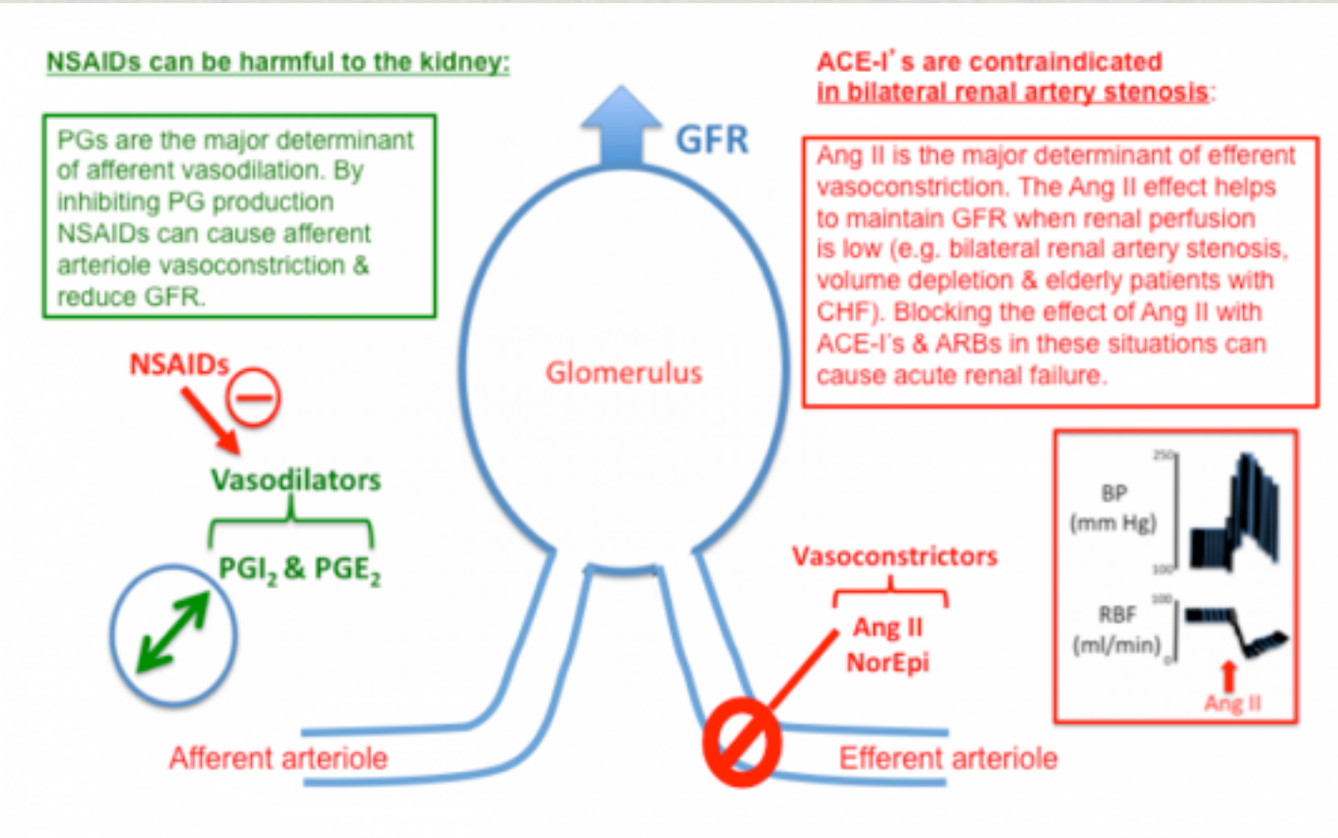
NSAIDs and renal function



http://tmedweb.tulane.edu/pharmwiki/doku.php/ace_inhibitor_pharmacology

	COX-1	COX-2
expression induced by	constitutive	inflammation
RENAL expression*	constitutive	constitutive
NSAIDs side effects	GI bleeding & ulcer ↓ GFR	↓ GFR

- Traditional nonsteroidal antiinflammatory drugs (NSAIDs) inhibit both isoforms of the enzyme cyclooxygenase (COX). The first, **COX-1**, is **constitutively expressed** in most cells throughout the body, and its inhibition has been associated with **gastrointestinal bleeding** and **ulceration**.

- In contrast, **COX-2 expression** is induced in the presence of **inflammation** and its inhibition results in the therapeutic effects of NSAIDs. Thus, the development of selective COX-2 inhibitors brought about a new way to produce potent antiinflammatory actions with a decreased risk of significant gastrointestinal adverse effects

- NSAIDs (which also **inhibit renal prostaglandin synthesis**) result in **afferent arteriole vasoconstriction** and that's why NSAIDs can cause a **reduced GFR** in patients who rely heavily on prostaglandin synthesis to maintain renal blood flow.

- Interestingly, however, the **kidney constitutively expresses COX-2,** and therefore **COX-2 inhibitors can lead to alterations in renal hemodynamics** similar to the NSAIDs **(reduced GFR)**

http://renalfellow.blogspot.com/2009/04/prostaglandin-basics.html