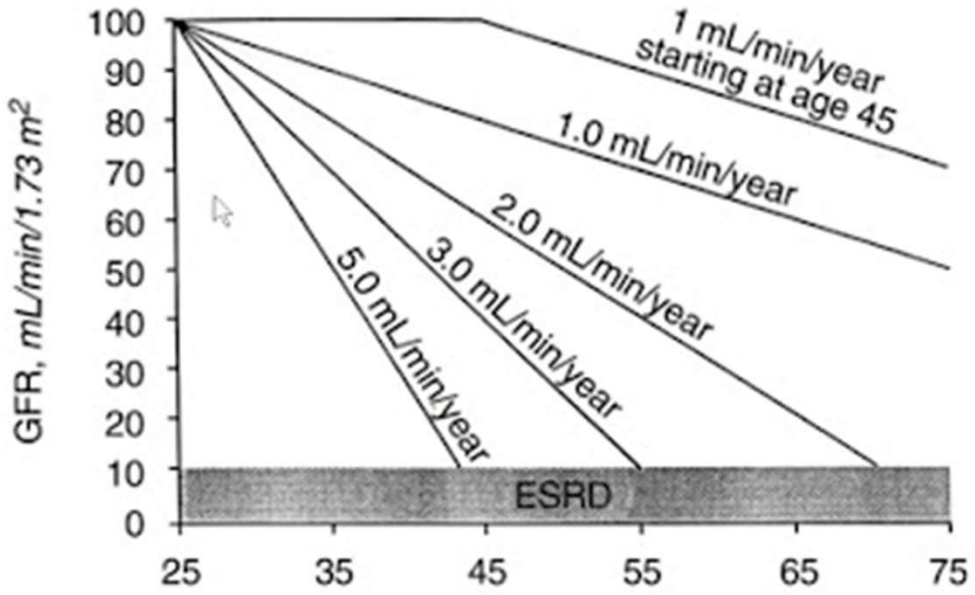
#5

Glomerular filtration rate (GFR) decline… the facts

Herbert et al *Kidney International* **59** (4) p1211



What's actually happening in "stable CKD 3a, like Grace's?

In many patients, CKD 3a (eGFR 45–59) with:

- minimal proteinuria
- stable function over years
- normal urine sediment
- controlled BP
- …is **not an active disease**. It's a **state**.
- What you're seeing is often:
- reduced nephron reserve, not ongoing injury
- kidneys that have aged, adapted, or been "used hard" earlier in life
- a new baseline rather than a trajectory toward failure
- Many of these patients **never progress**.

The End

- Questions?
- Comments?

Anti-hypertensives, Mechanism of Action:

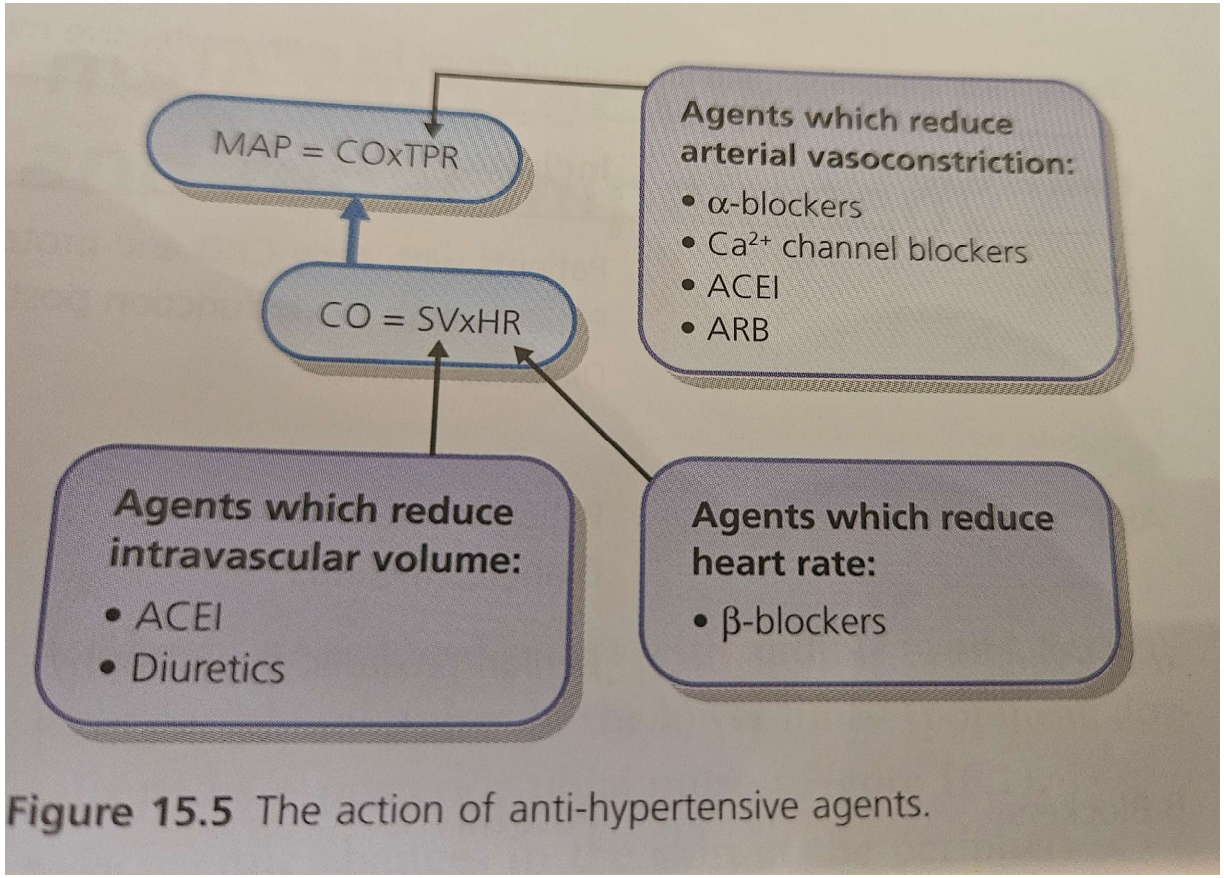


Figure 15.5 The action of anti-hypertensive agents.

RAAS blockade and kidney function

#Pro-tip

ACE inhibitors and ARBs can cause a reversible reduction in GFR when treatment initiated

If the reduction is less than 25% and stabilises within two months of starting therapy...
the ACE inhibitor or ARB should be continued

If the reduction in GFR exceeds 25% below the baseline value... the ACE inhibitor or ARB should be ceased and consideration given to referral to a Nephrologist for bilateral renal artery stenosis

Secondary accelerants

- Obesity amplifies injury via:
- RAAS activation
- Insulin resistance
- Adipokines (leptin, TNF-α, IL-6)
- Oxidative stress
- Low-grade systemic inflammation

→ Vascular and glomerular injury progress together