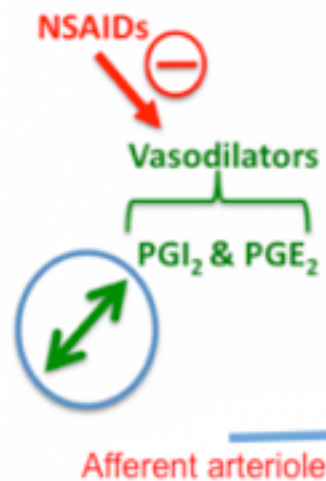


# NSAIDs and ACEIs/ARBs in renal function

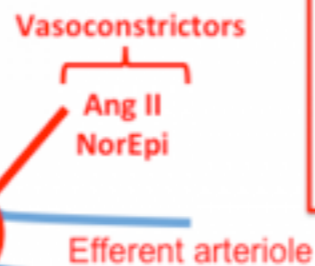
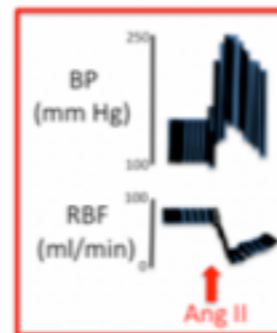
**NSAIDs can be harmful to the kidney:**

PGs are the major determinant of afferent vasodilation. By inhibiting PG production NSAIDs can cause afferent arteriole vasoconstriction & reduce GFR.



**ACE-I's are contraindicated in bilateral renal artery stenosis:**

Ang II is the major determinant of efferent vasoconstriction. The Ang II effect helps to maintain GFR when renal perfusion is low (e.g. bilateral renal artery stenosis, volume depletion & elderly patients with CHF). Blocking the effect of Ang II with ACE-I's & ARBs in these situations can cause acute renal failure.



[http://tmedweb.tulane.edu/pharmwiki/doku.php/ace\\_inhibitor\\_pharmacology](http://tmedweb.tulane.edu/pharmwiki/doku.php/ace_inhibitor_pharmacology)

- \* NSAIDs produce ↓ GFR (both non-selective and COX2 selective) by inhibiting vasodilator action of PGE<sub>2</sub> and PGI<sub>2</sub> on the afferent arteriole.
- \* At renal level, **ANG II constricts the efferent arteriole more than the afferent** resulting an increased perfusion pressure (↑ GFR). ANG II also stimulates PG E<sub>2</sub> and I<sub>2</sub> which dilates afferent arteriole and also ↓ GFR.
- \* **ACEIs/ARBs blocks ANG II effect --- >> ↓ GFR.** So pay attention when ↓ GFR like : **bilateral renal stenosis, volume depletion or elderly patients (GFR ↓ since only 1/3 of the nephrons are functional) since it may cause acute renal failure.**