Interventional Cardiology
Contrast and Post Cath Complications

Reading can include from texts:
Grossman and Baim
Kern
Reviews on contrast in *Rev Cardiovasc Med*. 2003;4 Suppl 5 (free on internet)
But I suggest you focus on the ACC/AHA guidelines: ACC/AHA Coronary Angiography Guidelines. JACC, 1999; 33: 1756

Contrast:

Contrast agents were introduced to radiology between in the early 1900's[21]. Later production of contrast compounds which were ionic and high osmolar compounds, (> 1,900 mOsm/kg H_2O) were the leading agents with angiography[21]. These agents provided visualization, but contained calcium binding additives, such as ethylenediamine tetra-acetic acid or sodium citrate[22]. These calcium binding additives had associated risks including bradycardia, prolong QT interval, ventricular arrthymias or hypotension [22]. Furthermore, when these agents were injected directly into a coronary artery, a variety of arrythmias have been documented including bradycardia, heart block, sinus arrest, sinus tachycardia, ventricular tachycardia or fibrillation [22]. The development of non ionic and low osmolar contrast agents, (600 mOsm/kg H_2O) had a lower incidence of cardiac arrythmias. A clinical randomized trial found significantly less people with rise of creatinine > 1mg/mL in those who received the low osmolar agent iohexol compared to the high osmolar agent diatrizoate[23]. Decreasing levels of renal function act as a major adverse prognostic factor after contrast exposure, with or without percutaneous coronary intervention[1-4]. The etiology is considered to have multiple etiologies, including direct tubular toxicity, ischemia and oxygen free radical formation [5]. The diagnosis of CIN can be made 5 days out from angiogram. Various strategies for the prevention of CIN have been investigated[6], with some detrimental[7] and some neutral to kidney function[7,8]. Clinical trials have reported that intravenous hydration with normal saline[9], N-acetylcysteine [10-12], the iso-osmolar contrast agent iodixanol[13,14], hemofiltration [15], sodium bicarbonate [16]and limiting the volume of contrast [17] may help prevent CIN. Although reduced, CIN was still a significant problem with the low osmolar contrast agents. One study of 59 patients with diabetes mellitus and severe kidney disease, found the use of greater than 30 mL was associated with a significantly higher incidence of CIN[17]. The correlation of contrast volume to worsening kidney function reinforced the need for delaying or staging of percutaneous intravascular procedures after diagnostic angiography.

The next generation of contrast agent, iodixanol, was non ionic and iso-osomolar (290 mOsm/kg H_2O)[21]. Two randomized trials found a significant reduction in worsening of kidney function with use of iodixanol, compared to the low osmolar non ionic contrast agent iohexol[13,14]. An argument has been made there is no clear difference in the incidence of CIN between low osomolar and iso osmolar contrast agents [24]when comparisons are made between the trials. The concern in comparing the incidence of CIN between trials is variation in the definition of CIN, some using >25% from baseline [15,16], others use change in creatinine >0.5mg/dL [10,25]or >1.0mg/dL [23]or a combination of either >25% from baseline or >0.5mg/dL[26]. The strongest risk factor appears to be

preexisting kidney disease, but diabetes, hypertension, intra aortic balloon pump, intravascular depletion, high osmolar contrast agents, cholesterol emboli, multiple myeloma in a dehydrated patient, concomitant use of nephrotoxic agents, congestive heart failure or large volume of contrast are also associated with CIN[27,28]. Recommendations now include choice of contrast agent, hydration, oral N-acetylcysteine, limiting the amount of contrast and allowing adequate time between multiple contrast exposures[27,28].

Basic points:

1. Who are at risk
    a. DM, old age, NSAIDS use, multiple myeloma, heart failure, hypotension, use of intra aortic balloon pump
2. How to prevent kidney injury
    a. Iso-osmolar better than low osmolar better than high osmolar contrast
    b. N-acetylcysteine probably works, especially for diabetes with severe kidney disease (GFR < 30mL/min).
    c. Normal saline fluids
    d. Stop offending medications
    e. Careful on removing catheters, especially larger French amplatz or JL4
    f. Low volume of low osmolar contrast, < 30mL
    g. Use of $NaHCO_3$ with low osmolar contrast
3. Contrast agents available:
    a. High-osmolar agents
    b. Low-osmolar agents
    c. Iso-osmolar agents
    d. Gadolinium (poor visualization)
    e. Carbon Dioxide (poor visualization)

Post Cath Access Site Complications

Grossman and Baim Catheterization text, chapter on complications

1. Post cath important to do the following:
    a. Discuss with the patient restrictions with sheath in place, immediately after sheath removed and for next 2-5 days (depending on sheath size and location)
        i. Lying flat with leg straight that was used for access for set time based on arterial sheath size: 4F – 2 hours; 6F – 6 hours; 8F – 8 hours
        ii. Keeping wrist straight with brace, with arm sling for 24 hours (variable)
        iii. No lifting heavy objects for 5 days (not been tested)
        iv. No driving for 2 days (not been tested)
        v. No sex for 2 days (again, not tested, but a fun IRB proposal)

   b. Examine the patient post cath same day and following AM regardless of presence of sheath:
      i. Access site for hematoma, echymosis, mass or bruit
      ii. Distal pulse(s) and skin, looking for signs of cholesterol embolization
      iii. ECG if post PCI
      iv. IABP pressures are appropriate and pt. on anticoagulation unless contraindicated
   c. Following AM post PCI:
      i. Review restrictions in (a) above
      ii. Review any new medications
      iii. Review follow up blood test eg. serum creatinine 3-5 days after angiogram if pt has kidney disease and not on dialysis
      iv. Review Lab tests for troponin, serum creatinine
2. Complications to be aware of post cath:
   a. Pseudoaneurysm – diagnosis with ultrasound, management with: nothing if small < 2cm and follow with serial ultrasound; compression with hand; ultrasound probe or injection with thrombin.
   b. AV fistula – diagnosis with ultrasound. Management majority of cases do nothing, follow serial ultrasound or surgery.
   c. Bleeding – retroperitoneal, rectus sheath, deep femoral (exacerbated by thrombocytopenia from 2b3a inhibitor), rare source of bleeding (eg. pulmonary hemorrhage due to 2b3a inhibitor)
   d. Cholesterol embolization – think of abdominal pain, rash on extremities – biopsy, urinalysis to confirm diagnosis. Treatment is palliative. Start statin and prevention is key.
   e. Cerebral Vascular Accident/Transient ischemic event – rare, but can be secondary to catheter manipulation around the arch without use of wire; dislodgement of thrombus from left ventricle or left atrial appendage (as with ASD closure). Prevention is key – understanding the risks and explaining them to the patient prior to starting the procedure. Treatment may include
   f. Contrast nephropathy (see above)
   g. Vascular compromise
   h. Infection of groin site – very rare unless place device that is non absorbable – pacemaker, sutures from perclose device in femoral artery to close arteriotomy from sheath…etc.
   i. Nerve compression – generally related to local anesthetic, but can be secondary to bleeding or pseudoaneurysm. Symptoms are generally numbness in the extremity, but can be rarely associated with muscle weakness.
   j.