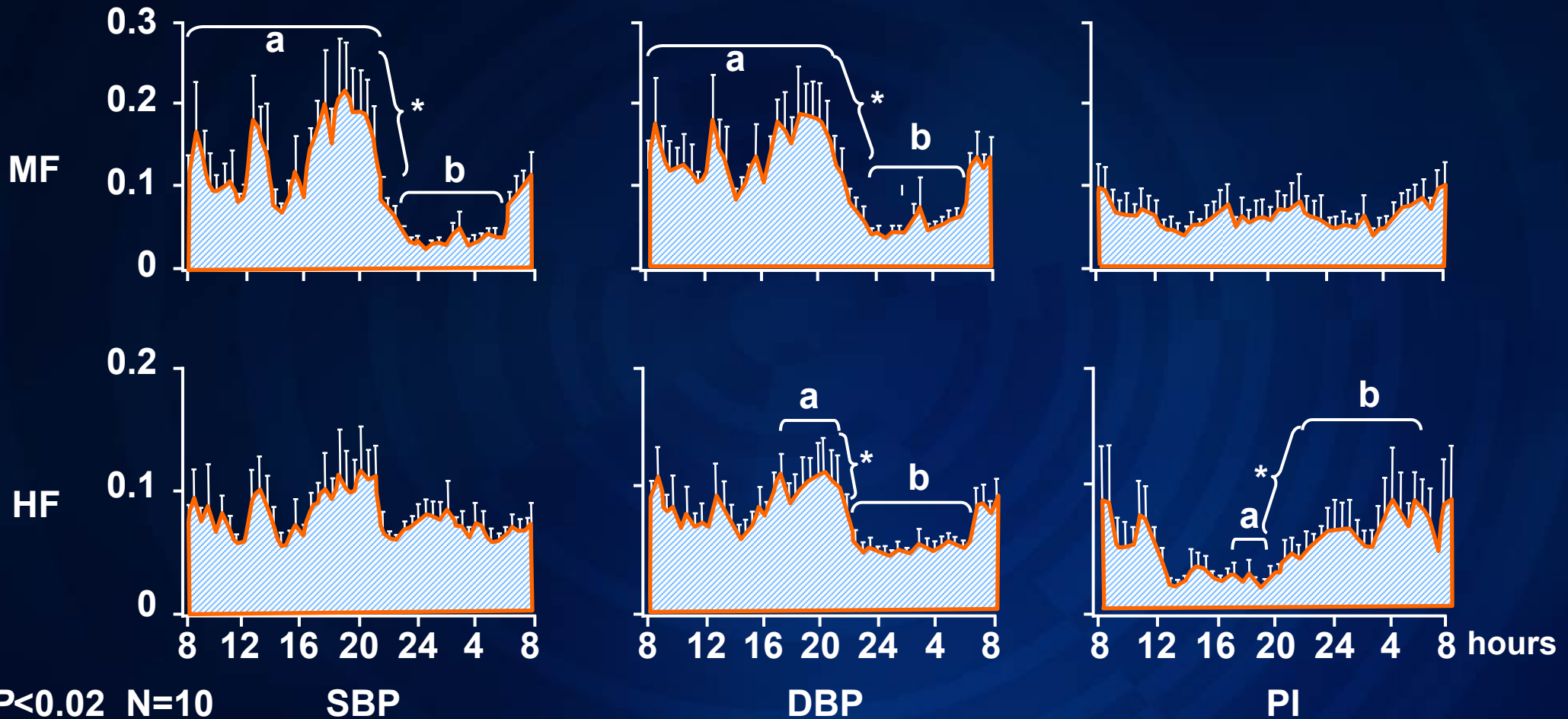
Central adrenergic agonists

Alpha blockers

Nitric oxide enhancers

Normal Physiology of Blood Pressure

Dippers vs Nondippers



Nondippers Have Higher Mortality

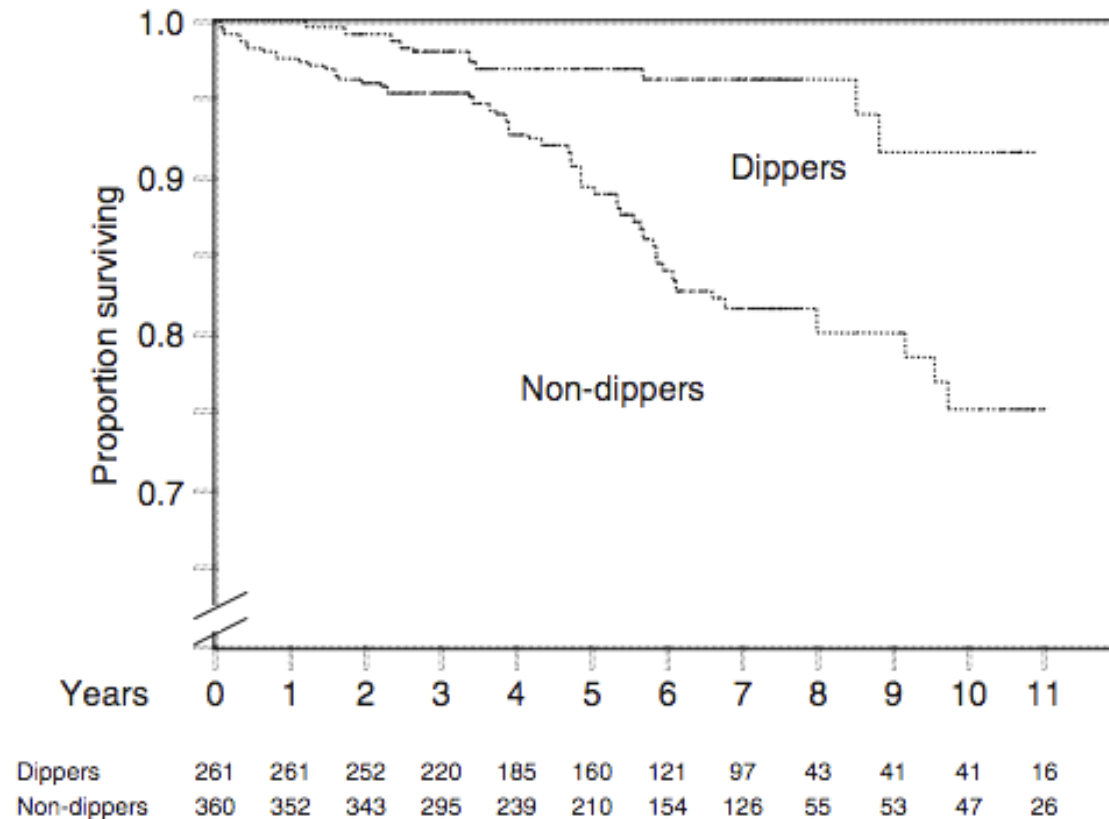
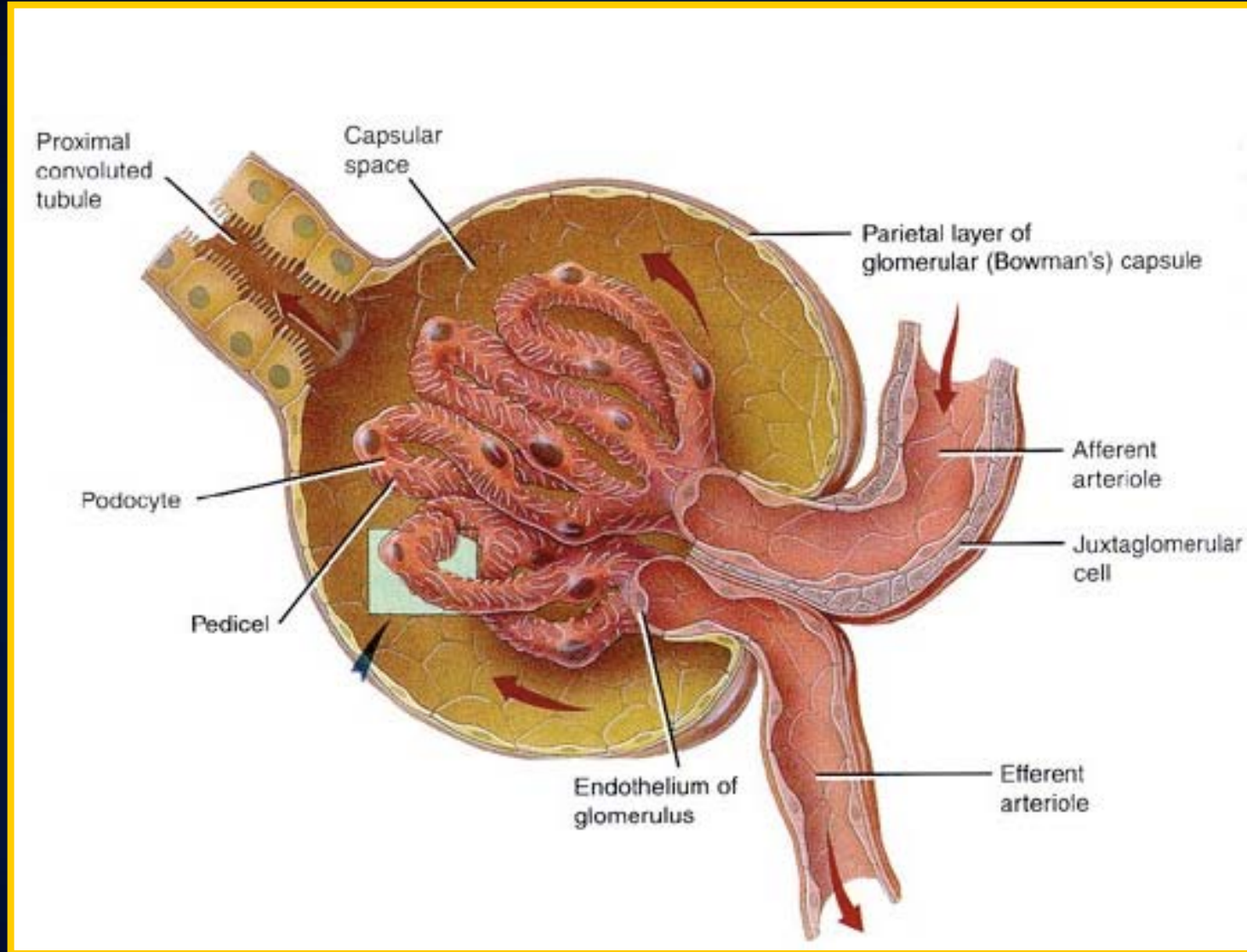


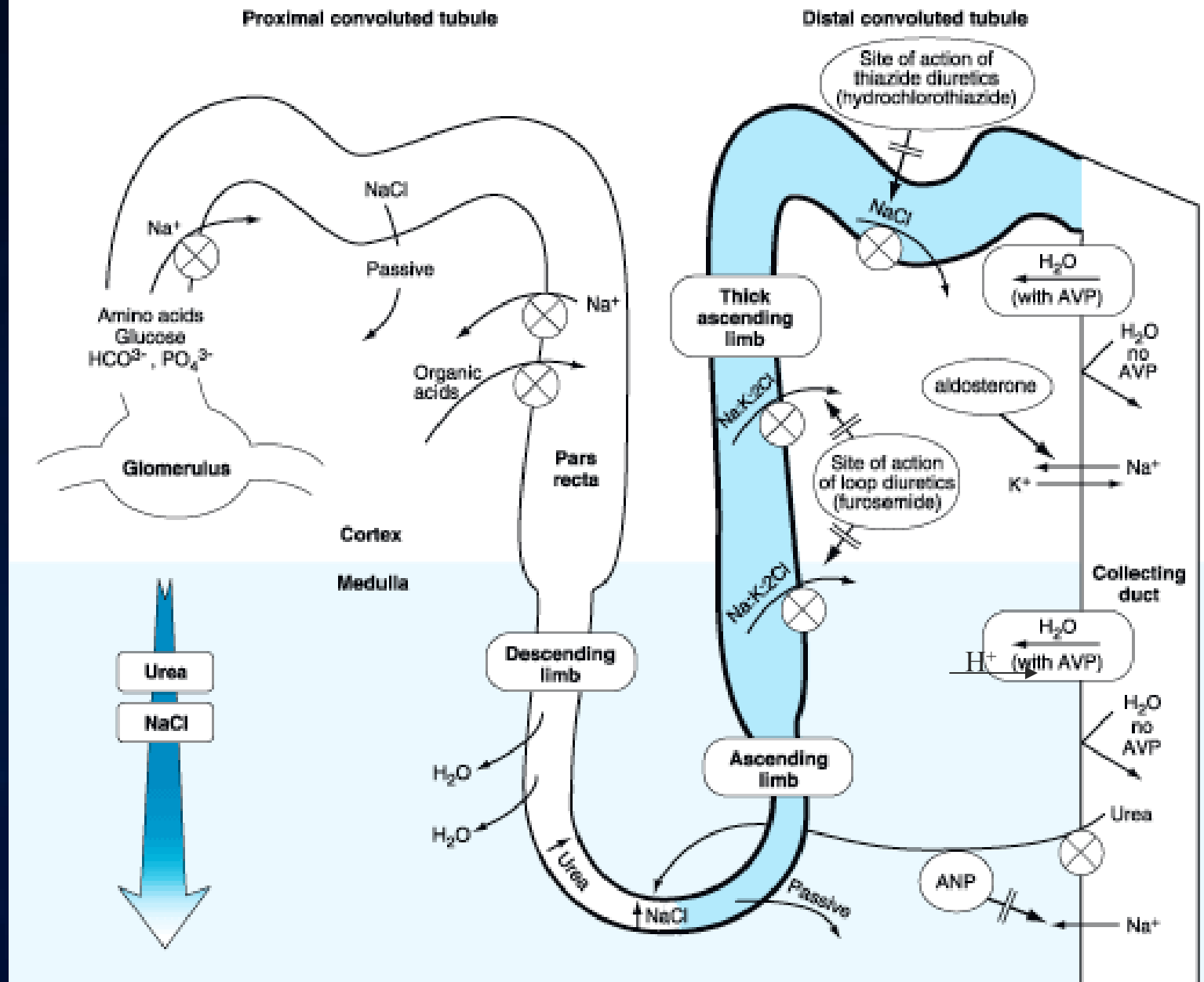
Figure 1 | Kaplan–Meyer plot of all-cause mortality in patients with normal diurnal variation in systolic blood pressure (dippers) vs. those with abnormal diurnal variation in systolic blood pressure (non-dippers) ($P < 0.0001$ by log-rank test).

Autonomic Nervous System and Hypertension

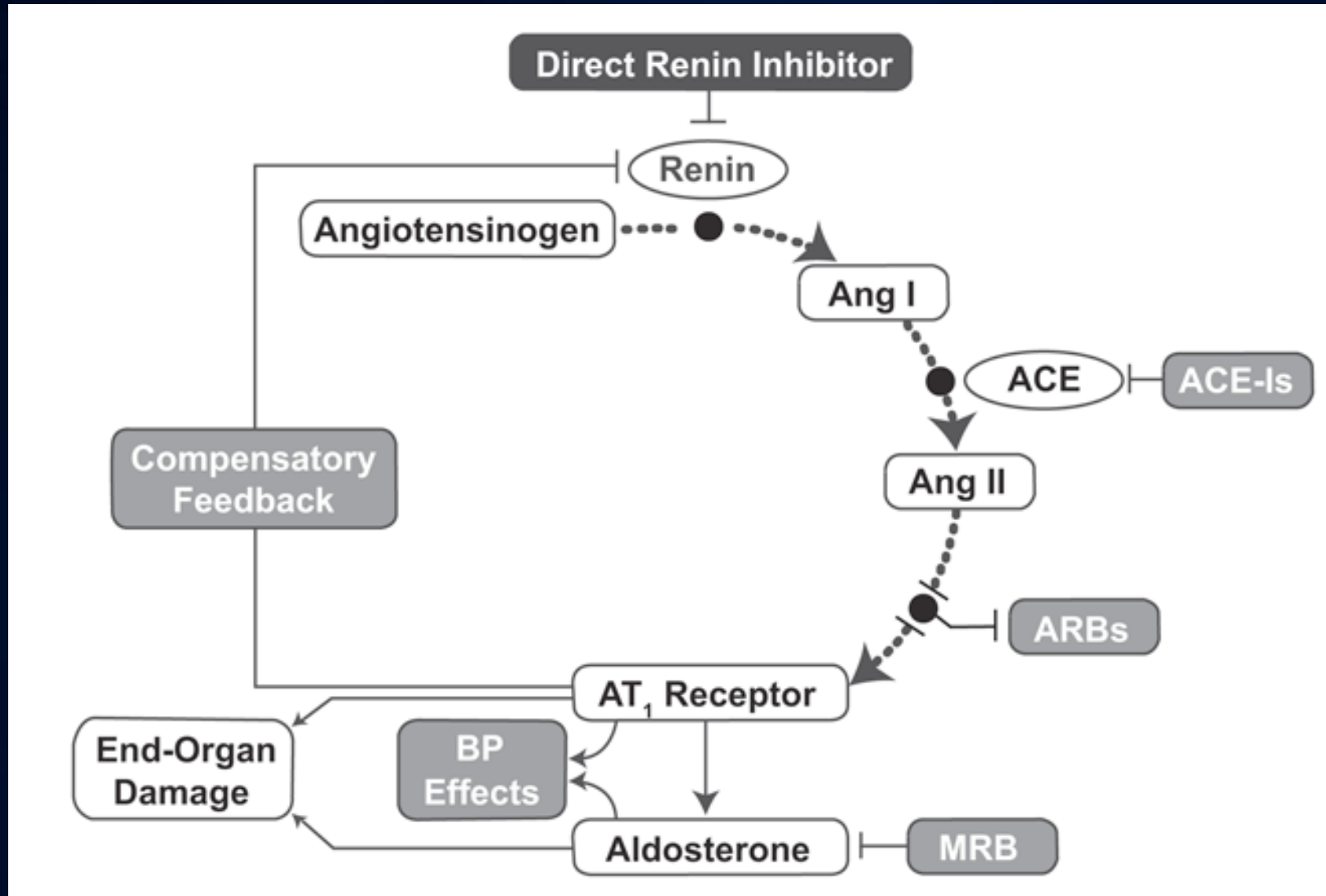
Glomerulus



Nephron



Renin-Angiotensin-Aldosterone System

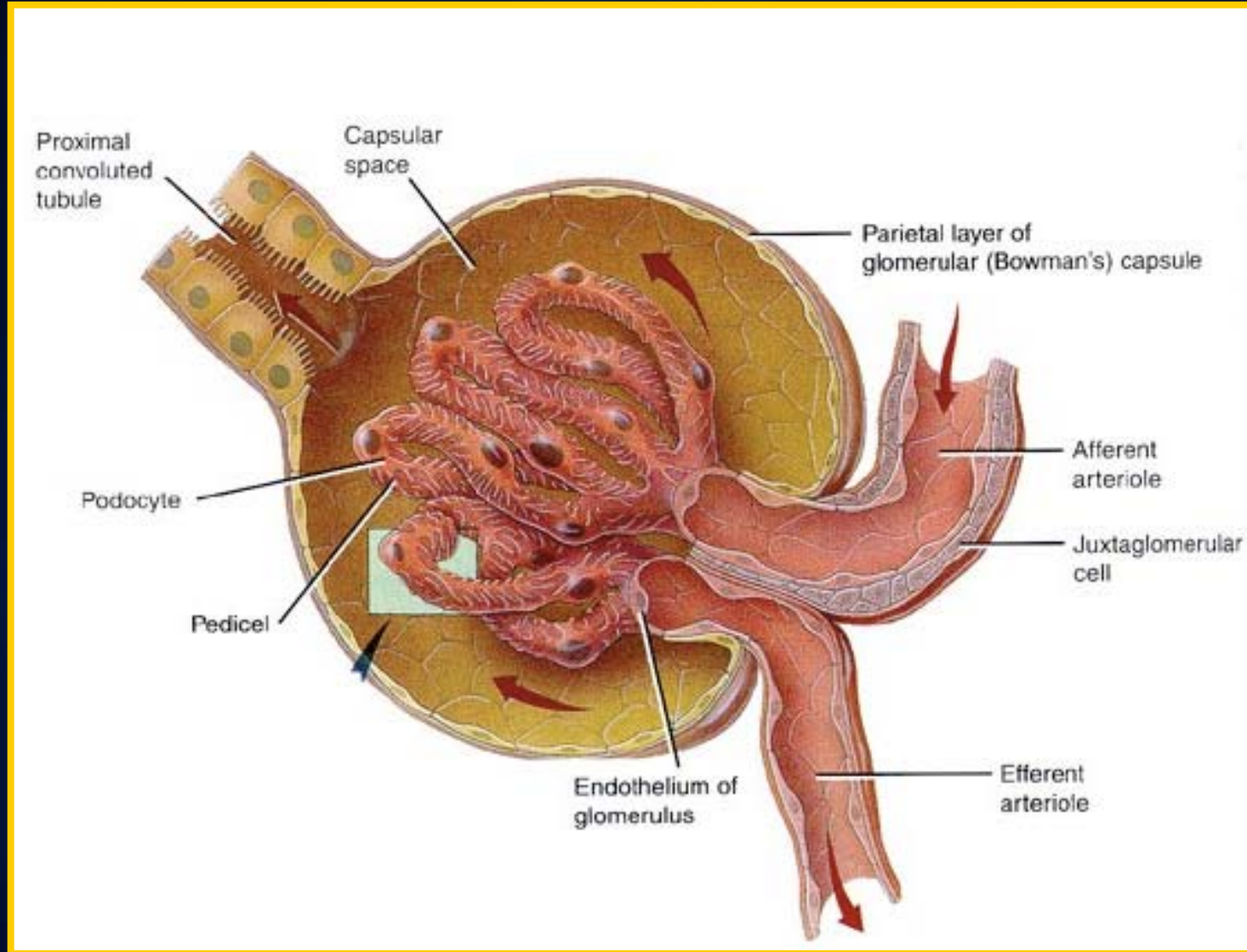


Vasodilators and Vasoconstrictors

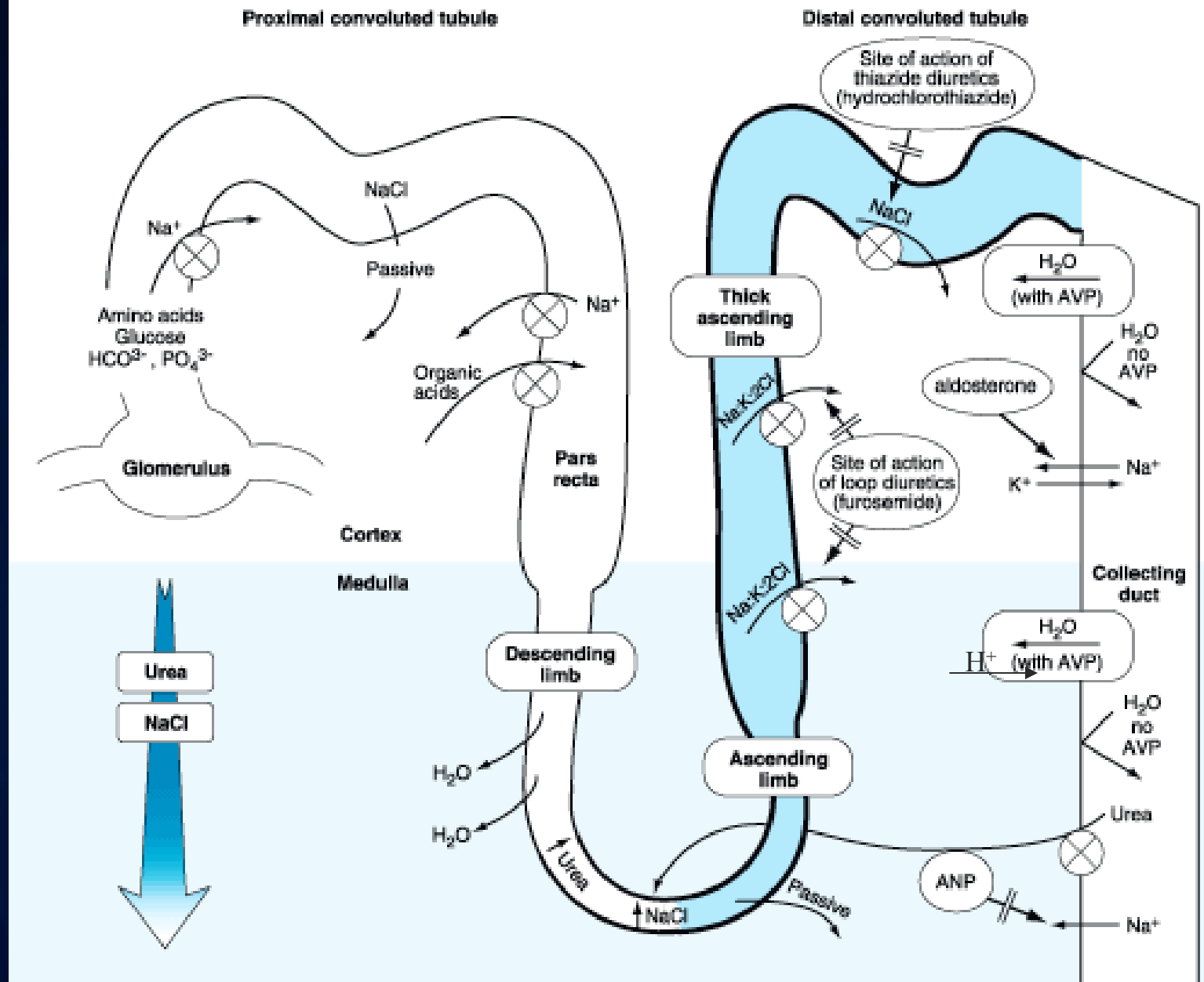
Secondary Causes of Hypertension

- **Renal artery stenosis**
- **Primary hyperaldosteronism**
- **Pheochromocytoma**

Glomerulus



Nephron



Salt Sensitivity

and

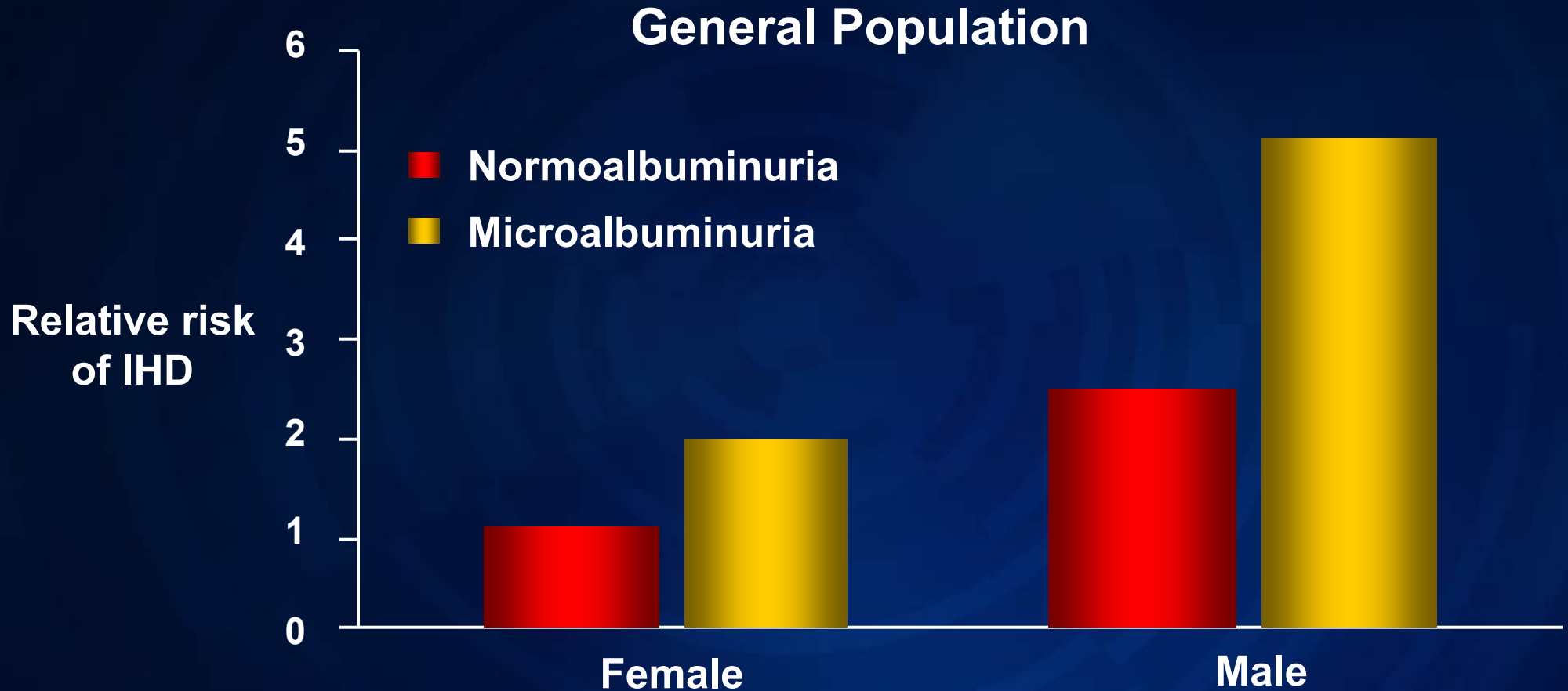
Epigenetic Factors

Known Genetic Causes of Hypertension

- Liddle's Syndrome
- 11- β hydroxylase deficiency

Urine Albumin and Endothelial Disease and Hypertension

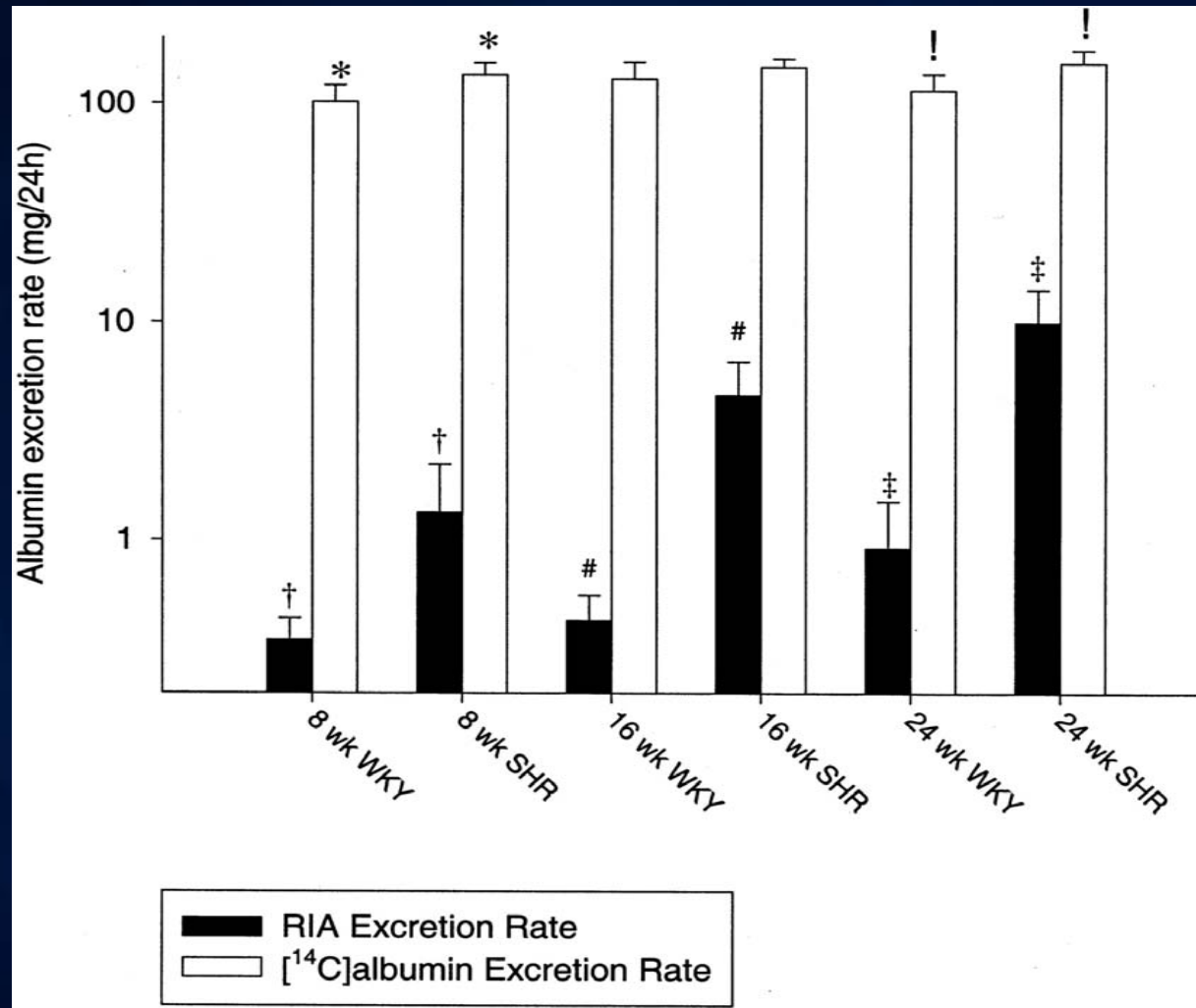
Microalbuminuria and Ischemic Heart Disease Risk



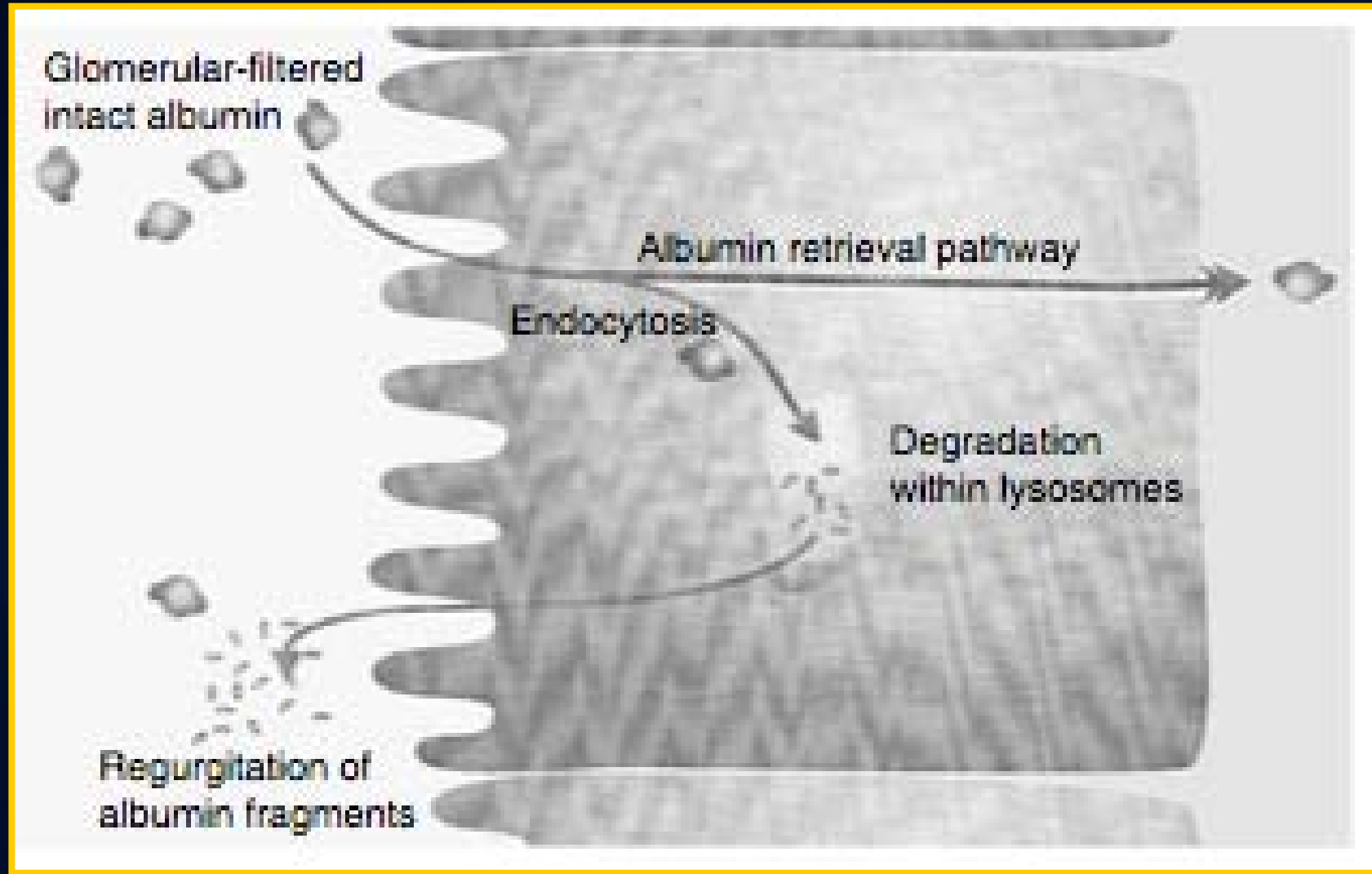
10-y follow-up, N=2085

Borch-Johnsen et al. *Arterioscler Thromb Vasc Biol.* 1999;19:1992-1997.

Excretion Rates of Intact Albumin (as Determined by RIA, Closed Bars) in 8- and 16-Week SHR and WKY (n=6), 24-Week SHR (n=4), and 24-Week WKY N=6)



More Than 2 g of Albumin Are Filtered Daily: Most Is Reabsorbed, Degraded, and Excreted as Albumin Fragments



**Albuminuria might be a marker for
generalized inflammation and
endothelial dysfunction.**