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# Contrast Induced Nephropathy

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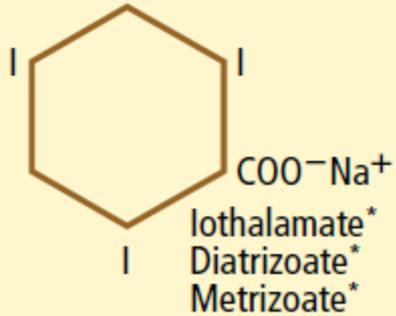
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- ▶ Contrast-induced nephropathy – a common form of hospital-acquired acute renal failure.
  - ▶ Incidence is low in patients with normal renal function, but much higher in those with severe renal insufficiency at baseline.
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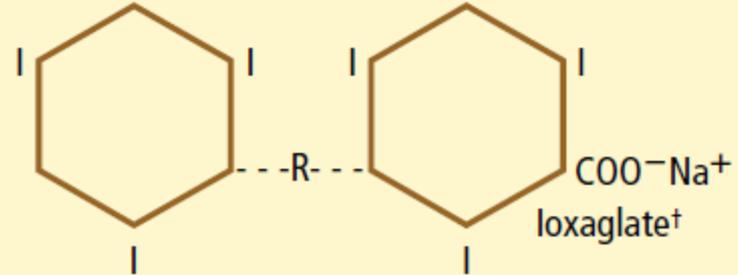
## Contrast agents

### MONOMERS

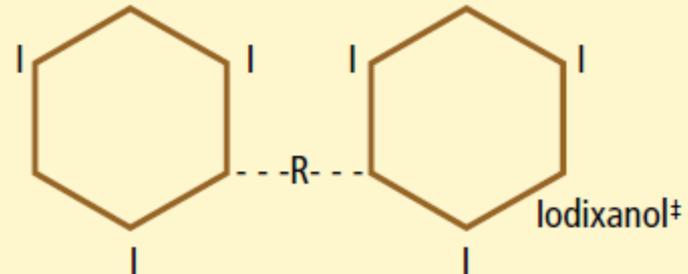
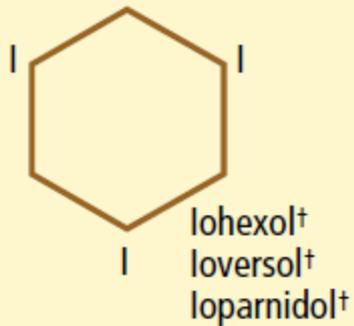
Ionic



### DIMERS



Nonionic



\*High-osmolar agents

<sup>†</sup>Low-osmolar agents

<sup>‡</sup>Iso-osmolar agent

ADAPTED FROM RUDNICK MR. THE ROLE OF OSMOLALITY IN CONTRAST-ASSOCIATED NEPHROTOXICITY. APPLICATIONS IN IMAGING—CARDIAC INTERVENTIONS. SCOTCH PLAINS, NY: ANDERSON PUBLISHING, 2003.

Patients undergoing percutaneous coronary interventions have a higher mortality rate if nephropathy develops

In most cases of contrast-induced nephropathy, serum creatinine begins to rise within 24 to 48 hours after exposure, reaches a peak within 3 to 5 days, and then returns to baseline levels within 7 to 10 days.



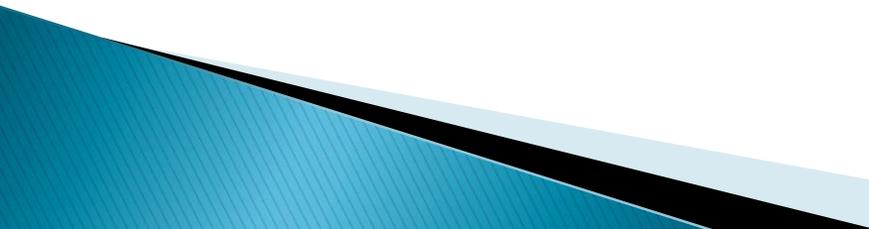
# RISK FACTORS

- ▶ **Preexisting renal insufficiency is the single greatest risk factor**
  - ▶ **Diabetes mellitus**
  - ▶ **Volume of contrast media**
  - ▶ **Multiple myeloma**
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# HOW DO CONTRAST AGENTS CAUSE NEPHROPATHY?

- ▶ by renal ischemia – after contrast is injected, renal blood flow transiently increases and then decreases over a longer time, suggesting that renal ischemia is a major factor in the pathogenesis of contrast induced nephropathy

- ▶ Contrast agents reduce the oxygen tension in both the cortex and the medulla.
  - ▶ This effect may be due to increased work of active transport in response to an osmotic diuresis from hyperosmolar agents, as well as the release of vasoconstrictive compounds such as endothelin
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- ▶ Hyperosmolality activates tubuloglomerular feedback or causes an increase in tubular hydrostatic pressures, either of which could lead to a decrease in GFR.
  - ▶ The osmotic diuresis produced by contrast media may result in increased active transport of sodium in the thick ascending limb and also in vasoconstriction
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- ▶ On the other hand, most studies in animals specifically comparing iso-osmolar contrast agents (iodixanol and iotrolan) with high-osmolar and low-osmolar contrast agents **have not** demonstrated any lower rate of renal abnormalities with the iso-osmolar agents.
  - ▶ Theory: they could increase red blood cell aggregation and decrease renal blood flow
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- ▶ **Reactive oxygen species**
  - ▶ **Direct cellular toxicity** (proximal cell vacuolization, interstitial inflammation, cellular necrosis, and enzymuria)
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## **Strategies for preventing contrast-induced nephropathy**

### **Strategies that do not work**

- Mannitol
- Furosemide
- Dopamine
- Atrial natriuretic factor
- Fenoldopam
- Hemodialysis

### **Strategies that may work**

- Calcium channel blockers
- Theophylline
- Iso-osmolar contrast media
- N*-acetylcysteine
- Hemofiltration
- Sodium bicarbonate
- Ascorbic acid
- Prostaglandins

### **Currently recommended strategies**

- Employ noniodinated contrast studies
- Avoid nonsteroidal anti-inflammatory drugs
- Provide adequate time between contrast procedures
- Minimize contrast volume
- Parenteral hydration
- Low-osmolar or iso-osmolar contrast media

# CURRENT RECOMMENDATIONS

- ▶ Nonsteroidal anti-inflammatory drugs should be discontinued before contrast exposure.
  - ▶ hydration with intravenous normal saline starting **2 to 4 hours before** receiving the contrast, during the radiographic procedure, and continuing **4 to 6 hours afterward**.
  - ▶ The duration of the saline infusion should be longer with more severe CKD or if underlying **diabetic nephropathy** is present.
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# CURRENT RECOMMENDATIONS

- ▶ Use the smallest volume of contrast needed to obtain the critical imaging.
  - ▶ There may be an advantage in using iso-osmolar contrast media.
  - ▶ Hypotension in the peri-imaging period should be avoided if possible.
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# CURRENT RECOMMENDATIONS

- ▶ *N-acetylcysteine and bicarbonate hydration* can be used since they are safe and inexpensive, although in somewhat controversial.
- ▶ Serum creatinine should be measured approximately 48 hours after contrast exposure to determine if contrast-induced nephropathy has occurred.