Acute Kidney Injury & CRRT
(Cardiorenal Syndrome)

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Outline

• Epidemiology of AKI in the CTICU
• Risk factors for AKI & what actually happens to the kidney after cardiac surgery
• Strategies and common medications involved in the AKI patient in the CTICU: can we ameliorate AKI?
• Summary and personal recommendations

The Wizard of Oz
(American Film Industry ranking #6 all-time)

Paradigm Shifts: Unlearning the Learned

• For many years (decades?), we were taught that if PRBCs were needed, transfuse 2 units
• Conversation w/ gen’l surgeon while moonlighting:
  – Me: “The patient needs PRBCs; I’d like to give a unit.”
  – Surgeon: “You should give 2 units.”
  – Me: “Why is that?”
  – Surgeon: “Because giving 1 unit is like spitting in the wind.”
  – Me: “Errr….OK....”
Paradigm Shifts: Unlearning the Learned

Published estimates of incidence vary by
- Definition of AKI (e.g., 30% increase in Cr, 50% increase in Cr, doubling of Cr...)
- Type of surgery (elective CABG << emergent CABG + valve surgery)
- Extreme ranges are 1% to 50%
- A more helpful number is 7-8% when a rise in Cr of ≥ 1 mg/dL is used
- "Roughly 10% have a Cr increase of 1" – more if your pop has lots of CKD

How Often is Dialysis Required?
- Evidence here clusters a bit more closely: 1-2% of all patients (not AKI patients)
- This would suggest that roughly 1/6 of AKI patients are “AKI-D”
- Adding valve surgery to CABG roughly triples the risk of AKI and AKI-D

Epidemiology of AKI in CTICU

How Does AKI Impact Mortality?
- Again, heavily dependent of AKI def’n and period studied (e.g., hospital D/C, 30-d survival, etc)
- 15-30% overall mortality with AKI is a good estimate
- Dialysis-requiring AKI (AKI-D) is ominous – 40-60%
- AKI-D increases mortality rate by ~ 8-fold (multivariable-adjusted)
- In-hospital mortality 1% (no AKI) vs 19% (AKI) vs 63% (AKI-D)

1 Rosner et al CJASN 1:19, 2006
3 Conlon et al NDT 14:1158, 1999

1 Conlon et al NDT 14:1158, 1999

1 Conlon et al NDT 14:1158, 1999
2 Stafford-Smith et al Curr Opin Crit Care 15:498, 2005
Slight Rises in Cr Impact Mortality

Long-Term Impact of AKI on ESRD

- There’s great interest in past few years about CKD and ESRD following AKI
- Not specific data in post-CPB patients – but good epidemiologic data on AKI as a whole

Long-Term Renal Impact of AKI

- The 2000 5% Medicare Beneficiary Sample was used to examine individuals ≥ 67 (n = 233,803)
- 2-yr F/U
- 5.3 per 1000 developed ESRD

Long-Term Renal Impact of AKI

<table>
<thead>
<tr>
<th>AKI and CKD</th>
<th>Hazard Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>both AKI and CKD</td>
<td>79.45</td>
</tr>
<tr>
<td>AKI only</td>
<td>24.52</td>
</tr>
<tr>
<td>CKD only</td>
<td>19.88</td>
</tr>
<tr>
<td>no AKI or CKD</td>
<td>2.08</td>
</tr>
</tbody>
</table>
The Kidney After Cardiac Procedures: AKI Risk Factors

Table 1. Risk factors associated with ARF

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Preoperative</th>
<th>Intraoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female gender</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Diabetes</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Peripheral vascular disease</td>
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<td></td>
<td></td>
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<tr>
<td>Renal insufficiency</td>
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<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
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<td></td>
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</tr>
<tr>
<td>LV ejection fraction &lt;35%</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Need for emergent surgery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic shock (IABP)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left main coronary disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Length of CPB</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cross-clamp time</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Off-pump time</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonpulsatile flow</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemolysis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemodilution</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1Rosner et al CJASN 1:19, 2006

The Kidney After Cardiac Procedures: Pathophysiology

Days

Vasomotor nephropathy | ATP | Vascular injury | Obstruction | Inflammation | Coagulation |

<p>| | | | | |</p>
<table>
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<tr>
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</thead>
<tbody>
<tr>
<td>Vasomotor nephropathy</td>
<td></td>
<td>Vascular injury</td>
<td>Obstruction</td>
<td>Inflammation</td>
</tr>
<tr>
<td>ATP</td>
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</tbody>
</table>

1Rosner et al CJASN 1:19, 2006

Risk Stratification: The Cleveland Clinic AKI Scoring System

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female gender</td>
<td>2</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>2</td>
</tr>
<tr>
<td>LV ejection fraction &lt;35%</td>
<td>2</td>
</tr>
<tr>
<td>Preoperative use of IABP</td>
<td>2</td>
</tr>
<tr>
<td>COPD</td>
<td>1</td>
</tr>
<tr>
<td>Insulin-requiring diabetes</td>
<td>1</td>
</tr>
<tr>
<td>Previous cardiac surgery</td>
<td>1</td>
</tr>
<tr>
<td>Emergency surgery</td>
<td>2</td>
</tr>
<tr>
<td>Valve surgery only (reference to CABG)</td>
<td>1</td>
</tr>
<tr>
<td>CABG + valve (reference to CABG)</td>
<td>1</td>
</tr>
<tr>
<td>Other cardiac surgeries</td>
<td>2</td>
</tr>
<tr>
<td>Preoperative creatinine 1.2 to &lt;2.1 mg/dl (reference to 1.2)</td>
<td>2</td>
</tr>
<tr>
<td>Preoperative creatinine &gt;1.2</td>
<td>5</td>
</tr>
</tbody>
</table>

1Rosner et al CJASN 1:19, 2006

1Thakar et al JASN 16:162, 2005
Timing of Events in AKI

- When to call the nephrologist
  - No consensus; nephrologists argue
  - If calling when dialysis needed, too late (in an ideal scenario)

Timing of Events in AKI: Concepts

- When dialysis is commenced?
  - No consensus; nephrologists argue
  - From 2000-2010, trend was to start early (BUN > 40 mg%)”
  - BUN alone is frequently the least important metric
  - K > 5.0, pH < 7.2, rising vent support (>60% FIO2), 6+ hours of anuria warrant a consult in most instances
  - Increased vent support: though many insults to gas-exchange are not due to volume overload, a dialyzer allows you to address what you can
  - What is overall direction of the patient? (“If they are going to be worse tomorrow then they are today, start today”)

Dialysis: Conceptual Issues

- Modalities:
  - “conventional” intermittent hemodialysis (IHD)
  - slow low efficiency extended dialysis (SLED)
  - continuous (CRRT)

- Conceptualization of dialysis is important for non-nephrologists
  - Clearance (BUN and other toxins)
  - Electrolyte & acid-base control
  - Ultrafiltration

Off- vs. On-Pump CPB

- CABG, but not valve replacement, can be done off-pump (OP)

- Potential benefits of OP-CABG
  - Less inflammation, oxidative stress, and C’ activation induced by the pump

- Potential debits of OP-CABG:
  - Greater hemodynamic instability
**Off- vs. On-Pump CPB**

- Largest study was an RCT of 2200+ patients
- Vast majority of patients w/o CKD
- AKI not an endpoint, but dialysis requirement was
- No difference in requirement for HD
- Most metanalyses reflect this; more studies with CKD patients are required

Shroyer et al NEJM 361, 1827, 2009

**Medications Involved in AKI**

- NAC (N-acetylcysteine)
- Dopamine
- Fenoldopam
- ANP (atrial naturetic peptide)
- Lasix

**Preoperative NAC**

- The evidence for N-acetylcysteine is extremely variable
- >24 studies and ≥ 3 metaanalyses have been performed
- A consensus around proof of efficacy simply has not emerged
- Even metaanalyses differ in their findings!
Perioperative NAC

- Most promising evidence is for high-dose NAC (1200, vs 600, mg), particularly in IV form
- One study\(^1\) showed benefit of ↓ AKI with 1200 mg IV before emergent angioplasty for MI
- Another\(^2\) showed decreased circulating oxidative stress markers with high-dose oral (no AKI benefit)
- Oral NAC is considered “harmless” but even oral NAC is associated with anaphylaxis, rates are much higher in IV

\(^1\)Marenzi et al NEJM 354:2273, 2006
\(^2\)Thiele JACC 55:2201, 2010

Role for Dopamine

- At low doses, dopamine (1-3 ug/kg/min) increase renal blood flow in experimental models and increases naturesis/diuresis (\(\beta_1\) at 5-10, \(\alpha_1\) at 10-20)
- This does not appear to be the case in humans; it seems to have a pressor effect at <5.
- Studies repeatedly show that is not effective in reducing AKI\(^{1-3}\)

\(^1\)Denton et al KI 50: 4, 1996
\(^2\)Tang et al Eur J CT Surg 15:717, 1999
\(^3\)Woo et al Eur J CT Surg 22:106, 2002

Dopamine is Arrhythmogenic

- Dopamine is not benign
- A retrospective registry study\(^1\) of 1700+ CABG patients found an OR of 1.74 for Afib or Aflutter
- An older study showed Dop. was most signifi. Afib predictor\(^2\)

\(^1\)Argalious et al CCM 33:1327, 2005
\(^2\)Chiolero Thor CV Surg 39:81, 1991

Fenoldopam

- Long-studied with both animal and human evidence suggesting higher eGFR but no major change in AKI incidence w/ the drug
- Best RCT\(^1\) so far tested fenoldopam in 193 patients with ≥1 risk factor
  - Cr >1.5 mg/dL
  - Age >70 y
  - DM
  - Previous CABG
- 0.1 \(\mu\)/kg/min vs. placebo as incision was made

\(^1\)Cogliati et al J CT Vasc Anaesth 21: 847, 2007
Fenoldopam

- Fenoldopam group had less AKI ($p = 0.02$), defined as $\text{Cr} \geq 2.0$, and less need for HD ($p = 0.004$)

<table>
<thead>
<tr>
<th></th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fenoldopam group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine clearance (mL/min)</td>
<td>38.7 ± 7.2</td>
<td>68.1 ± 14.2</td>
<td>66.4 ± 16.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Urine output (mL/H)</td>
<td>—</td>
<td>156 ± 56</td>
<td>110 ± 38</td>
<td>NS</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>1.6 ± 0.4</td>
<td>1.8 ± 0.2</td>
<td>1.5 ± 0.3</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Fluid intake (mL/H)</td>
<td>—</td>
<td>120 ± 10</td>
<td>115 ± 40</td>
<td>NS</td>
</tr>
<tr>
<td>Plasma group</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Creatinine clearance (mL/min)</td>
<td>46.7 ± 12.5</td>
<td>39.6 ± 5.3</td>
<td>33.7 ± 11.2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Urine output (mL/H)</td>
<td>—</td>
<td>110 ± 47</td>
<td>82 ± 32</td>
<td>NS</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>1.9 ± 0.3</td>
<td>2.5 ± 0.8</td>
<td>2.8 ± 0.4</td>
<td>&lt;0.003</td>
</tr>
<tr>
<td>Fluid intake (mL/H)</td>
<td>—</td>
<td>120 ± 25</td>
<td>126 ± 34</td>
<td>NS</td>
</tr>
</tbody>
</table>

NOTE: Data are given as mean ± standard deviation.
Abbreviations: T1, before surgery; T2, 24 hours after surgery; T3, 48 hours after surgery; NS, not significant.

A "definitive" RCT of 1000 patients is currently underway (NCT00621790)


ANP: The Most Promising Intervention to Date

- Peptide made by atria; there is a human recombinant form (hANP)
- Previous small hints of promise; approved in Japan for AKI prevention
- Three fairly recent RCTs1-3 and a metaanalysis4 have recently all suggested benefit
- The best one in CABG patients3 involved randomizing 504 patients to hANP at 0.02 μg/kg/min

1Mentzer et al JACC 49:716, 2007
2Sezai et al JACC 54:1058, 2009
3Sezai et al JACC 55:1844, 2010
4Nigwekar et al J CT Vasc Anaesth 23:151, 2009

ANP in CABG for AKI Prevention

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<tbody>
<tr>
<td></td>
<td>hANP</td>
<td>Placebo</td>
<td></td>
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</tbody>
</table>

Lasix Mechanism

- Lasix works on the Na/K/2Cl pump in the thin ascending limb
- This pump is very metabolically active; theoretically, poisoning it could ↓ metabolic demands

Sezai et al JACC 54:1058, 2009
Lasix Decreases Time on Ventilator

- RCT of 1000 patients randomized to “liberal” vs. “conservative” fluid management in the MICU
- The conservative group (naturally) got much more diuretics
- At 7 days, conservative group was ~ -100 cc fluid balance; the liberal (“traditional”) group was +7 L
- No difference in mortality

NHLBI ARDSNet NEJM 354:2564, 2006

Lasix Decreases Time on Ventilator

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Conservative Strategy</th>
<th>Liberal Strategy</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death at 60 days (%)</td>
<td>25.5</td>
<td>28.4</td>
<td>0.30</td>
</tr>
<tr>
<td>Ventilator-free days from day 1 to day 28</td>
<td>14.6±0.5</td>
<td>12.1±0.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ICU-free days†</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Days 1 to 7</td>
<td>0.9±0.1</td>
<td>0.6±0.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Days 1 to 28</td>
<td>13.4±0.4</td>
<td>11.2±0.4</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Volume Overload May Be Associated with Mortality

- Fluid overload may result in endothelial injury, volume overload, and other deleterious events in critical illness
- An observational study of >600 patients at 5 AMCs assessed risk of fluid overload in AKI and AKI-D

Volume O/L May Be Assoc. w/ Mortality

- a, dialyzed
  OR = 2.1

- b, non-dialyzed
  OR = 3.1

Bouchard et al KI 76:422, 2009

1Prowle et al Nat Rev Nephrol 6:107, 2010
2Bouchard et al KI 76:422, 2009
Lasix in AKI: The Nuances

- Non-oliguric AKI has a better prognosis than oliguric/anuric AKI
- This does not mean that “converting” a patient to a better state improves AKI or other outcomes.
- 3 older trials all showed no difference in outcomes with lasix\textsuperscript{1,2,3}

\textsuperscript{1}Kleinknecht et al Nephron 17: 51, 1976
\textsuperscript{2}Brown et al Clin Nephrol 15:90, 1981
\textsuperscript{3}Shilliday NDT 12:2592, 1997

Lasix May Be Harmful

- 126 cardiac surgery patients with normal renal function randomized to lasix, dopamine, or placebo

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{figure1}
\caption{Lasix May Be Harmful}
\end{figure}

Lasix and Forced Euvolemic Diuresis

- Perhaps it is throughput of the kidney, not diuresis per se, that is helpful
- This was tested in an RCT of 138 patients getting contrast w/ cardiac cath\textsuperscript{1}
- All patients had CKD (Cr \geq 1.7), and were assigned saline alone vs (saline + mannitol + lasix)
- Diuresis group matched mL-for-mL
- Outcome: 25% increase in Cr within 48 hrs

\textsuperscript{1}Majumdar AJKD 54:602, 2009
Lasix and Forced Euvolemic Diuresis

- Trial terminated for futility after < 50% enrollment
- AKI occurred in 50% in intervention arm vs. 23% in controls ($P = 0.03$, adj. OR 3.7)
- Within hours, Cr increase 0.8 in intervention arm vs. 0.2 in controls

Lasix: Summary

- The kidney is smarter than any doctor
- While lasix will decrease vent/ICU days, it will not help in AKI prevention; if anything, it is likely to be harmful in at-risk kidneys
- Lasix should be used to protect the cardio-pulmonary status; there is no evidence for any renoprotective effects

What Information Can the ICU Nurse Provide for the Nephrologist?

- Hemodynamic trends
- Filter performance
- Citrate anticoagulation

Hemodynamics

- Nephrologists, as consultants, generally work with primary physicians on UF goals
- Nephrologists sometimes have a differing perspective
  - “Is that infiltrate on CXR really pulmonary edema? We’ll do what we can, but…”
  - “We can pull fluid off, but are you prepared to support the patient with pressors? Are you prepared to treat the consequences of a tachyarrhythmia?”
- When asked to aggressively UF, nurse input is critical

*Majumdar AJKD 54:602, 2009*
Hemodynamics

• Discussion with the nurse about the (hemodynamic) course of the patient is essential
  – What is the trend in BP, HR? -- timecourse of interest is minutes to hours
  – What is trend in other pressors or cardioactive meds (CCU)?
  – What is overall trend in patient’s course (better/worse/same)?
  – What events are known to be planned for the day (OR or procedure, extubation, etc)?

Hemodynamics

• As UF rate is increased (say, 100cc/hr to 150cc/hr), BP/HR must be carefully watched
• At the first sign of a true trend in BP or HR, nephrologist should be notified
• While not rigorously studied, a UF “holiday” of perhaps 4 hrs is probably appropriate – i.e., no UF to allow for vascular refill, followed by a re-challenge with UF
• If the vent support is high, or if the primary team accedes to increase in pressors, UF holiday is probably not optimal course

Filter Performance: Clotting and Clogging

• Clotting causes filter failure due to obstruction of the membrane pores from the molecular products of the clotting cascade
• Clogging is an entity – at least theoretical and perhaps of clinical significance – in which inflammatory mediators degrade filter performance
  – Some claim this process to be beneficial since these mediators are components of SIRS

Filter Performance

Note that the delta P should be recorded at the beginning of the filter life and at other “natural” intervals, like beginning of a shift.

If the delta P increases beyond 100 (a delta-delta P!) this suggests clotting is occurring

A rise in TMP (which should be <450) suggests clogging
Citrate: Special Challenges

- Citrate is used to chelate Ca\(^{2+}\), a component of the clotting cascade, in the circuit
- Citrate is introduced prefilter, depleting Ca locally
- A separate Ca\(^{2+}\) infusion (thru another central line) is used to reverse this
- Blood flow rate affects citrate rate, and citrate rate affects Ca\(^{2+}\) rate — true cascade

Citrate: CRRT Prescription Cascade

b. Infuse at ________ mL/hr (initial at 1.5 times BFR, not to exceed 300 mL/hr unless ordered by Nephrologist).
   (Example: BFR is 180 mL/min. The AC3-A infusion would be started at 1.5 x 180 = 270 mL/hr)

c. Titrate citrate AC3-A infusion rate per parameters (see hyperlink) using post-filter (blue port) ionized calcium.

d. Stop BOTH Citrate and Calcium infusions any time the blood pump is not running including when the filter clots and when CRRT is discontinued.

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Citrate: Calcium and Citrate Adjustments

<table>
<thead>
<tr>
<th>Systemic ionized Calcium (mmol/L)</th>
<th>Citrate Infusion Adjustment</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.85</td>
<td>Rate 1 mL/hr, clamp to 1 g/1 L 1g/1 L calcium acetate (1 mL calcium acetate in 1 L)</td>
</tr>
<tr>
<td>0.81 – 0.84</td>
<td>Rate 1 mL/hr &amp; clamp to 1 g/1 L calcium acetate</td>
</tr>
<tr>
<td>0.8 – 0.80</td>
<td>Rate 0 mL/hr</td>
</tr>
<tr>
<td>0.79 – 1.20 (Optimum Range)</td>
<td>No change</td>
</tr>
<tr>
<td>0.71 – 1.35</td>
<td>1 mL/hr</td>
</tr>
<tr>
<td>0.65 – 0.70</td>
<td>2 mL/hr - consult lead nurse</td>
</tr>
</tbody>
</table>

Citrate: Special Challenges

- A patient in steady state should require only a few dose changes — perhaps 3 — of Ca\(^{2+}\) before equilibration
- If the systemic ionized Ca\(^{2+}\) is, say, 0.93 mmol/L, this doesn’t look too bad at first glance
- Nurse gives an amp and increases rate by 10 mL/hr
Citrate: Special Challenges

- 1st reading: 0.93 → amp given and rate increased
- 2nd reading: 0.92 → amp given and rate increased
- 3rd reading 0.94 → amp given and rate increased

- This is a warning sign! The Ca\(^{2+}\) is going somewhere!
- The nephrologist should be made aware that by the end of the first shift, steady-state has not been achieved
- The nephrologist should ask – but the nurse should tell

“Citrate Lock”

- Anion-gap metabolic acidosis
- Persistently low systemic (patient) ionized Ca\(^{2+}\), refractory to calcium boluses
- A rising total serum calcium.

- Typically seen in patients with liver failure (citrate not metabolized to bicarbonate, so citrate is binding ionized Ca\(^{2+}\) -- while total Ca\(^{2+}\) increases as it is dumped in
- Citrate must be held or ceased

Other Citrate Complications

- Alkalemia can increase by the citrate infusion
- ABG or VBG will show steady rise in pH – 7.45, 7.50, even 7.60 over 6-12 hours
- Nephrologist should be ordering extra VBGs on citrate patients – ICU teams won’t be thinking of this
- Nephrologist should ask for gases – but nurse should tell!

Other Citrate Complications

- Long-term, changing protocols to use less citrate while maintaining anticoagulation is the answer
- Short-term, holding citrate for several hours then resuming citrate at 70% of previous rate is appropriate
Lowering the Risk of Postoperative AKI: Personal Recommendations

- For non-emergent procedures, hold diuretics and ACE-I/ARBs for 2-3 days prior and thereafter (admittedly, not possible in decompensated CHF)
- Hydration with IVF (Na bicarb preferred) for several hours before in CKD patients (eGFR < 60 mL/min); no fluid if CKD not present (to reduce dangers of volume O/L)
- Use diuretics to protect cardiopulmonary status only; do not give solely for oliguria, rising Cr, etc.
- Fluid challenge certainly reasonable in oliguria if pulm status will tolerate
- Follow the literature on ANP, but dopamine and fenoldopam have no demonstrated utility

Thanks for your attention!

Questions?

AFI top-ranking movie list (by critical acclaim)
#6 Wizard of Oz (Judy Garland)
#5 Lawrence of Arabia (Peter O'Toole)
#4 Gone with the Wind (Clark Gable)
#3 The Godfather (Marlon Brando)
#2 Casablanca (Humphrey Bogart)
#1 Citizen Kane (Orson Welles)