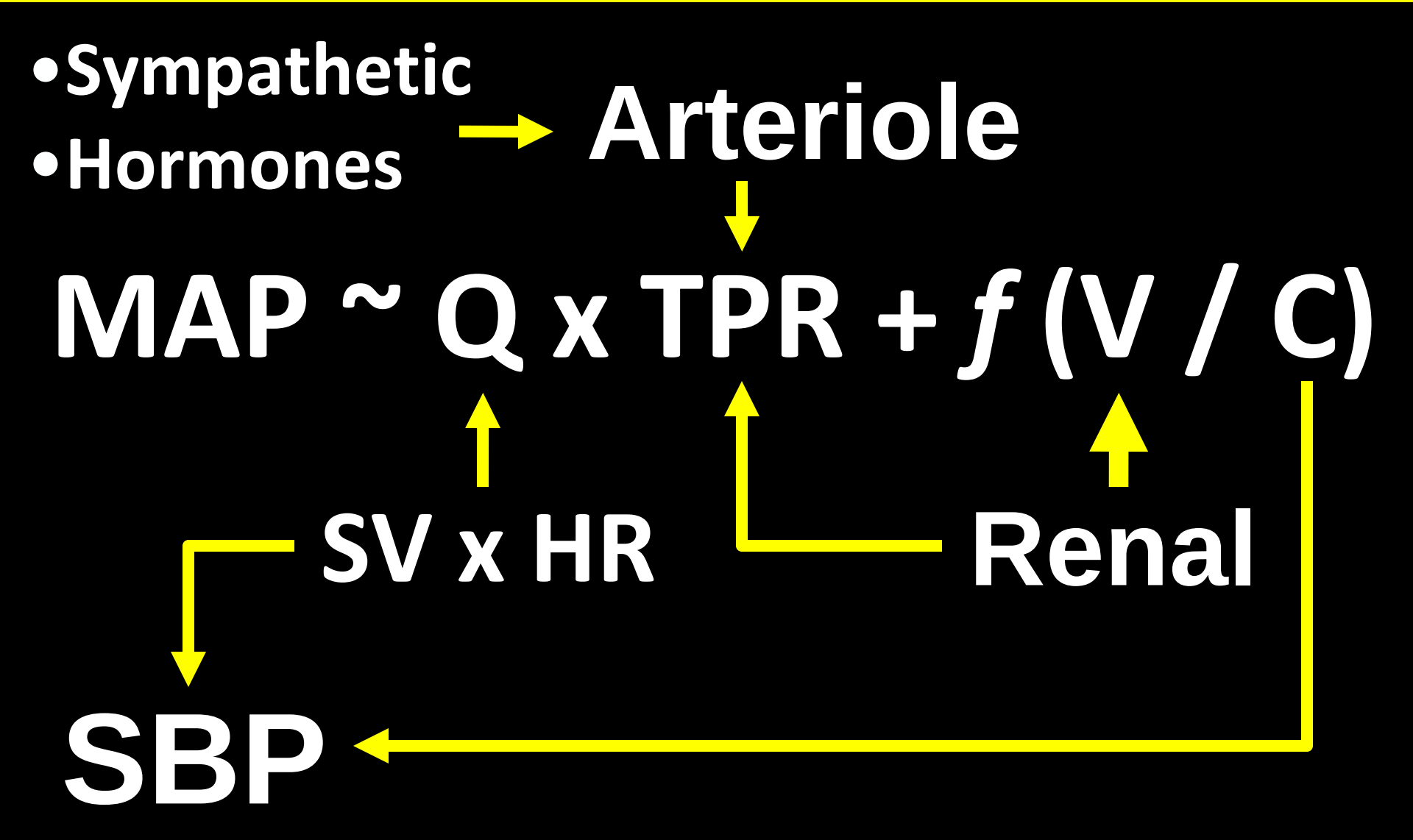
Special Lecture – 11/08/2013

Hypertension – Dr. HN Mayrovitz

Arterial Blood Pressure (ABP)
Major Factors Summarized



Hypertension = High Blood Pressure

So ……. What's High?

Hypertension

BP CLASSIFICATION	SBP (mmHg)	DBP (mmHg)	MAP
Normal	< 120	AND <80	< 93
Prehypertension	120-139	OR 80-89	93-106
Stage 1 Hypertension	140-159	OR 90-99	107-119
Stage 2 Hypertension	>= 160	OR >= 100	> 120

If DBP is normal but SBP is high then called Isolated Systolic Hypertension

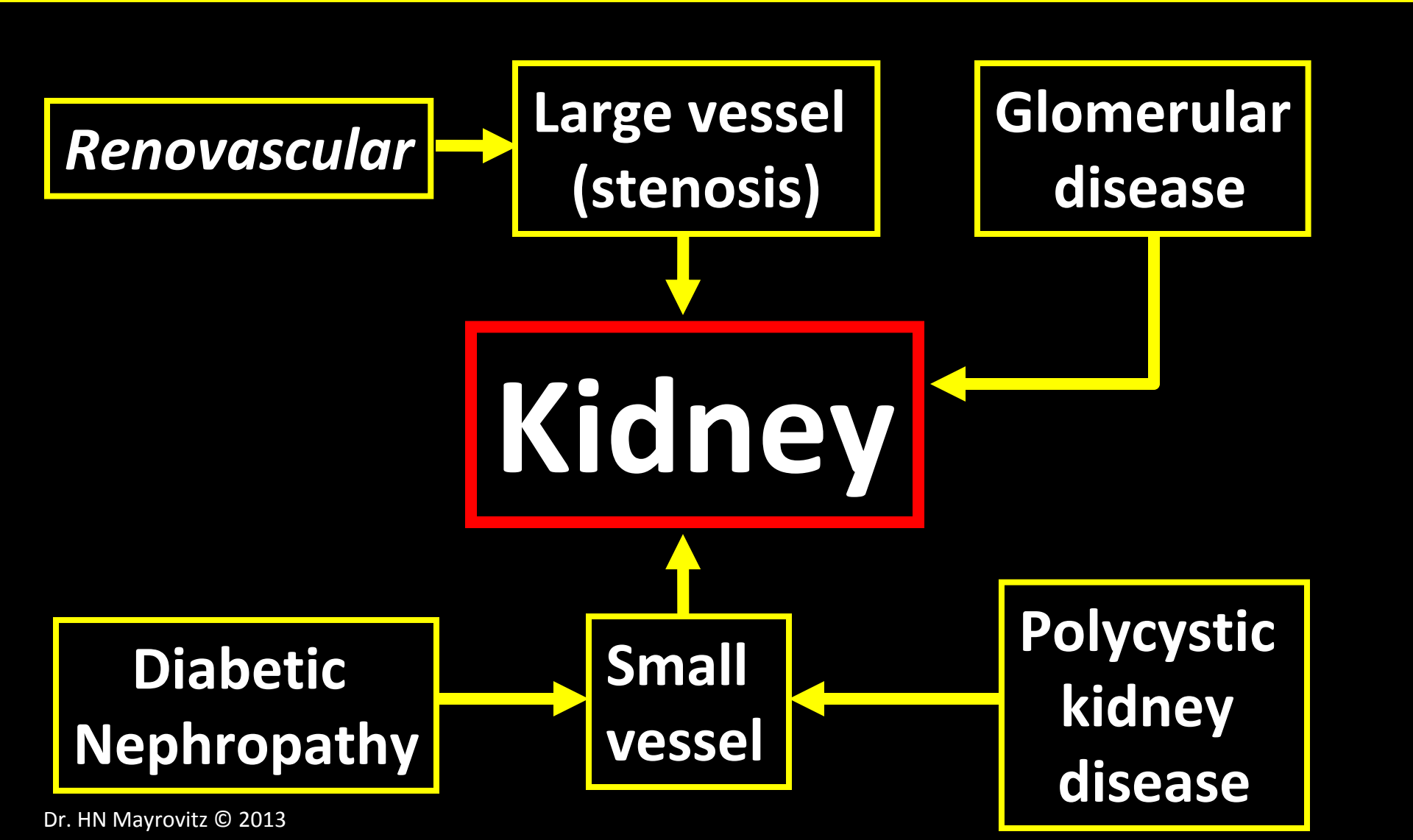
→ ***Decreased Arterial Compliance***

Specific Known Hypertension Causes (Secondary Hypertension)

~ 10 % of Hypertension Patients

Remainder → Essential HTN (Primary HTN)

Specific Known Hypertension Causes (Secondary Hypertension - Renal)



Specific Known Hypertension Causes (Secondary Hypertension - Other)

Overproduction of:

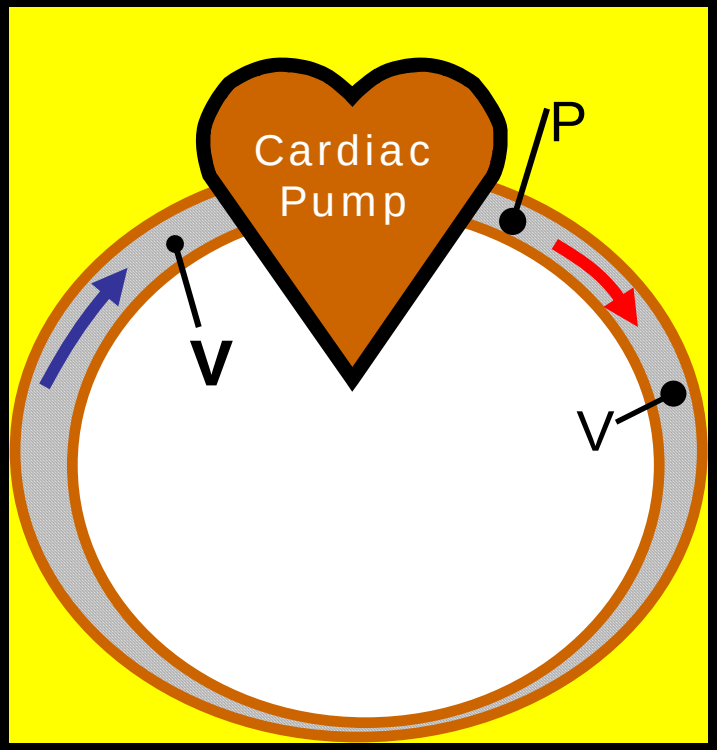
- adrenal Cortisol (pituitary/adrenal tumor-Cushings)
- adrenal E or NE (adrenal tumor – Pheochromocytoma)
- thyroid hormones (Hyperthyroidism)
- parathyroid hormones (Hyperparathyroidism)
- aldosterone (adrenal tumor – Aldosteronism)

- Aortic coarctation (narrowed aorta)
- Pregnancy-induced (preeclampsia)

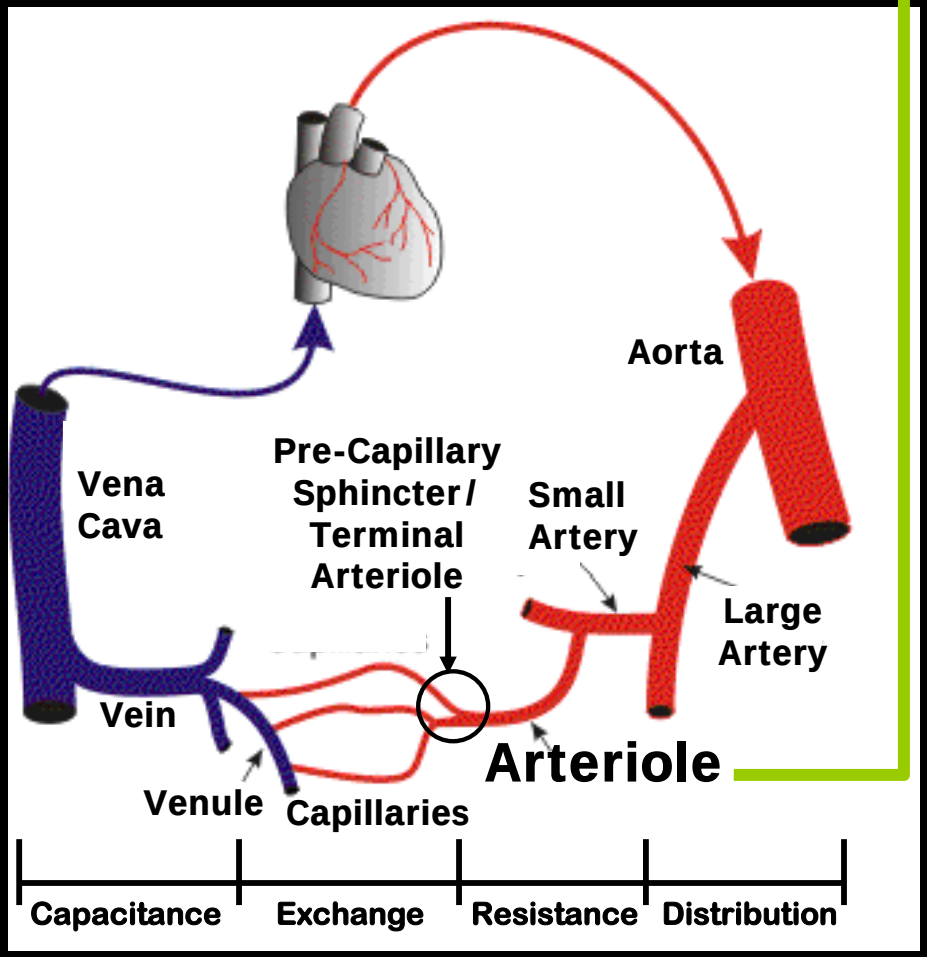
Some ABP Determinants

- TPR
- Blood Volume
- Vascular Compliance

Resistance as a ABP Determinant

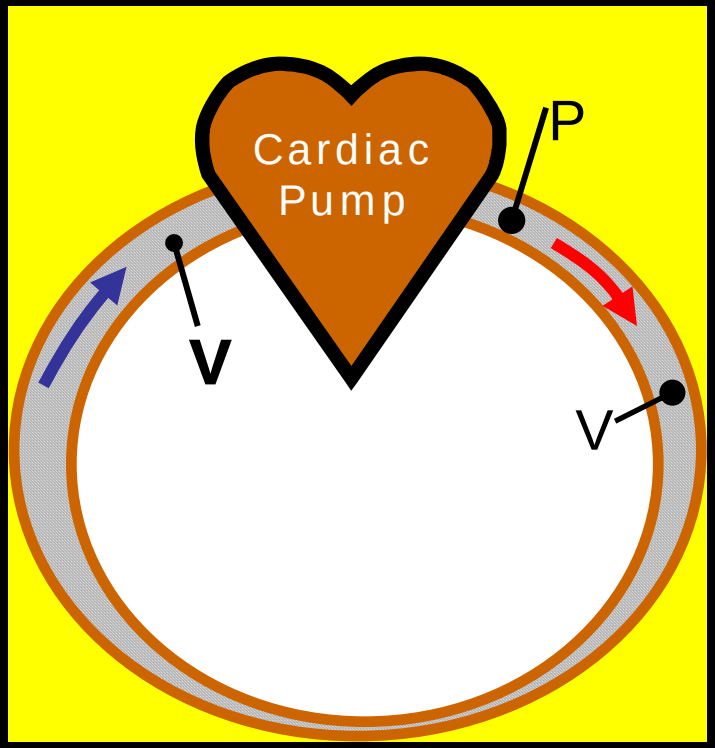


MAP = CO x TPR



Blood Volume as an ABP Determinant

2. F-S
+ Volume
+ CVP
+ Filling
+ SV
+ CO
+ MAP



1. Mechanical
+ Volume
+ Pressure

Compliance Related V/C

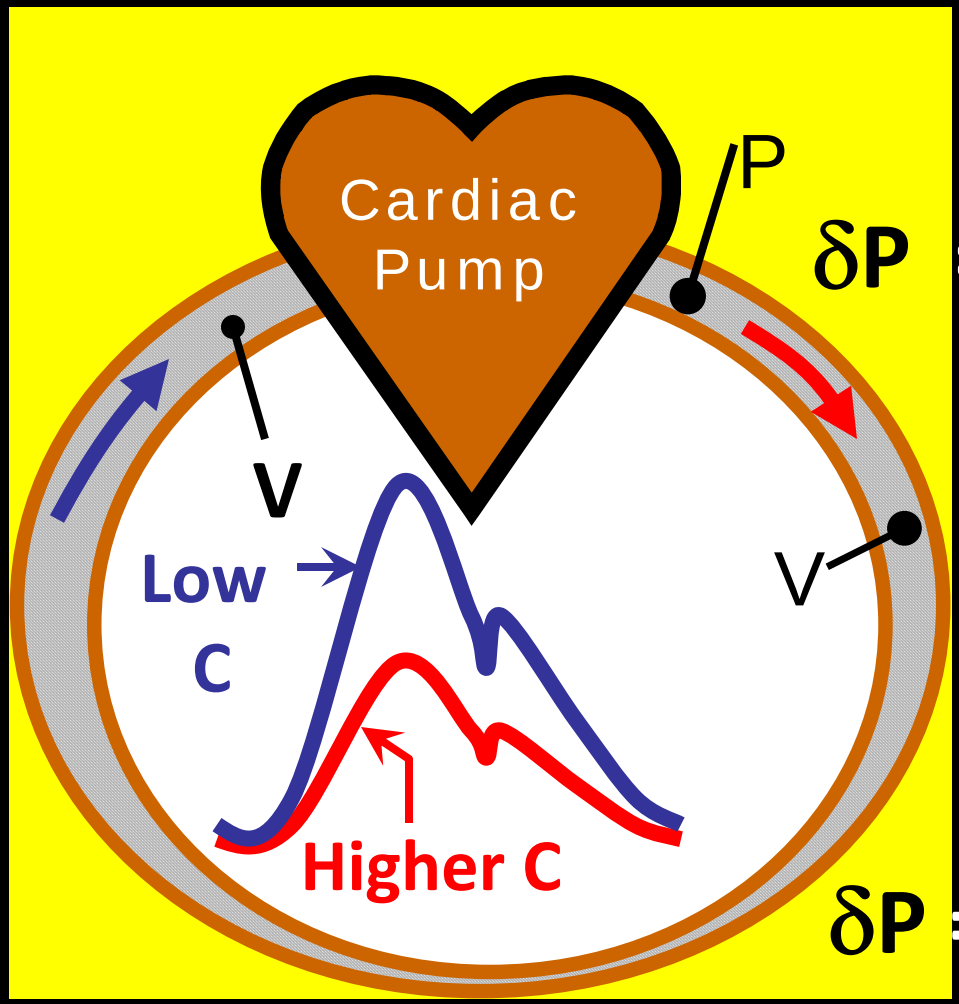
Compliance as an ABP Determinant

+Systolic

+ dV/dt

+ SV

- C



$\delta P = \delta V / C$

↓

SV

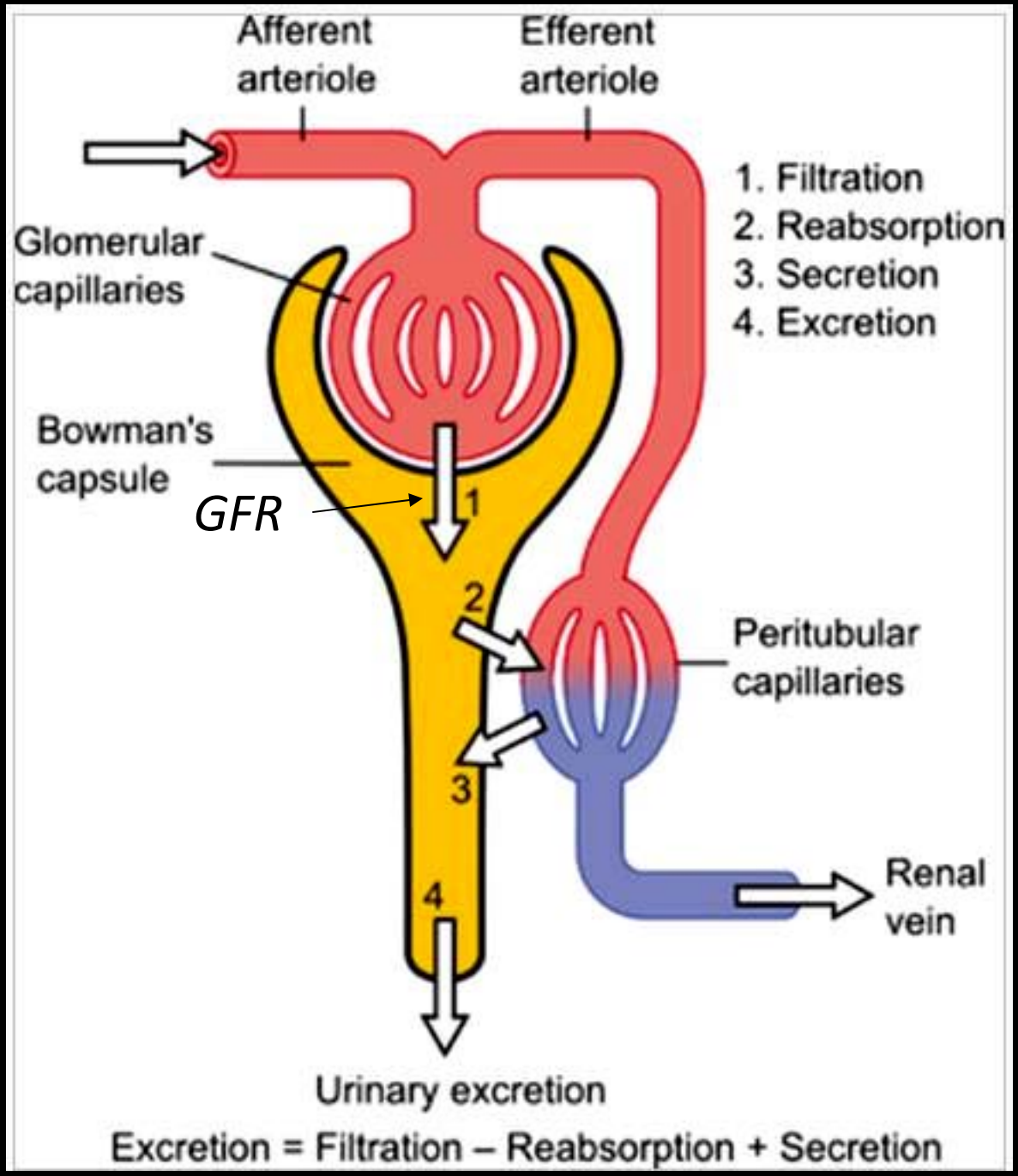
$\delta P = SV / C$

Mainly Systolic and Pulse Pressure Effects

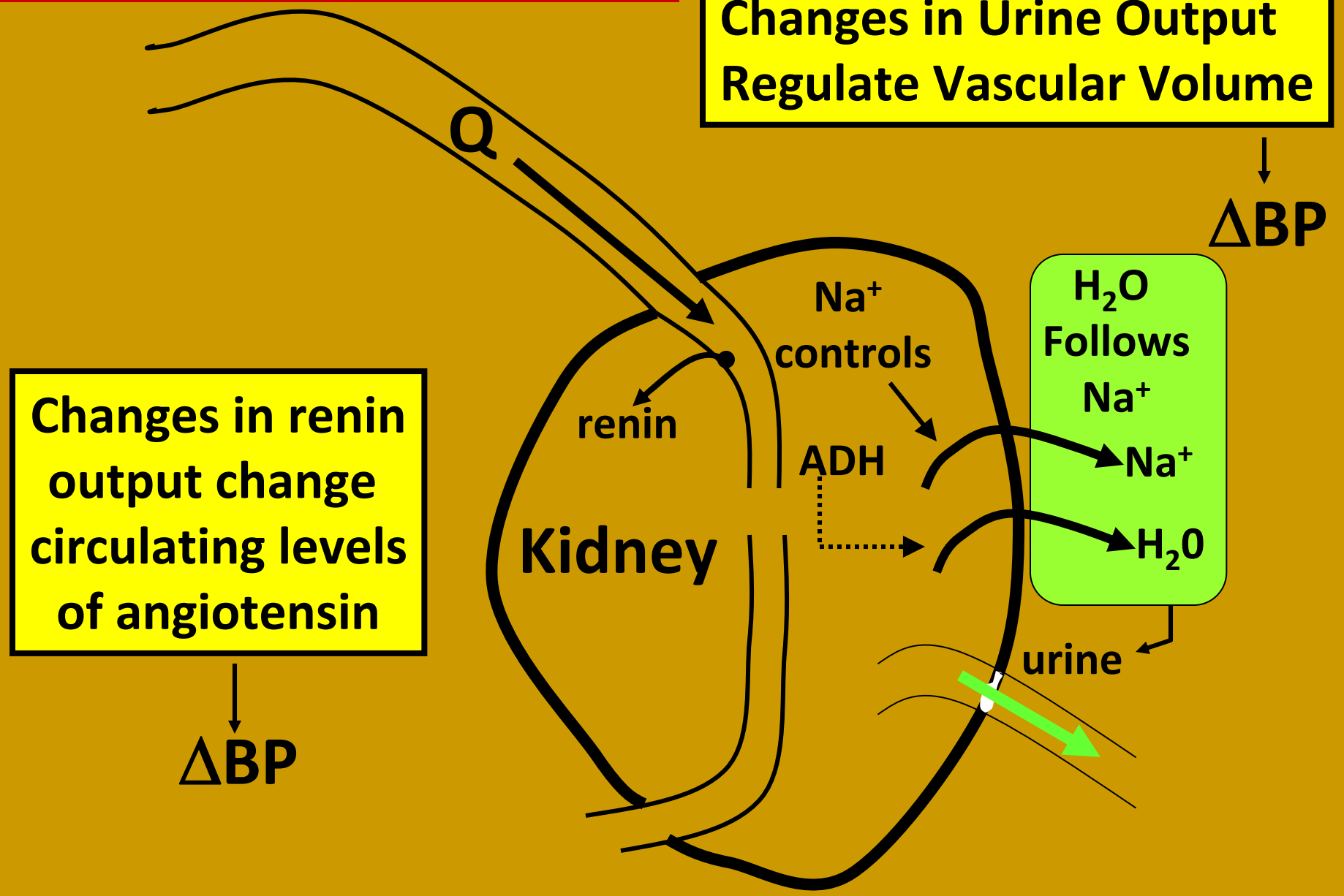
Renin – Angiotensin – Aldosterone System

Volume and ABP Changes via Effects of:

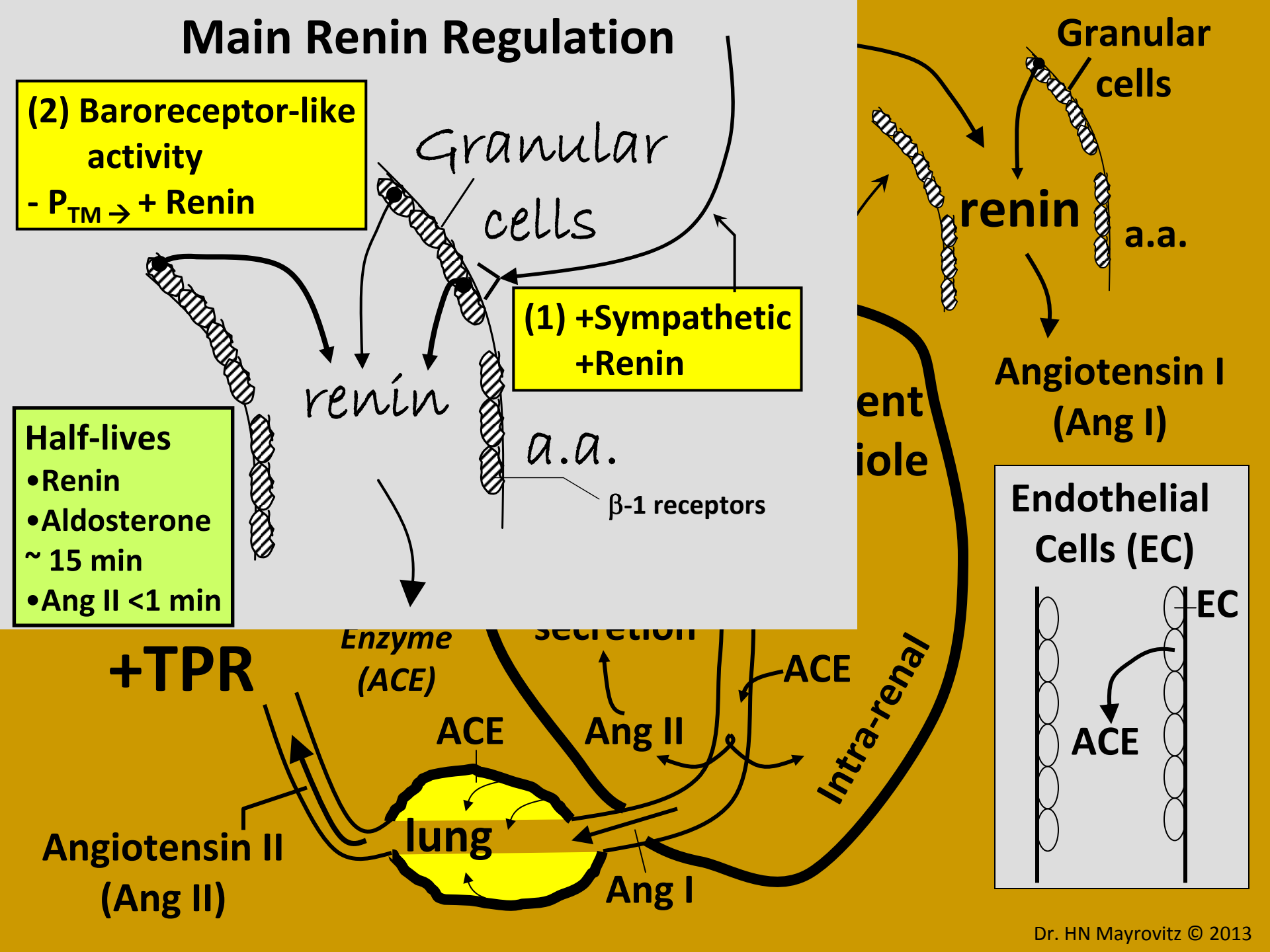
- **Renin**
- **Angiotensin**
- **Antiduretic Hormone (ADH)**
- **Aldosterone**


Afferent arteriole
Efferent arteriole
1. Filtration
2. Reabsorption
3. Secretion
4. Excretion
Glomerular capillaries
Bowman's capsule
GFR
1
2
3
4
Peritubular capillaries
Renal vein
Urinary excretion
Excretion = Filtration – Reabsorption + Secretion

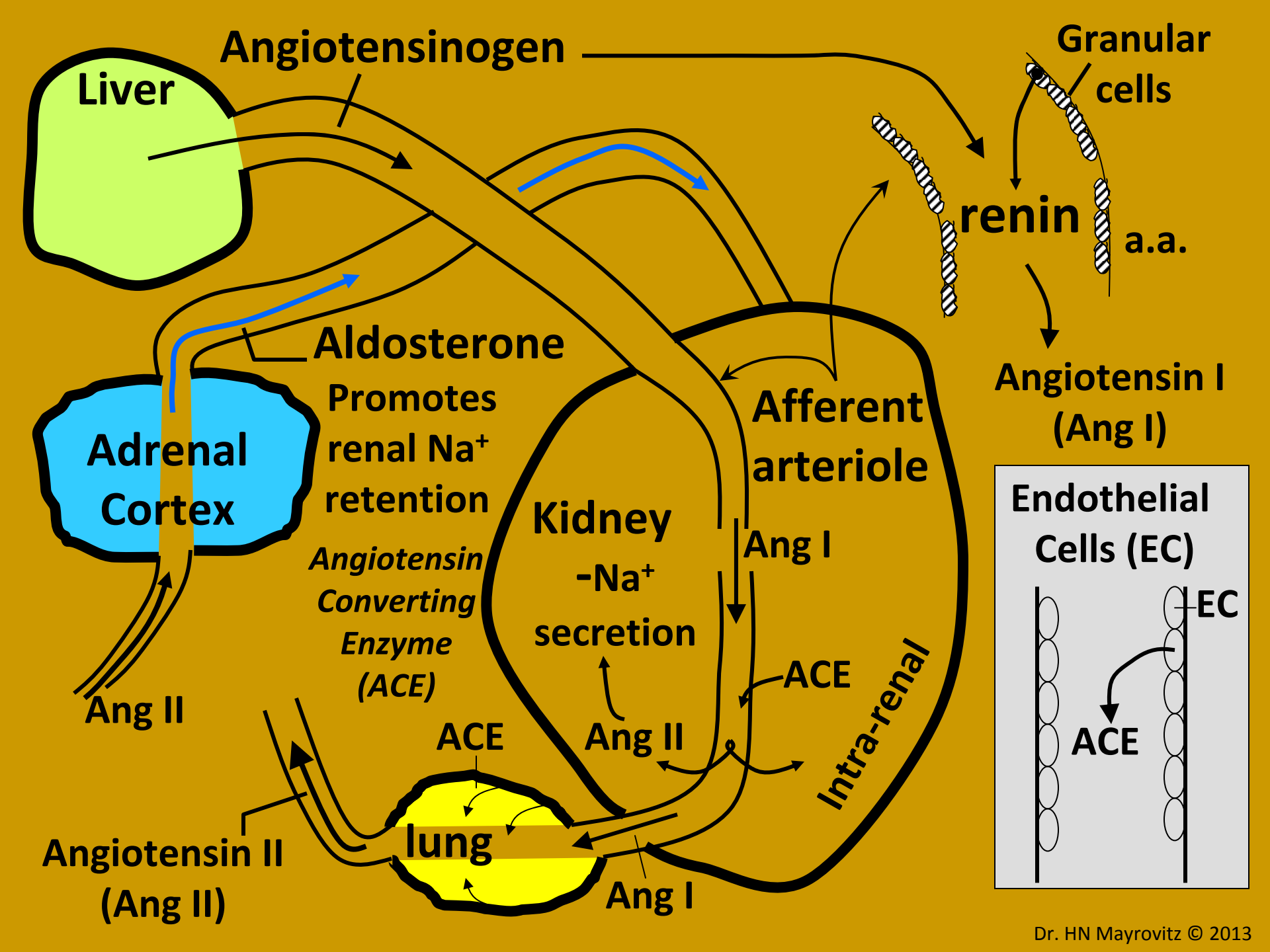
General Renal Effects



1. Renin – Angiotensin
Main Pathways

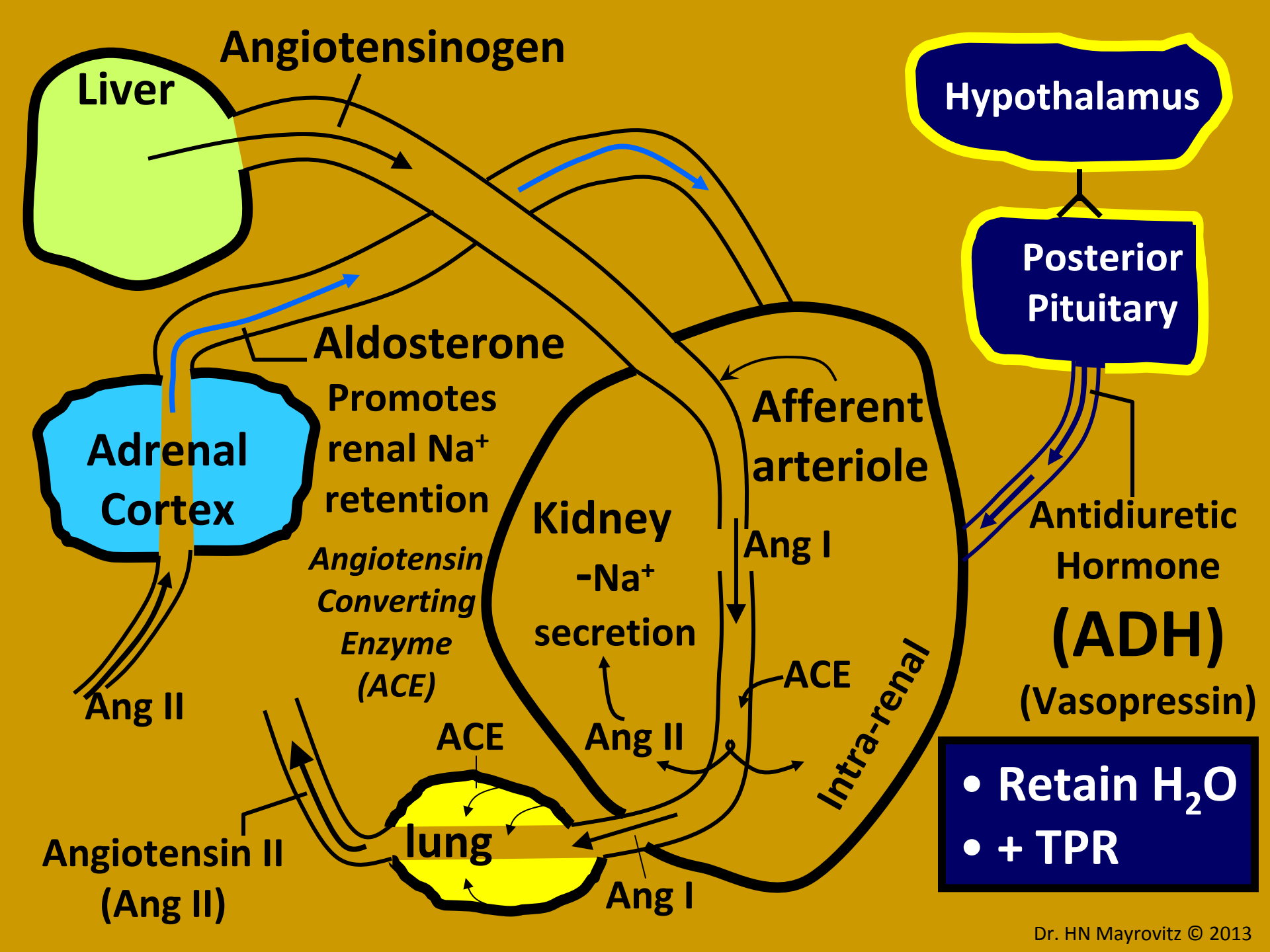


2. Include Aldosterone as a Modulator of Na^+ Retention and Urine Output



3. Include Effects of Antidiuretic Hormone

ADH=Vasopressin as a Modulator of H_2O Excretion

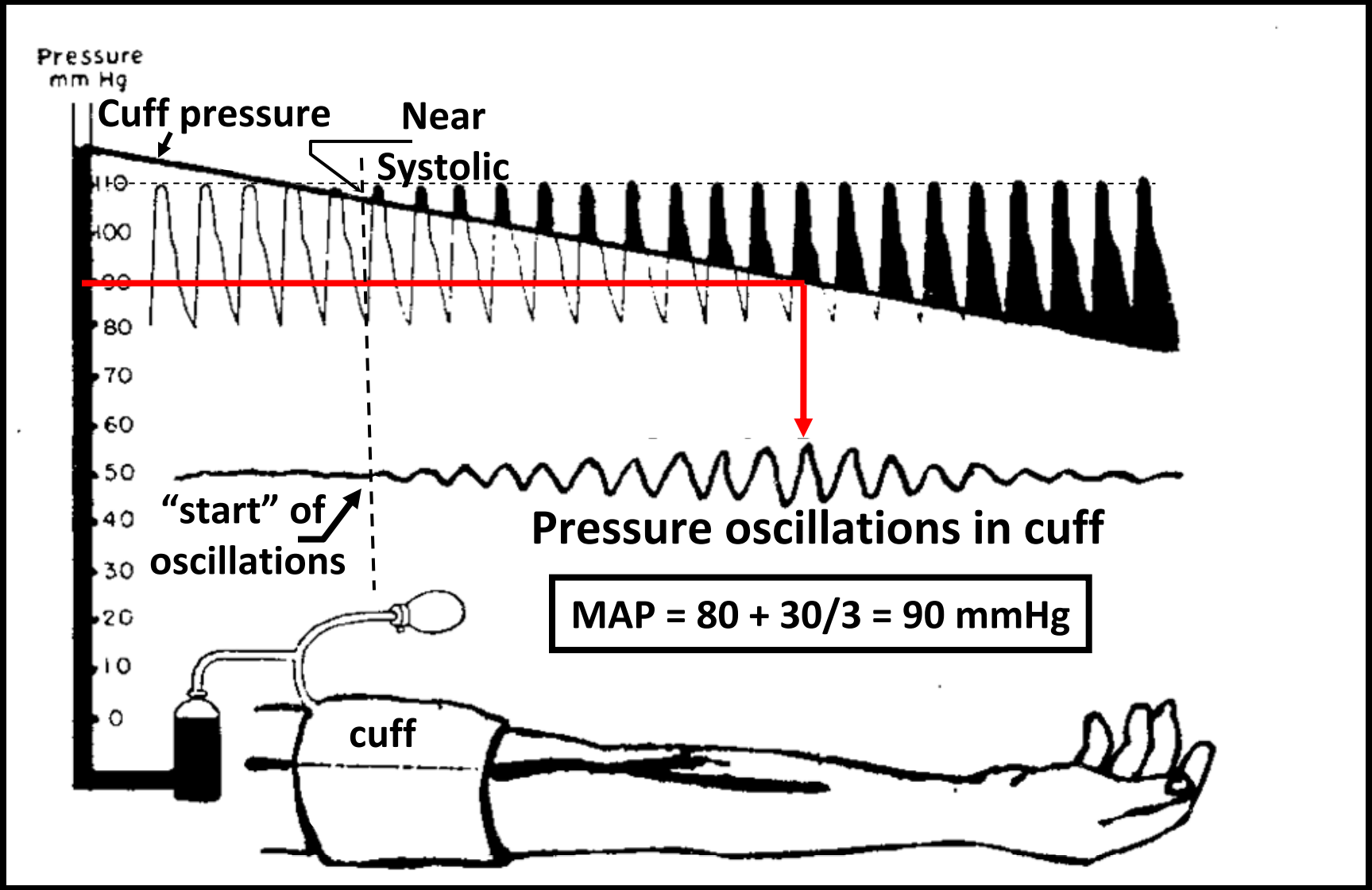


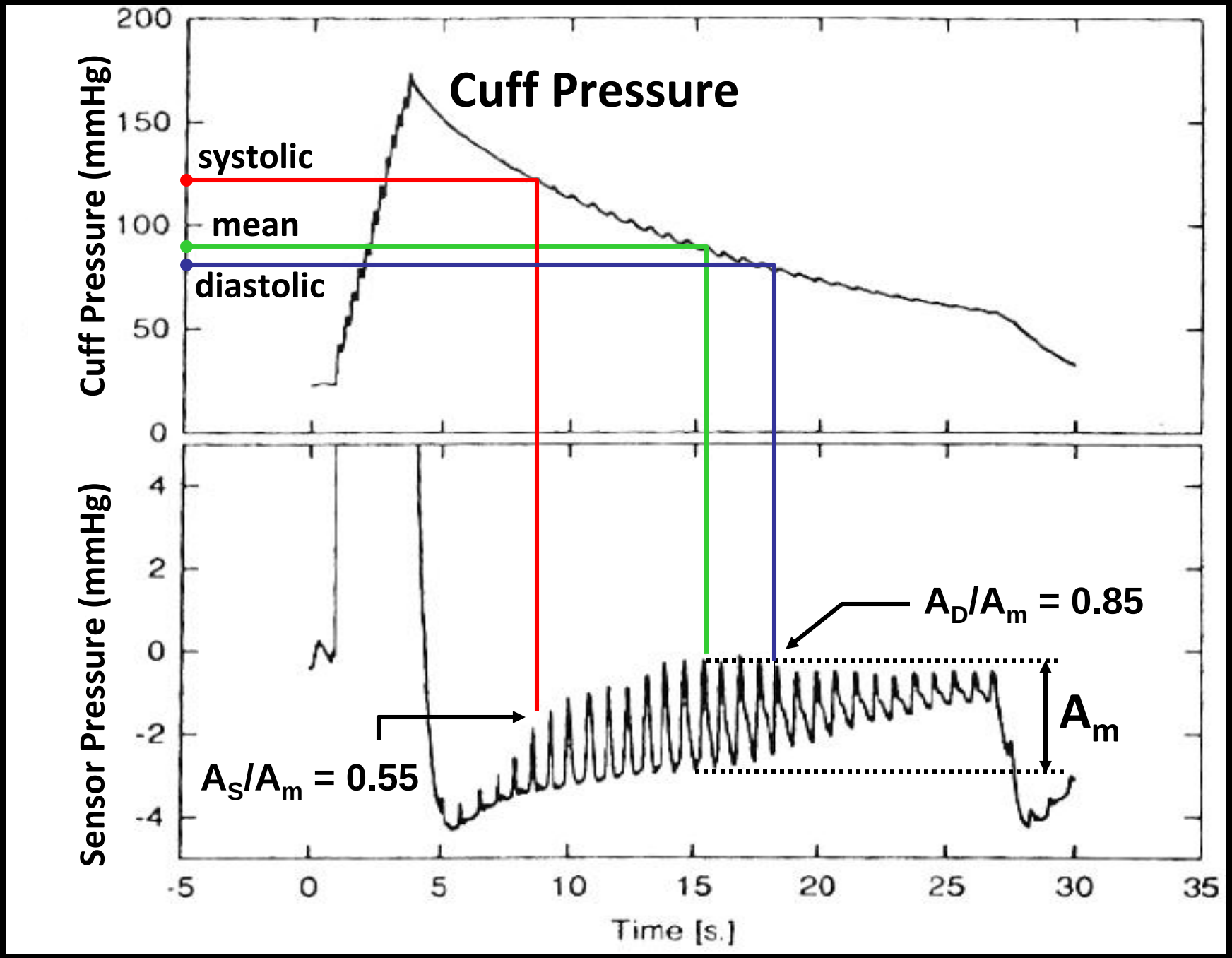
Non-invasive Indirect Measurement

Oscillographic

Auscultation

ABP via Oscillographic Method

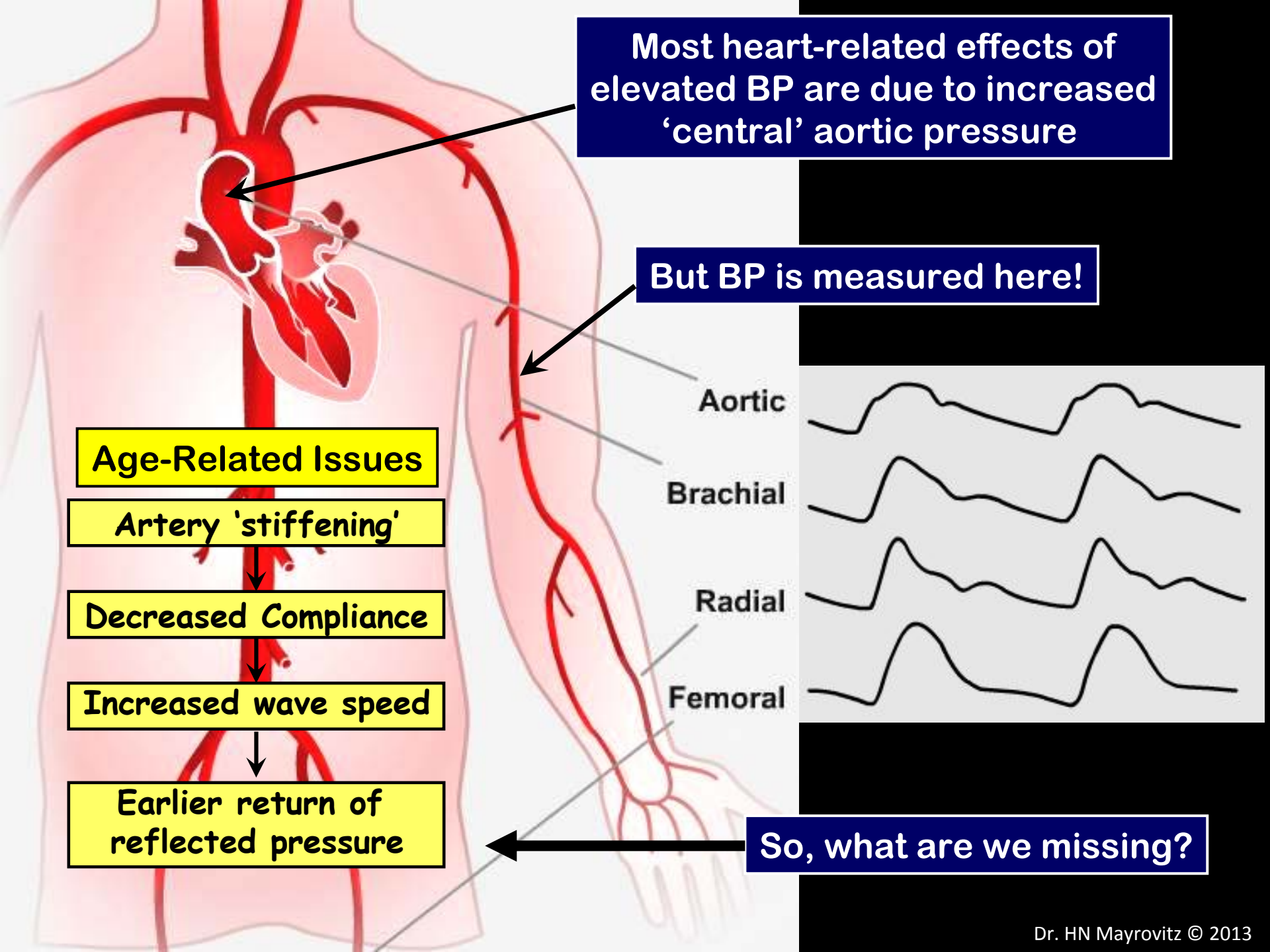




“Central Aortic Pressure”

Most heart-related effects of elevated BP are due to increased 'central' aortic pressure

But BP is measured here!



Age-Related Issues

Artery 'stiffening'

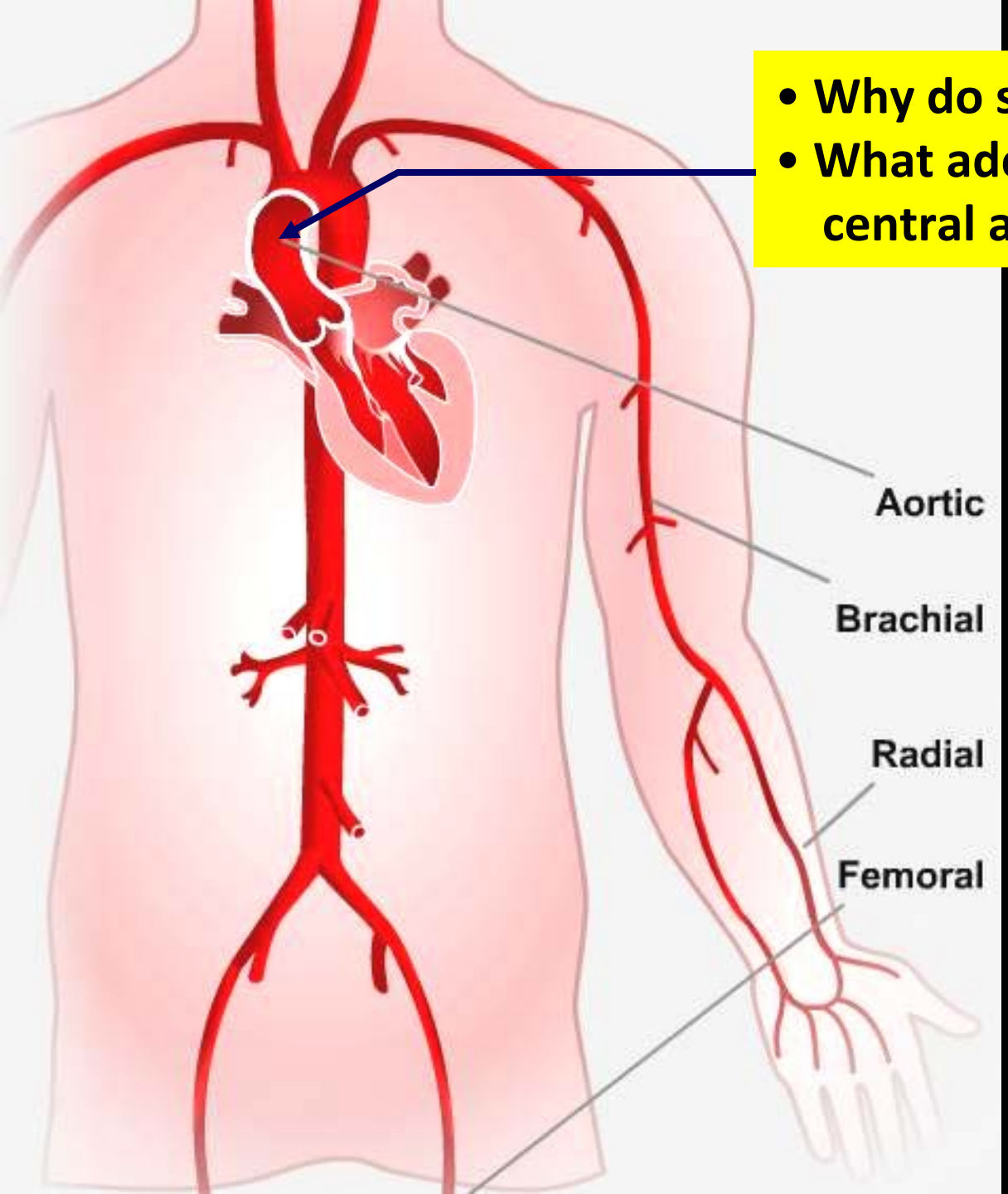
Decreased Compliance

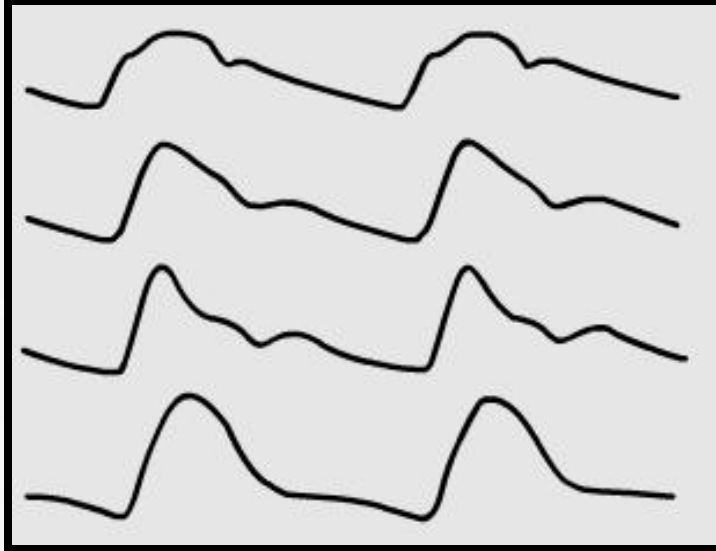
Increased wave speed

Earlier return of reflected pressure

So, what are we missing?

- Why do shapes & values differ?
- What adequately represents central aortic pressure?

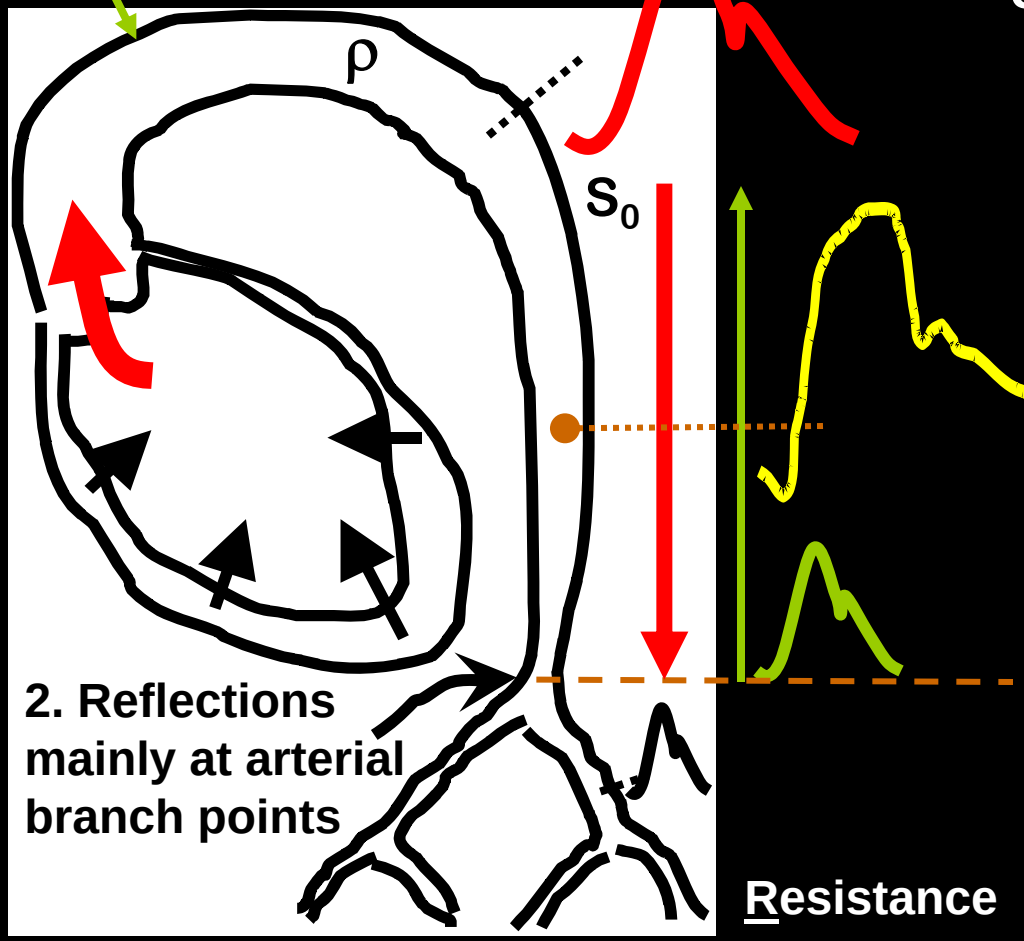




Values and Shapes Differ by site

Transmission and Reflection of Pulses

Transmission and Reflection of Pulses



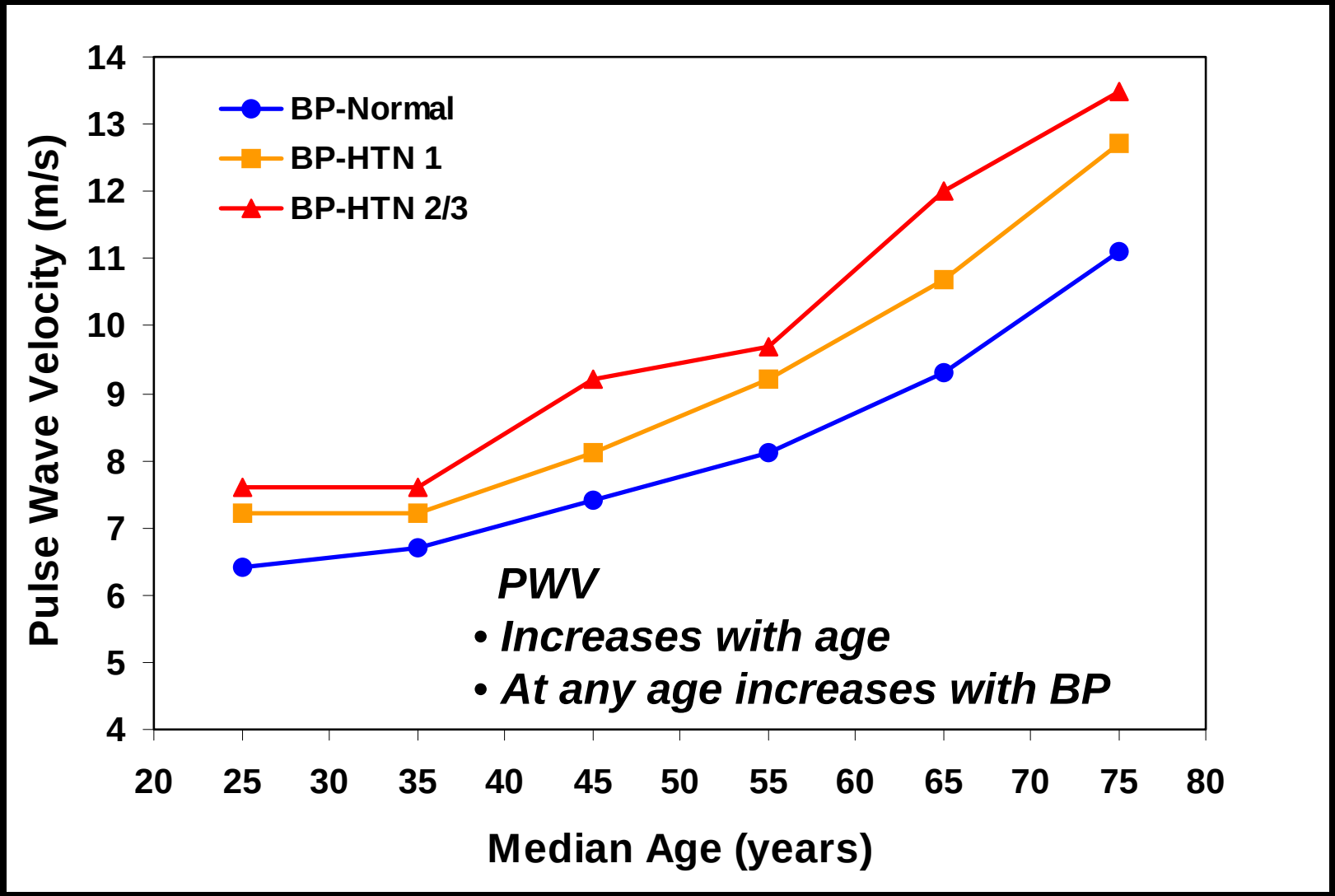
1. Pulse "wave-speed" (S_0) is inverse to Compliance (C)
Stiffer arteries ~ higher speed

$$S_0 \sim \sqrt{\frac{1}{\rho C}}$$

3. Pulses at any point in the artery are the algebraic sum of forward and reflected pulses

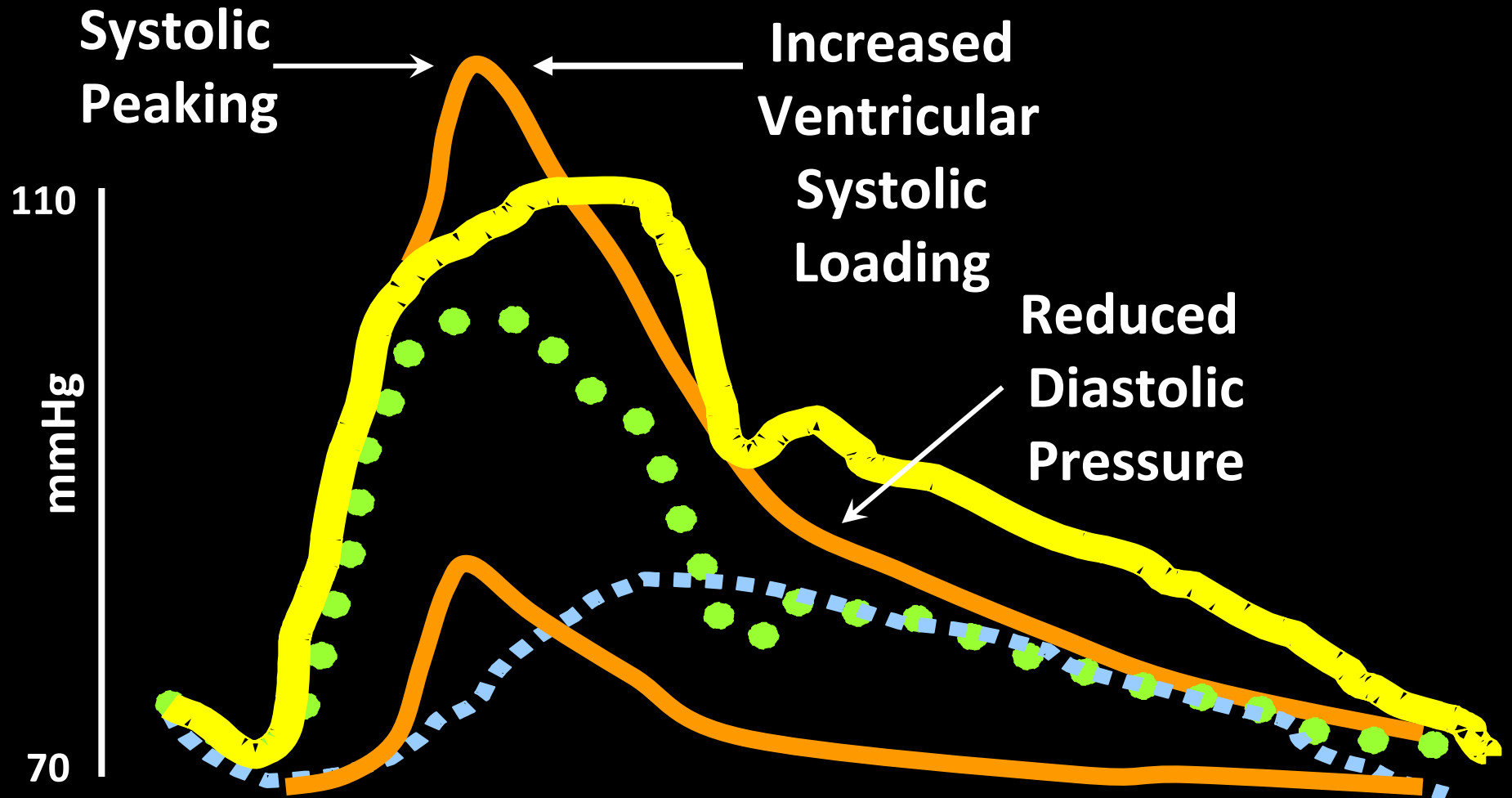
Composite is what is measured!

Pulse Wave Velocity (PWV)

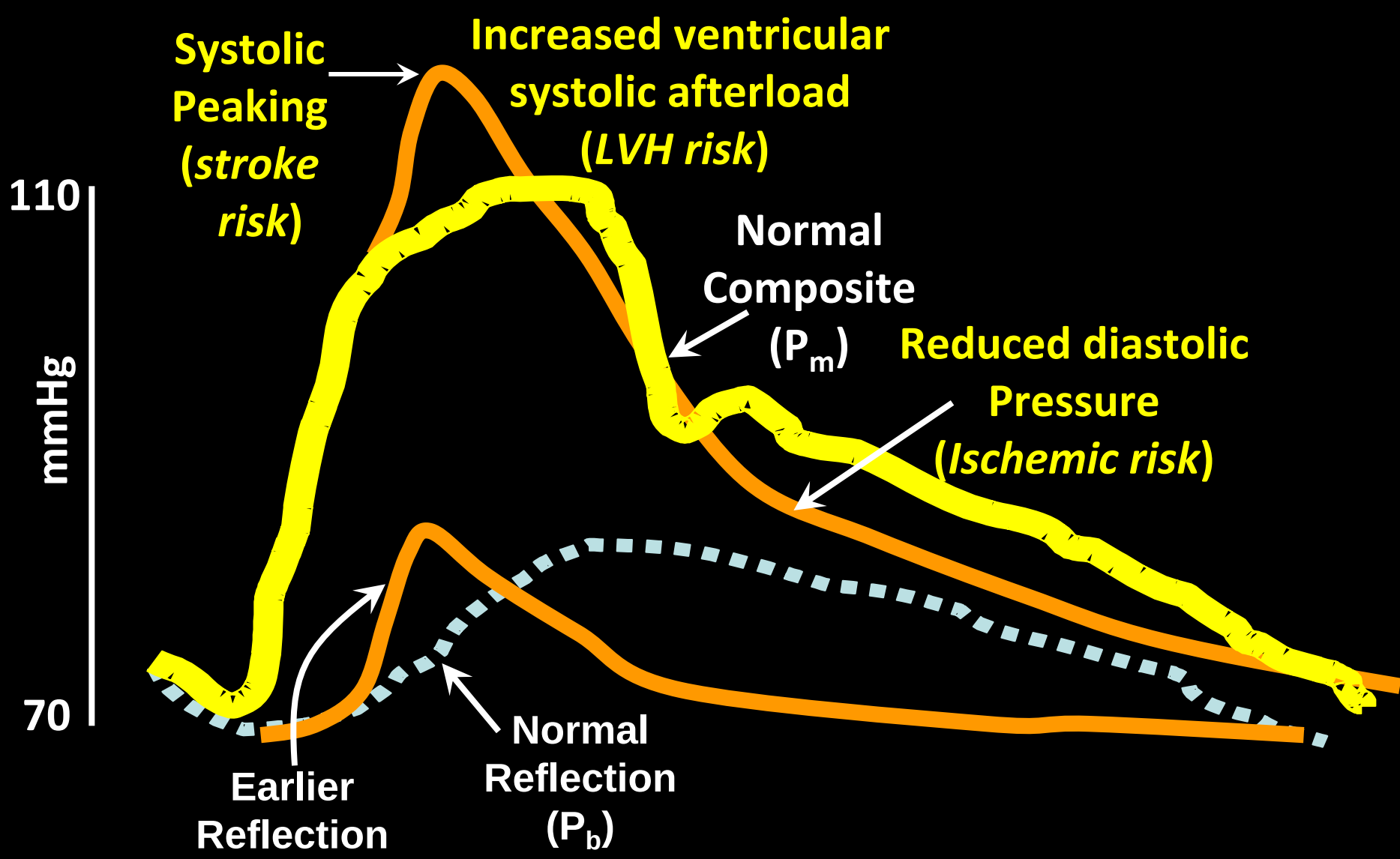


Adapted and Modified from European Heart Journal (2010) 31, 2338–2350

Earlier Reflection Arrival



Summary of Major Aspects



So – Standard BP by sphygmomanometry, though important and clinically useful only tells PART of the story

Aortic Central Pressure may be a more accurate risk assessment

Reasons have to do with pressure wave interactions that are most directly influenced by:

A. Pulse wave speed (Artery compliance)

B. Reflection amplitudes (Vasoconstriction state)

Both tend to increase with ageing and HTN

QUESTIONS?