Pre and Post Cardiac catheterization and Intervention 2011

- Thanavit Sakulsaengprapha MD.
- Division of Cardiology
- Department of Medicine
- Bhumibol Adulyadej Hospital

Indications for Cardiac catheterization

- 1. Need to choose among therapeutic options
 - Revascularization ACS, Angina, CAD high risk
 - Valve replacement/repair
- 2. Obscure or confusing clinical picture
 - -CAG in uncertain origin of chest pain
- 3.Research study

Indication for coronary angiography

ASYMPTOMATIC

- CLASS 1
 - EVIDENCE FOR HIGH RISK PATIENTS ON NONINVASIVE TESTING
 - INDIVIDUALS IN HIGH RISK OCCUPATIONS(AIR PILOT)
 - successful CPR
- CLASS II
 - ETT +
 - multiple risk factors for CAD
 - prior MI with ETT +
 - following cardiac transplantation
 - after CABG or PTCA with +ischemia
 - before noncardiac surgery with + ETT

symptomatic patients

- Class I
 - INADEQUATE RESPONSE TO MEDICAL TREATMENT
 - UAP
 - VARIANT ANGINA
 - CANADIAN CVS SOCIETY FC I-II
 - +ETT
 - history MI or hypertension with ECG change
 - side effect of medical treatment
 - need to know
 - episodic pulmonary edema
 - before major vascular surgery
 - after CPR
- class II
 - any angina in the following groups
 - female <40 yrs with + ETT
 - male <40 yrs
 - patients requiring major nonvascular surgery
 - class II-IV angina that improves on medical treatment
 - patients who can not be risk stratified by other techniques

Indications for CAG in planning revascularization procedures

- SAP
 - CAG preferred method for stratifying risks and clarifying therapeutic alternatives in these group of patients
- UAP
 - recent onset angina
 - progression of symptoms
 - occurrence of rest pain
 - prolonged pain
- AMI
 - primary PTCA
 - rescue PTCA
 - shock stage
 - conservative treatment with thrombolytic treatment and ETT +
 - recurrent pain

Common cases for CAG and coronary intervention

- -ETT+ ve at low to mod work load
- -Chest pain unknown etiology, even patient was treated as CAD but symptom is still bad
- -pre op for valve operation
- -mod to high risk ACS USA, NSTEMI)
- -S/P AMI (prognostic cases)
- -Primary/rescue PTCA

Acute myocardial infarction

- class II(evolving MI)
 - within 6 hrs for PTCA or CABG
- completed MI
 - -class I
 - recurrent chest pain
 - VSD or severe MR
 - suspected pseudoaneurysm
 - -class II
 - cardiogenic shock
 - MI due to coronary emboli
 - CHF or hypotension
 - recurrent VT or VF

Atypical chest pain

- ETT +ve
- suspected CAD
- associated LV dysfunction

Relative Contraindication

- Uncontrolled ventricular irritability
- Uncontrolled hypokalemia or digitalis intoxication
- Uncontrolled HT
- Intercurrent febrile illness
- Decompensated heart failure
- Abnormal coagulogram
- Severe allergic to contrast agent
- Severe renal insufficiency and or anuria

Choice to approach

- Brachial
- Radial
- Femoral

Preparation and Premedication of the patient

- <1 % of serious complication (stroke, heart attack, death)
- Prefer to have INR <2, no heparin >4 hours
- Our order-NPO at less 6 hours
 - clean and shave perineum both groins
 - Iv fluid after NPO
 - urinate prior to cath lab, condom or Foley cath
 - record VS before patient leaves for Cath Lab
 - Pre med tranquilizer, antihistamine
 - aware about LMWH effect
- Consent must be done risk and benefit of procedure have to be explained to patient and relatives clearly

Preparation protocol for cardiac cath

- NPO 6 hrs
- clean and shave both groins, perineum
- IV hep lock
- check PT,PTT if history on heparin or coumadin
- omit LMWH morning dose If possible
- check lab HIV, HBS AG, BUN, Cr.E LYTE
- consent
- Sedative medication

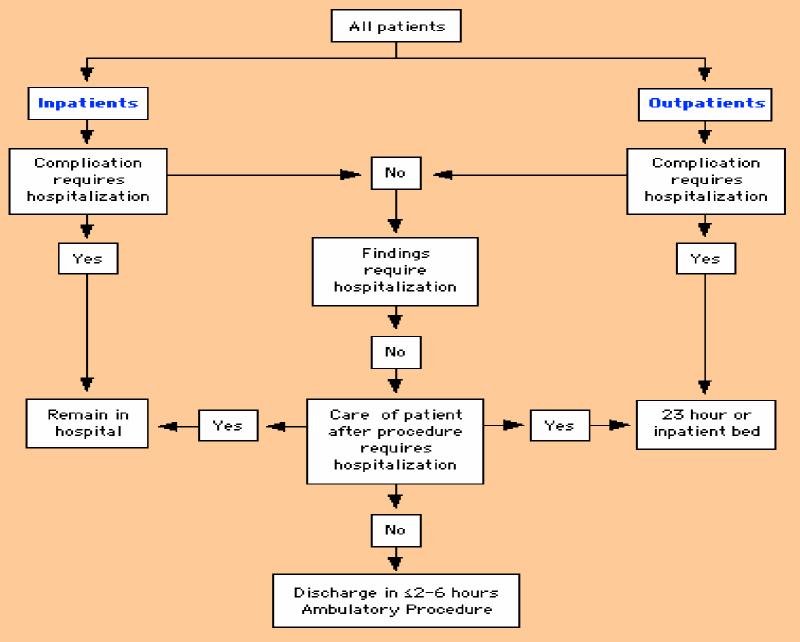
Protocol for PCI

- ASA 5 days before+-Plavix 4 tabs stat
- EKG
- CCU bed available
- lab routine and cardiac enzyme

Finding which we should find out

- LM,LAD,LCX,RCA
- LIMA,RIMA
- Coronary venous bypass graft
- LV function
- gradient across valve
- collateral circulation

General scheme for the disposition of adult patients after diagnostic cardiac catheterization*



*With permission from Smith, SC, Dove, JT, Jacobs, AK, et al, J Am Coll Cardiol 2001; 37:2230.

How to operate high risk cases

- Steroid and antihistamine in allergic cases
- IABP in LV dysfunction
- Temp pace in EKG abnormality
- Hydrate patient in CRI, role of acetyl cysteine for prevent further renal failure
- Stand by Adhoc PCI in high risk CAD
- Good cooperate team

Suggested General Exclusion Criteria for Early (<2-6 h) Discharge After Invasive Cardiac Procedures[†]

Type of patient	Diagnostic procedures	Therapeutic procedures
Adult	High risk due to identification of left main disease NYHA Class III or IV heart failure Unstable ischemic symptoms at any time after the procedure Recent MI with post-infarction ischemia Pulmonary edema thought to be caused by ichemia Severe aortic stenosis with LV dysfunction Severe aortic insufficiency with a pulse pressure >80 mmHg Poorly controlled systemic hypertension Inadequate or unreliable follow-up over the next 24 hours Generalized debility or dementia Renal insufficiency (creatinine >1.8 mg/dL) Need for continuous anticoagulation therapy or treatment of a bleeding diathesis Large hematoma or vascular complication	All procedures are excluded at this time, but the early discharge of selected patients after coronary intervention is under investigation (ie, patients with radial arterial access or after the use of percutaneous closure devices)
Pediatric	Young age (45 years of age) Complex congenital heart disease Any condition resulting in important cyanosis Most cases requiring large arterial sheath	All procedures excluded

[†] Smith, SC, Dove, JT, Jacobs, AK, et al, J Am Coll Cardiol 2001; 37:2230.

Risk of Cardiac Catheterization and Coronary Angiography, (based on 59,792 patients)[†]

	Percent
Mortality	0.11
Myocardial infarction	0.05
Cerebrovascular accident	0.07
Arrhythmia	0.38
Vascular complications	0.43
Contrast reaction	0.37
Hemodynamic complications	0.26
Perforation of heart chamber	0.28
Other complications	0.28
Total of major complications	1.70

Noto, TJ Jr, Johnson, LW, Krone, R, et al. Cardiac catheterization 1990: A report of the Registry of the Society for Cardiac Angiography and Interventions (SCA&I).Cathet Cardiovasc Diagn 1991; 24:75. Copyright © Wiley-Liss, Inc. Reproduced with permission of John Wiley & Sons, Inc.

Variables	Definitions
Age	Date of birth as stated by the patient or family
Gender	Male or female
LVEF-calculated	Calculated by left ventriculogram, echocardiography, blood pool scan
LVEF-estimated	Estimate left ventriculogram, echocardiography, blood pool scan
No. of vessels >70 percent	By angiography measured, quantified or estimated diameter stenosis; "vessel" defined as RCA and its branches, proximal LAD (before 1st diagonal), mid/distal LAD and its branches, and Cx and its branches
Unstable angina	Progressive or new onset or occurs at rest accompanied by ECG changes, hypertension or pulmonary congestion
CCS class IV	Highest CCS angina class leading to hospital admission and/or intervention; O = no angina by history
Heart failure	History of heart failure before intervention
Myocardial infarction (MI) at this admission	Within 24 hours of acute MI
Previous MI	>1 day; <7 days of acute MI
Urgency of the procedure	Elective: Patient clinically stable, procedure routinely scheduled Urgent: Unstable patient; procedure scheduled before discharge Emergent/: Ongoing ischemia including rest angina despite maximal ongoing therapy (medical or intraaortic balloon pump [IABP}) ischemia Emergent/: Arrest with cardiopulmonary resuscitation immediately before entering the laboratory
Cardiogenic shock	Hypoperfusion with SBP < 80 mmHg and central filling pressure >20 mmHg or cardiac index <1.8 liters/min/m2, also present if inotropes or IABP needed to maintain these values
Preprocedural IABP/CPS	IABP/CPS assisted device placed before intervention
Aortic valve disease	Aortic valve area <1.0 cm2 and/or aortic regurgitation >2+
nor tro runte discuse	
Mitral regurgitation >2+	Presence of mitral regurgitation >2+

Clinical Risk Factors Associated with In-Hospital Adverse Events**

* More than 50 percent of databases that evaluated the variable showed an odds ratio >2.0 or variable chosen on multivariable analysis.

AMI, acute myocardial infarction; CCS, Canadian Cardiovascular Society; CPS, cardiopulmonary support; Cx, left circumflex artery; IMA, internal mammary artery; LAD, left anterior descending coronary artery; LMCA, left main coronary artery; LV, left ventricle; LVEF, left ventricular ejection fraction; RCA, right coronary artery; SBP, systolic blood pressure

† Smith, SC, Dove, JT, Jacobs, AK, et al, J Am Coll Cardiol 2001; 37:2230.

Patient characteristics associated with increased mortality from cardiac cath

- Age <1 year ,>60 years
- Functional class IV > I-II 10 times
- Severe CAD
- Valvular HD
- LV dysfunction EF <30% is more than 10 times greater than EF >50%
- CRI, IDDM,CVA,PVD,COPD

Procedural complications	Definitions
Primary cause of death	Patient died during this hospitalization
Periprocedural myocardial infarction (MI)	The NEW presence of an MI as documented by at least 1 of the following criteria: 1. Evolutionary ST-segment elevations, development of new Q-waves in 2 or more contiguous ECG leads, or new or presumably new left bundle branch block pattern on the ECG. 2. Biochemical evidence of myocardial necrosis; this can be manifested as (1) CK-MB 23 × the upper limit of normal or if CK-MB not available, (2) total CK 23 × upper limit of normal. Normal limits of certain blood tests may vary and nned to check with local lab for normal limits for CK-MB and total CK.
Coronary artery bypass grafting (CABG) during this admission	If the patient had a CABG during this admission indicate the CABG status using the following categories: I Elective: The procedure could be deferred without increase risk of compromised cardiac outcome. II Urgent: All of the following conditions are met: A. Not elective B. Not emergency C. Procedure required during same hospitalization in order to minimize chance of further clinical deterioration III Emergency: The patient's clinical status includes any of the following: A. Ischemic dysfunction (any of the following): 1. Ongoing ichemia including rest angina despite maximal medical therapy (medical and/or intraaortic balloon pump) 2. Acute evolving MI within 24 hours before intervention 3. Pulmonary edema requiring intubation B. Mechanical dysfunction (either of the following):

- 1. Shock with circulatory support
- 2. Shock without circulatory support
- IV Salvage: The patient is undergoing cardiopulmonary resuscitation en route to the operating room.

Cerebrovascular accident/ strokePatient experienced a cerebrovascular accident (CVA) as documented by a loss of neurological function caused by an ischemic event with residual symptoms at least 24 hours after onset.

[†] Reproduced with permission. ACC/AHA guidelines of percutaneous coronary interventions: a report of the ACC/AHA Task Force on Practice Guidelines. JACC 2001; 37(8):2215-38.

Side Effects of Contrast agents

- Arteriolar vasodilatation (hypotension)
- Increase intravascular volume
- EPS effect (QT prolong, Bradycardia ST-T change)
- N/V
- LV depressant
- Anaphylaxis
- Myocardial ischemia
- Renal toxicity

Ischemic pain

- SL GTN, IV GTN
- start coronary artery angiogram first
- emergency PTCA or CABG

Acute CHF

- avoid or reduce contrast media volume in LV gram
- oxygen, morphine, diuretic, nitrate
- IABP

Anaphylactoid reactions to contrast media

- cutaneous and mucosal
 - urticaria
 - pruritis
 - flushing
 - angioedema
 - laryngeal edema
- cardiovascular
 - vasodilatation
 - hypotension
 - arrhythmia
- · smooth muscle
 - bronchospasm
 - GI spasm
 - uterine contraction

Recognition and prophylaxis of high risk patients

- antihistamine
- steroid
- history of dye or seafood allergy
- IV access
- high risk after prophylaxis with steroid
 - -minor 5-10%
 - -severe <1%

Treatment

- IV diphenhydramine 25-50 mg
- SC epinephrine
- IV hydrocortisone 200-500 mg
- IV aminophylline

Arrhythmia

- bradyarrhythmia
 - IV atropine 0.4-0.6 mg
 - temp pacing
- tachyarrhythmia
 - VF 0.3-0.5% incidence
 - treated by DC shock 300 watt
 - lidocaine
- cardiogenic shock
 - rare.LM disease
 - poor LV function
 - IABP
 - emergency revascularization

Contrast nephropathy

- creatinine>2mg/dl,incidence 0.5%
- serum Cr peak 7 days
- oliguria 2-4 days
- return to baseline in 80-90% of cases
- prevented by saline hydration
- avoid large volume of contrast media

Incidence of contrast nephropathy

- normal creatinine 0-0.6%
- non diabetic renal insuff

diabetic nephropathy

- after renal transplant 36-65%
- **MM** 1-2%

Factors increasing risk of contrast nephropathy

- DM
- MM
- renal transplantation
- dehydration
- previous episode of contrast nephropathy
- multiple contrast studies within 24-48 hrs
- renal insufficiency
- aging

Risk patients with prolonged cardiac cath

- severe PVD
- general debility, mental confusion, cachexia
- bleeding diathesis
- uncontrolled HT
- uncontrolled DM
- recent stroke <1 month
- renal insufficiency

Arterial entry site complications

- femoral
 - hematoma
 - false aneurysm
 - retroperitoneal bleeding
 - significant hypotension within 12 hrs
 - occlusion of femoral artery(absence of distal pulse)
 - severe PVD
 - emboli
 - using heparin
- Brachial
 - infection
 - median nerve injury
 - brachial artery spasm
 - occlusion

Post op care

- CAG
 - -observe groin, VS in 4-6 hours, if radial approach patient can be discharged in 2 hours
 - -I/O
 - -rash

Patient care after coronary angiogram

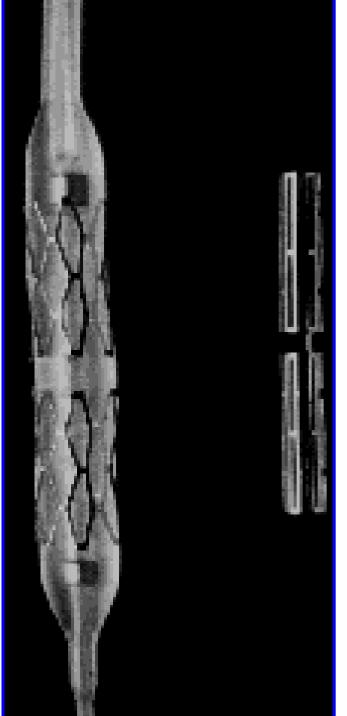
- observe vital sign
- observe groin complication
- rest procedured limb for 4 hrs
- line flat for 4hrs and semiupright for 2 hrs
- symptomatic treatment for pain or chest pain
- start diet, fluid
- discharge after 6 hrs except complicated cases

Post op care

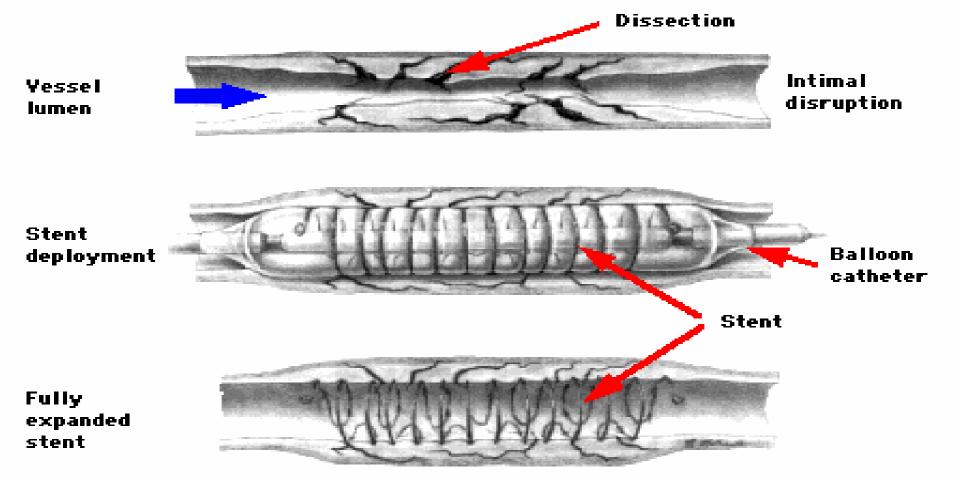
- PCI
 - -keep sheath in until ACT < 180 then sheath was removed and further observe hematoma and VS for 6 hours
 - -if patients are stable, they can be moved to private room in same day or tomorrow

Patient care after PCI

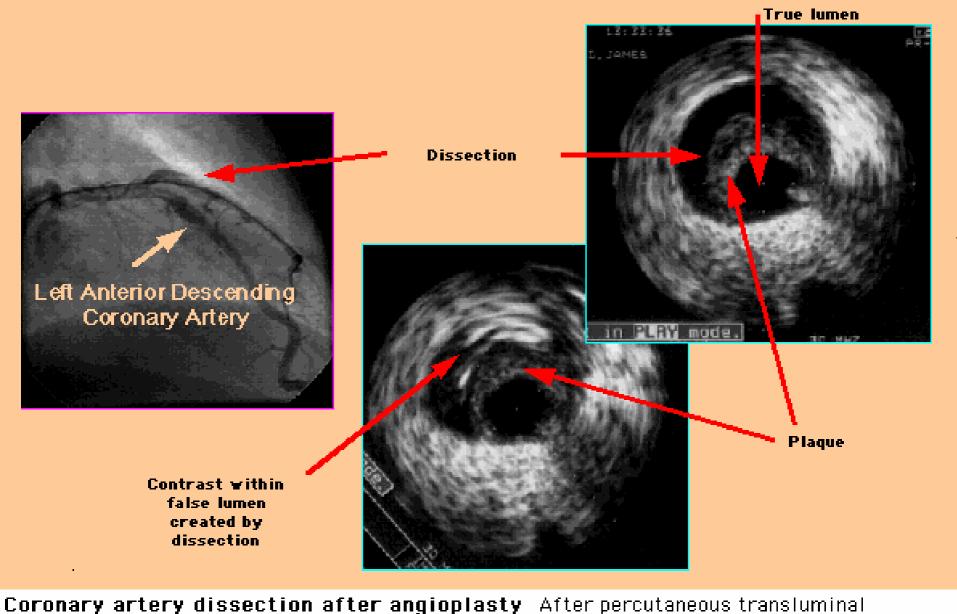
- EKG at ward immediately and the day after
- monitor vital sign in CCU
- observe symptom:chest pain observe groin
- start diet, fluid in uncomplicated procedures
- off sheath in uncomplicated cases after ACT 170,150 in reopro cases
- keep sheath over night with heparin flush in complicated cases or questionable cases+/-IV heparin
- check CK,MB,TPT,TPI
- off sheath with temazepam,atropine,plasil,morphine with 20-30 minutes compression and follow same as CAG protocol



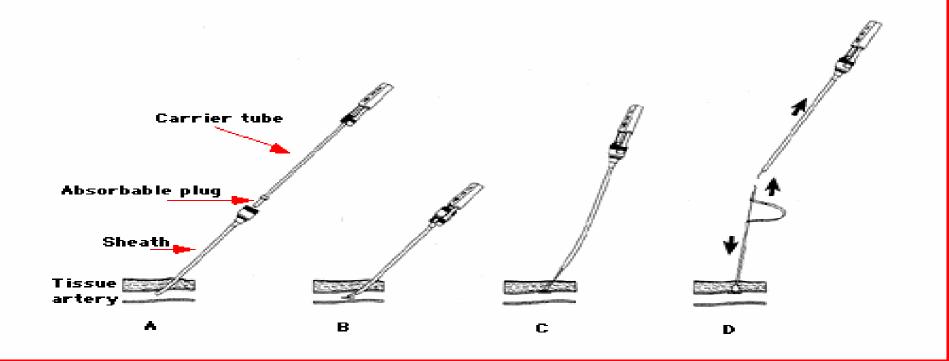
Types of intracoronary stents There are two major types of coronary stents: those that are balloonexpandable, such as the Palmaz-Schatz and Gianturco-Roubin stents (which are approved by the Food and Drug Administration in the United States); and those that are self-expandable, such as the Wallstent, The Palmaz-Schatz stent shown on the left is composed on two seven mm steel slotted tubes connected by a one mm central bridging strut.



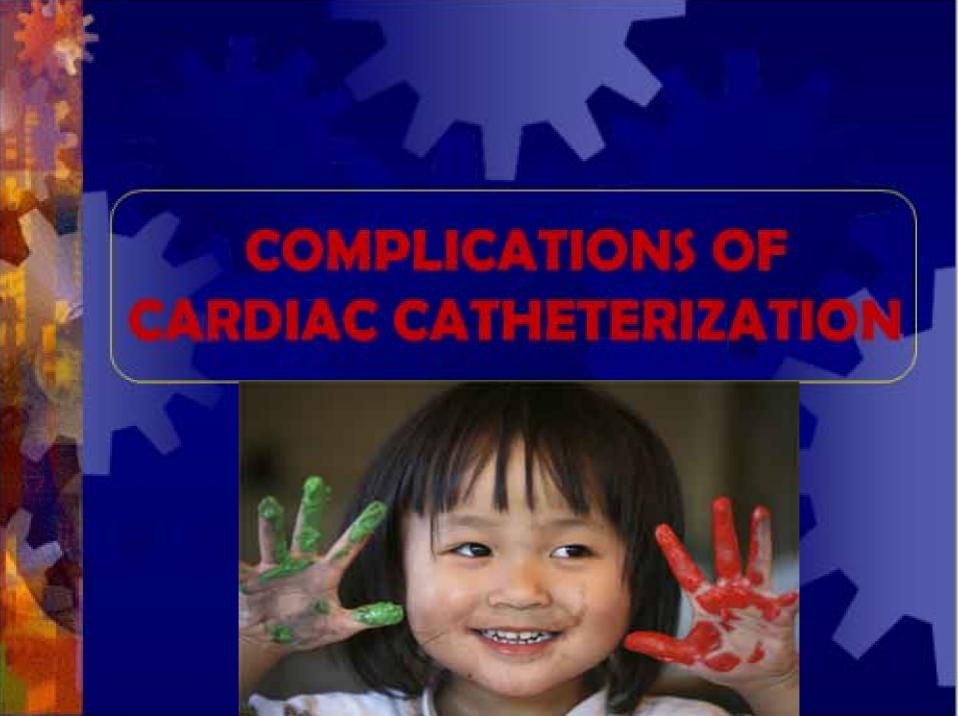
Deployment of coronary stent Steps involved in the deployment of a coronary stent. The step is placed after balloon angioplasty, which creates intimal disruption (top panel). Following placement (middle panel), the stent is expanded (bottom panel). (Adapted from Cannon, A, Roubin, GS, Interventional Cardiovascular Medicine, Roubin, GS, Califf, RM, O'Neill, WW, et al (Eds), Churchill Livingstone, New York, 1994, p. 866.)



coronary angioplasty, an extensive coronary artery dissection can create an intimal flap that limits coronary flow. This figures shows the dissection on arteriography (left panel) and intracoronary ultrasonography (middle and right panel) and its related to the atherosclerotic plaque and the false lumen created by the dissection.



Insertion protocol for the hemostatic puncture closure device. The hemostatic puncture closure device consists of an absorbable plug component and delivery system. The plug has a lactide and glycolide anchor attached to a 24- to 28-mg collagen sponge by an absorbable suture. The device is contained in a carrier tube. Delivery of the device is initiated by first exchanging the existing sheath over a wire for an 8 Fr sheath. Panels A and B: The carrier tube containing the plug is inserted through the sheath deploying the anchor within the arterial lumen. Panel C: The sheath and carrier tube assembly are then retracted, maintaining tension on the assembly while pushing down the tamper tube. Panel D: Through use of a tension spring, traction is maintained along the suture line, resulting in plugging of the anterior arterial wall puncture site by the anchor and collagen plug. After 20 minutes, the tension spring is removed and the suture is cut below skin level. (Reprinted from Ward, SR, Lasale, P, Raymond, R, et al. Efficacy and safety of a hemostatic puncture closure device with early ambulation after coronary angiography. Angio-Seal Investigators. Am J Cardiol 1998; 81(5):569-72. Copyright 1998, with permission from Excerpta Medica, Inc.)



Complications

Major:

Cerebrovascular accident

Death

Myocardial infarction

Ventricular tachycardia, fibrillation, or serious arrhythmia

Minor:

Aortic dissection

Cardiac perforation, tamponade

Congestive heart failure

Contrast reaction (anaphylaxis, nephrotoxicity)

Heart block, asystole

Hemorrhage (local, retroperitoneal, pelvic)

Infection
Protamine reaction
Supraventricular tachyarrhythmia, AF
Thrombosis, embolus, air embolus
Vascular injury, pseudoaneurysm
Vasovagal reaction

Death	0.1
MI	0.05
CVA	0.07
Arrhythmia	0.38
Vascular	
Contrast	0.37
Hemodynamic	0.26

Perforation......0.03 Other.....0.28

High Risk Pts for Complications

AMI Advanced age (>75yrs) Aortic aneurysm Aartic stenosis Congestive heart failure Diabetes Extensive 3VD Suspected or known LMCAD LV dysfunction (EF<35%) Obesity Prior stroke Renal insufficiency Uncontrolled HTN Unstable angina

Increased general medical risk

Age > 70 years

Complex congenital heart disease

Morbid obesity

General debility or cachesia

Uncontrolled glucose intolerance

Arterial oxygen desaturation

Severe chronic obstructive lung disease

Renal insufficiency with creatinine greater than 1.5 mg/dl

Increased cardiac risk

Three-vessel coronary artery disease

Left main coronary artery disease

Functional class IV.

Significant mitral or acetic valve disease or mechanical prosthesis

Ejection fraction less than 35%

High-risk exercise treadmill testing (hypotension or severe ischemia)

Pulmonary hypertension

Pulmonary artery wedge pressure greater than 25 mm Hg

Increased vascular risk

Anticoagulation or bleeding diathesis

Uncontrolled systemic hypertension

Severe peripheral vascular disease

Recent stroke

Score portir insufficiency

Landmarks for Femoral Access

What is your primary landmark?



Landmarks for Femoral Access

What is your primary landmark?

3. Bone landmarks

2. Maximal pulsation

1. Inguinal crease

Usual Approach

 Keep poking until you get a gusher





Local complications of FA access: 2-10%

- Hematema (1-10%)
- Pseudeaneurysm (1:6%)
- · AV fistula (<1%)
- · Vessel laceration (<1%)
 - Free bleeding
- Intimal dissection
 - · Ante- or retre-grade
- . Acute vessel clesure (<1%).
 - Thrombosis (small artery lumen)

- Retreperiteneal hemerrhage (0.2 = 0.9%)
- Thickening of the perivascular tissues
- Neural damage
- Infection
- Veneus thrembesis
- Pericatheter elet

Why Access is # 1 Cause of Cath Complications – A Half Century Later

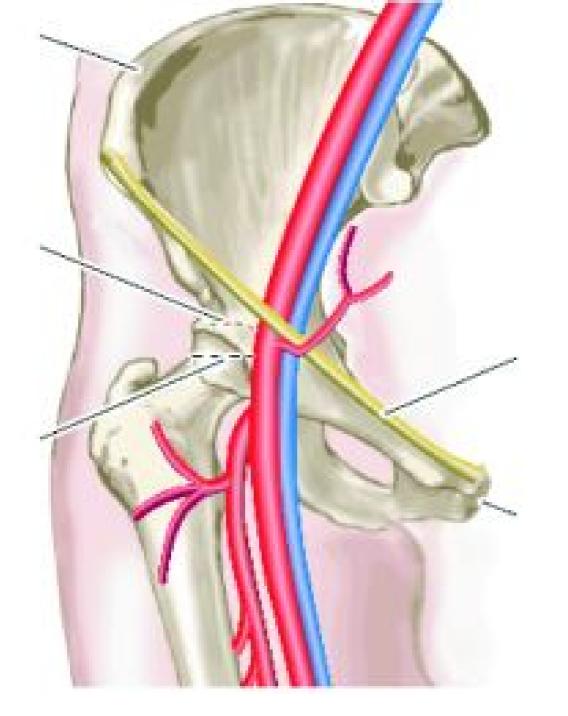
- Puncture technique
- Sheath management
- Adjunctive drug therapy
- Closure

Complications of Vascular Access

- Local Hematoma
- Pseudoaneurysm
- AV-Fistula
- Retroperitoneal Hematoma
- Infection

Local Hematoma

- Increase in frequency with the increasing size of sheath, the amount of anticoagulation, and the obesity
- More common than free bleeding
- If bleeding stops with manual compression, it will usually resolve within 1-2wk.
- Possible sensory or motor deficit that may take weeks or months to resolve
- Larger hematomas may require transfusion, but surgical repair is generally not required.



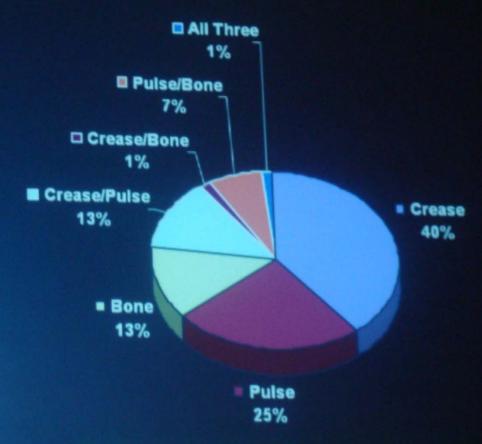
Landmarks Used for Femoral Puncture

Skin Crease

Maximum Pulse

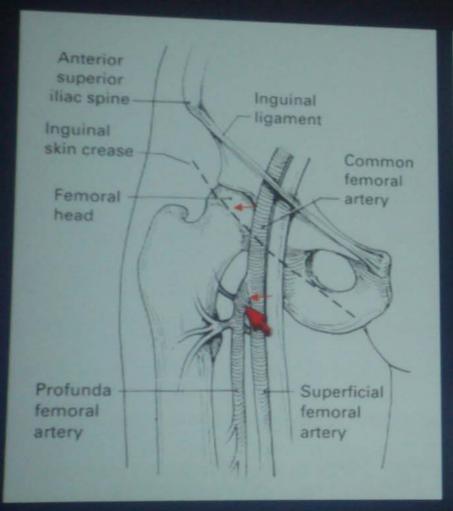
Bony Landmarks

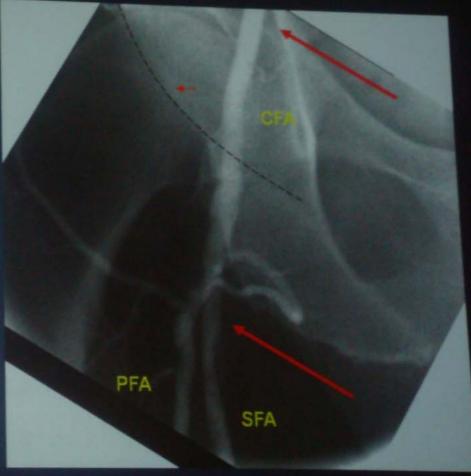




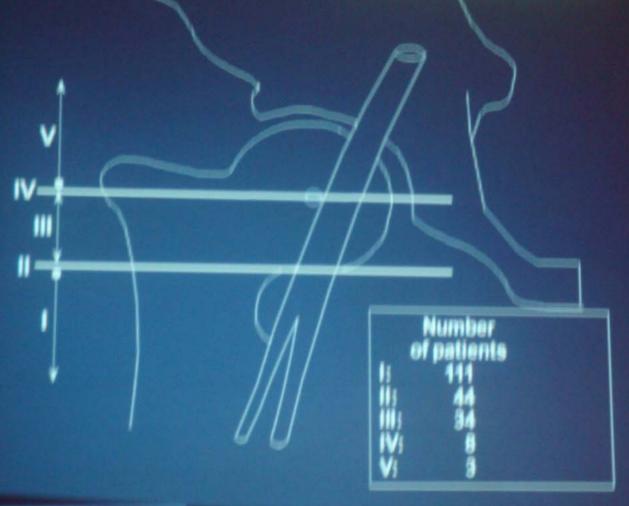
Skin Crease Most Common

This is NOT Normal Anatomy





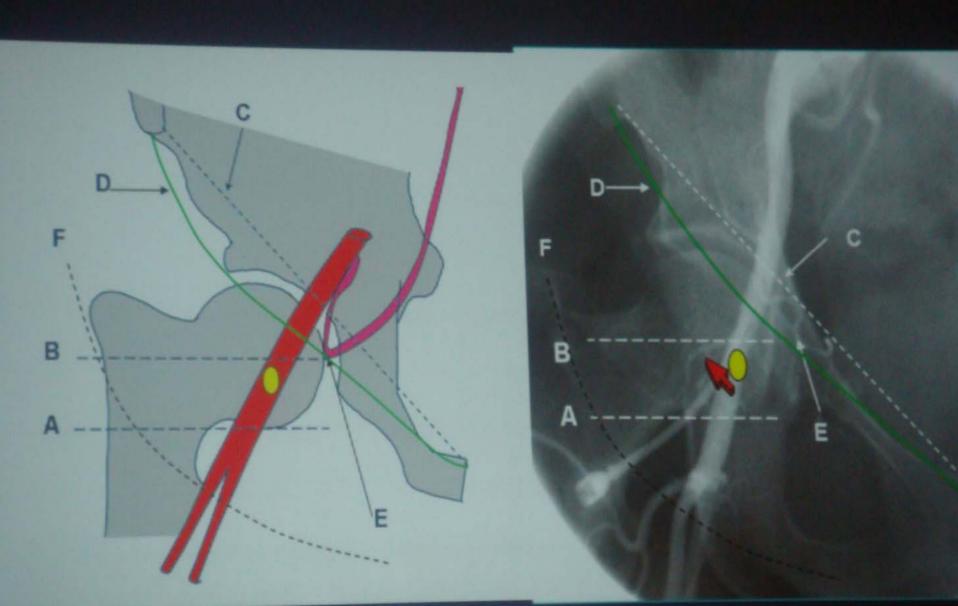
Femoral Head and the CFA Bifurcation



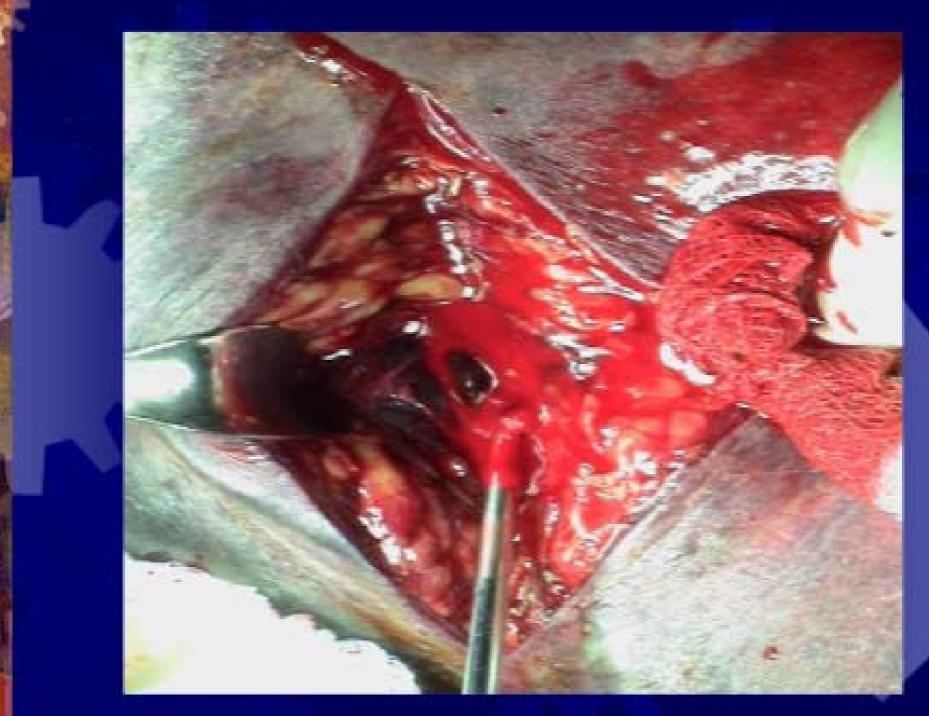
Target Zone



Recommended Approach











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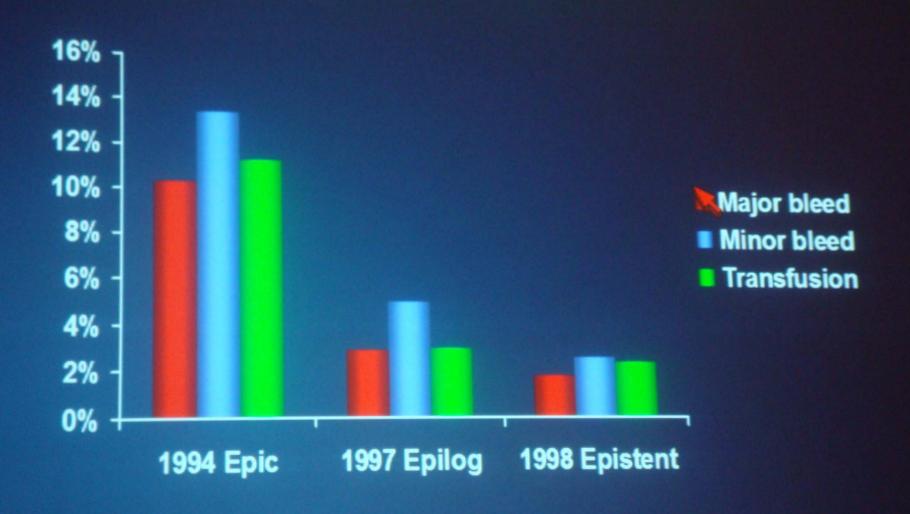
HR Sanati MD

Infections



- 0.3%
- Median incubation –
 8 days
- Staph aureus 75%
- BC + 86%
- Diabetics 80%
- PSA 42%

Bleeding Complications









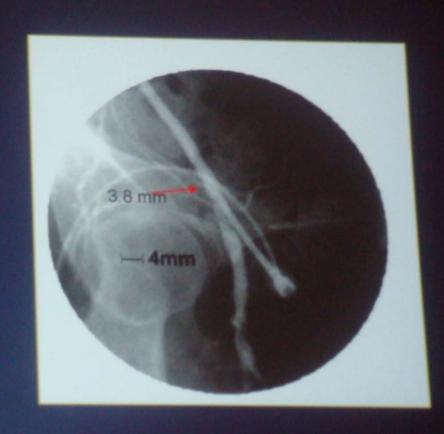


Strategies Minimizing Bleeding

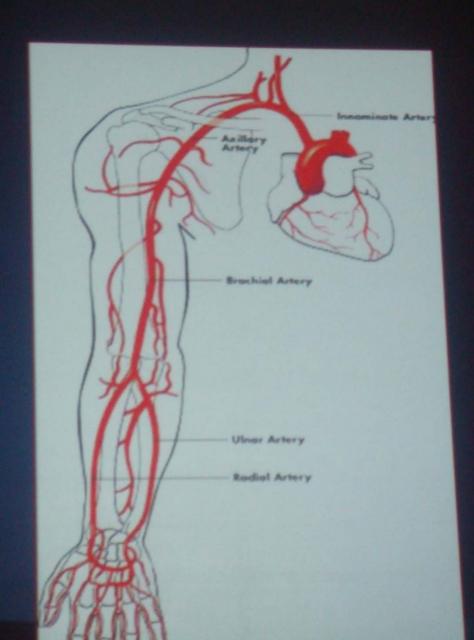
- Weight-adjusted heparin
- Lower heparin dose
- No postprocedure heparin
- Smaller guiding catheter

Predictors of Complications

- Age
- Gender
- Diabetes
- ↓ Body surface area
- Sheath size
- Vessel size*
- Anticoagulation
- Puncture location*
- Prior instrumentation
- Vascular disease at puncture site
- ? IIb/IIIa



Consider Radial

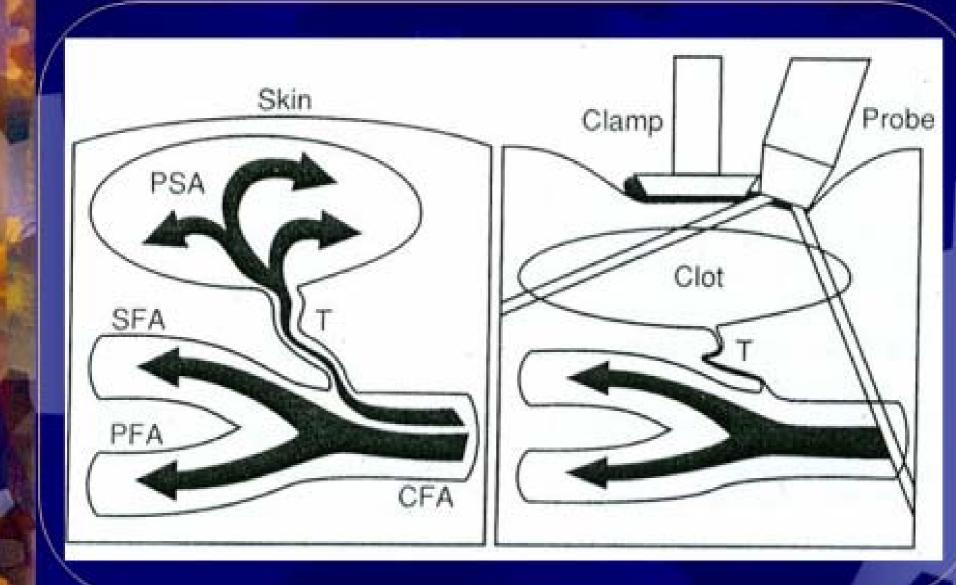




Pseudoaneurysm

- Low femoral arterial puncture (below the bifurcation of common femoral a. due to smaller caliber & the lack of bony structure against) and poor initial control of bleeding are the main causes.
- It may develop if a hematoma remains in continuity with the arterial lumen (dissolution of the plugging clot at the puncture site).
- Presence of pulsation or audible bruit over the site in contrast with

- Risk of neurovascular complication or rupture
- All but the smallest (<2cm) false aneurysms tend to enlarge and ultimately rupture.
- In the past, vascular surgery was the only treatment.
- with ultrasound imaging techniques these false channels can be easily identified and nonsurgical closure selected. Manual compression (30-60min) of the expansile growing mass guided by US (with or without thrombin or collagen injection) is an acceptable therapy.
- False aneurysms smaller than 2cm in diameter may be followed expectantly (up to half close before a 2-week follow up US)

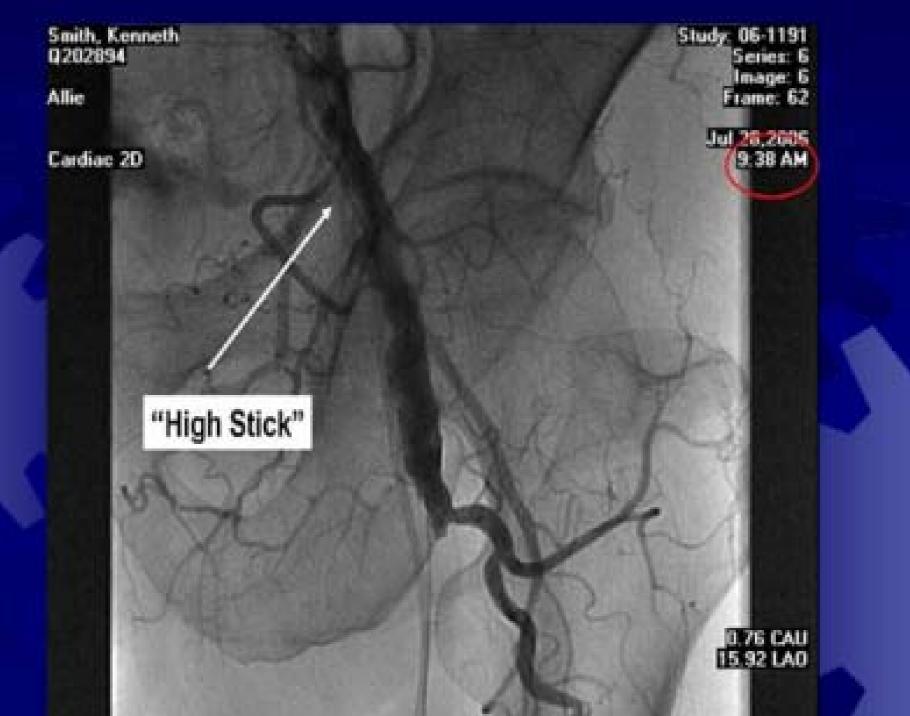


Retroperitoneal Hematoma

High femoral arterial puncture and full anticoagulation are the main causes. It should be suspected in pts with hypotension, tachycardia, pallor, a rapidly falling Hct, lower abdominal or back pain, or neurological changes in the punctured leg.

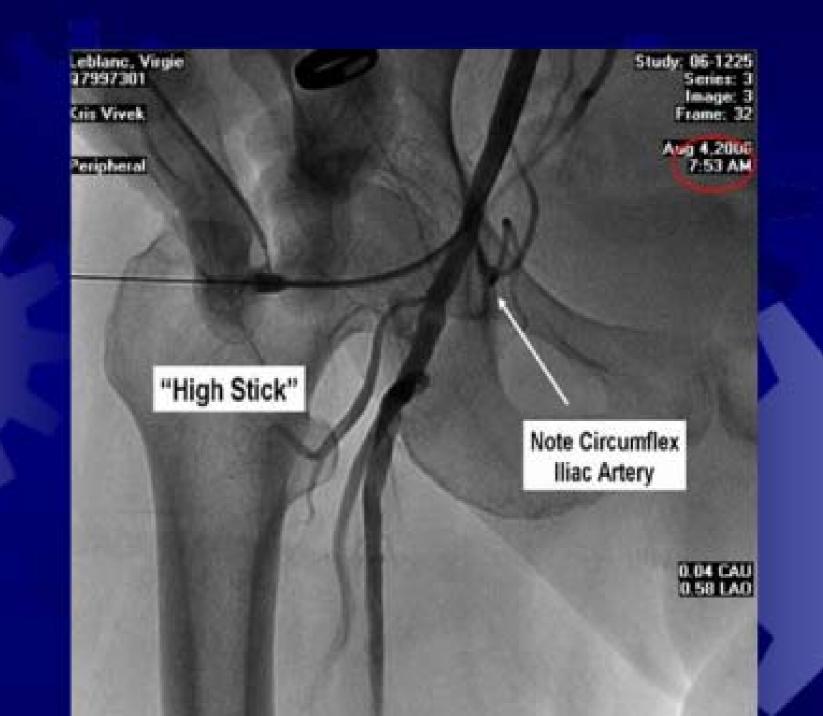
Confirmation by CTs or US, but the treatment is usually expectant (transfusion, bed rest) rather than surgical.

The possibility to tamponade the bleeding site by a peripheral angioplasty balloon followed by placement of covered stent (esp. in the case of sheath-induced laceration of tortuous iliac a. that is fatal within minutes)









AV-Fistula

Results from ongoing bleeding from the femoral a. puncture site that decompress into the adjacent venous puncture site.

Risk factors are low puncture site and poor bleeding control.

Recognized by to-and fro-continuous bruit over the site, and may not be clinically evident until days.

Construction These may enlarge with time, but at least one third close spontaneously within 1 year, after which surgical repair should be entertained.

Infection

Risk factors include: repeat ipsilateral femoral puncture or prolonged femoral sheath maintenance (1-5 days)

Contrast-Induced Nephropathy

- -Increase of serum Cr>%25 or > 0.5 mg/dl above baseline within 48 hours of contrast administration
- -In 2-10% of unselected population of patients undergoing CAG
- -The incidence of CIN reported in the literature has ranged between %1 and %45, largely depending on the comorbidities of the study population and the parameters used to define CIN.

In normal renal function → < % 2
Risk of CIN in nondiabetics → %13
Risk of CIN in diabetics → % 20

- -In pts with CIN → %5-10 transient dialysis and %1 long-term dialysis
- -The occurrence of CIN is associated with %36 inhospital mortality rate and a 2 year survival of only %19
- Given the age-related decline in renal function the elderly have a 3-fold higher risk of CIN compared with younger patients

Serum Cr peak: 3-5 days after contrast administration and return to baseline levels in most cases by 7 to 10 days

The single most important risk factor for development of CIN is baseline renal dysfunction Other factors: DM, CHF, dehydration, nephrotoxic drugs such as cyclosporine and NSAIDS

Pathophysiology of CIN

- Direct toxicity
- II. Micro showers of atheroemboli to both kidneys
- III. Intra renal vasoconstriction
- IV. Hypoxia triggers activation of the renal sympathetic nervous system



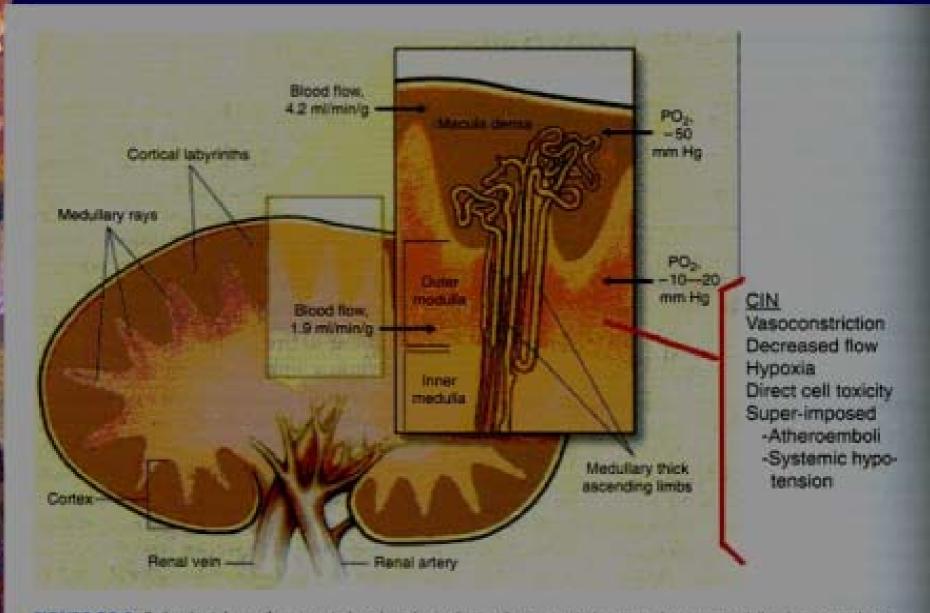
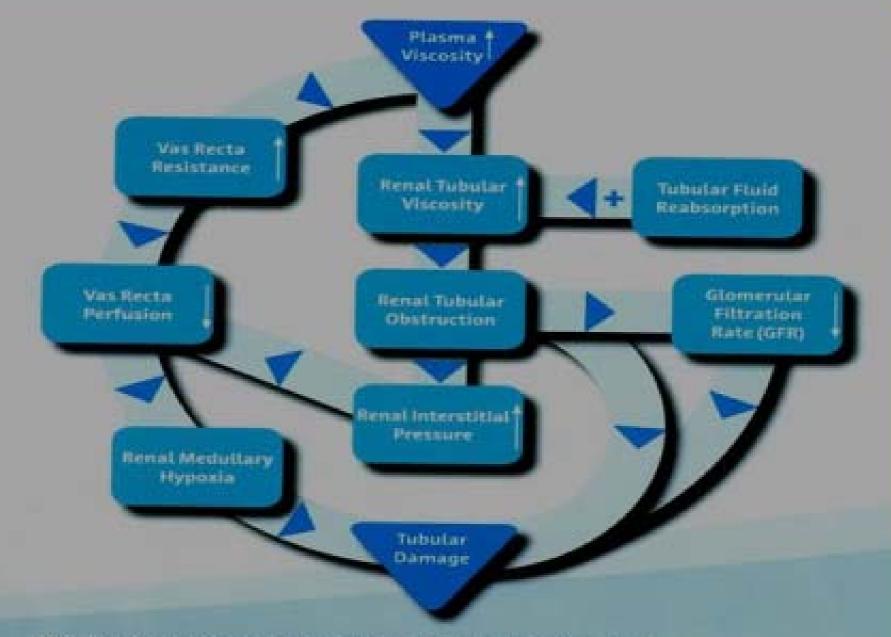


FIGURE 23-2. Pathophysiology of contrast-induced nephropathy involves acute ischemia to the outer medula, the most vulnerable part of the kidney, due to direct cellular taxicity and sustained intrarenal vasoconstriction and reduction in renal blood flow. This process is worsened by multiple factors including hypoxia, anemia, and systemic hypoperfusion. CIN = contrast-induced nephropathy. [Adapted from Brezis M, Rosen 5.22]



Flow chart of mechanisms linking tubular damage to CM viscosity*

* Increasd plasma vicosity is the trigger factor

Risk Factors for the Development of Contrast-induced Nephropathy

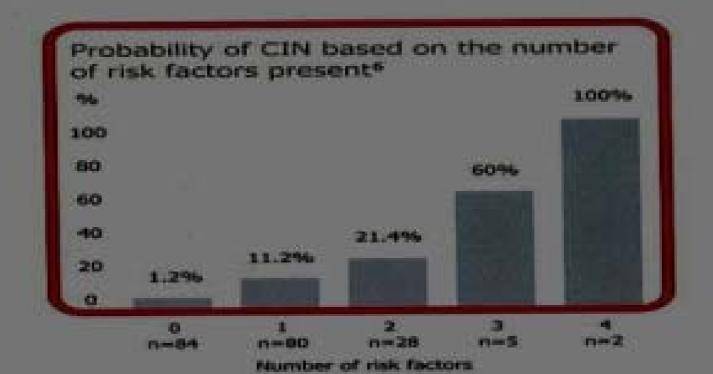
- eGFR \leq 60 mL/min/1.73 m²
- Diabetes
- Urine ACR >30
- Hypertension
- History of structural kidney disease or damage

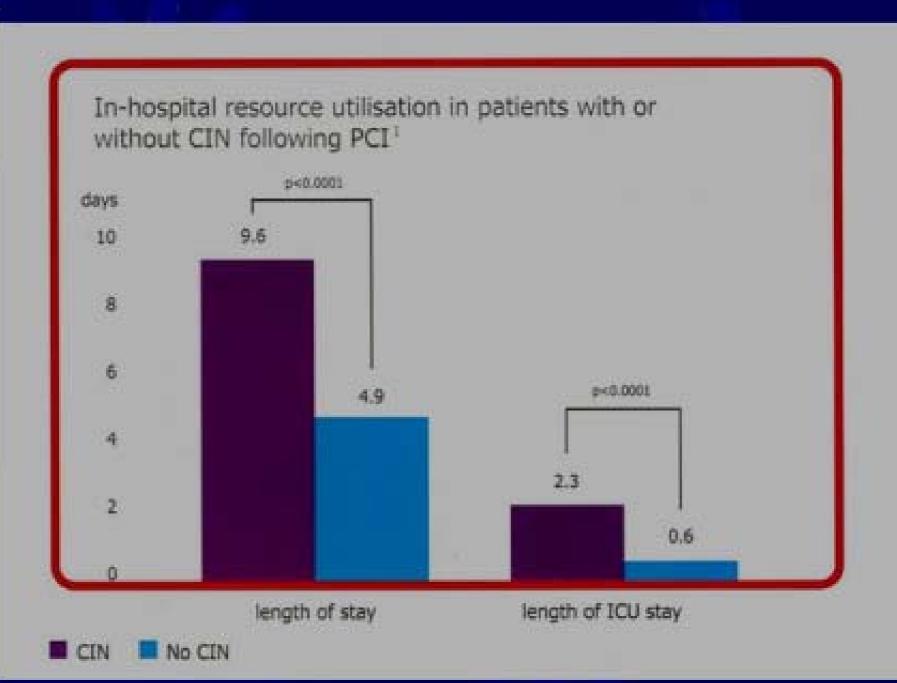
- Congestive heart failure
- Preprocedural volume depletion
- Intraprocedural hypotension
- Intra-aortic counterpulsation
- Cholesterol emboli syndrome
- Use of large volume of contrast

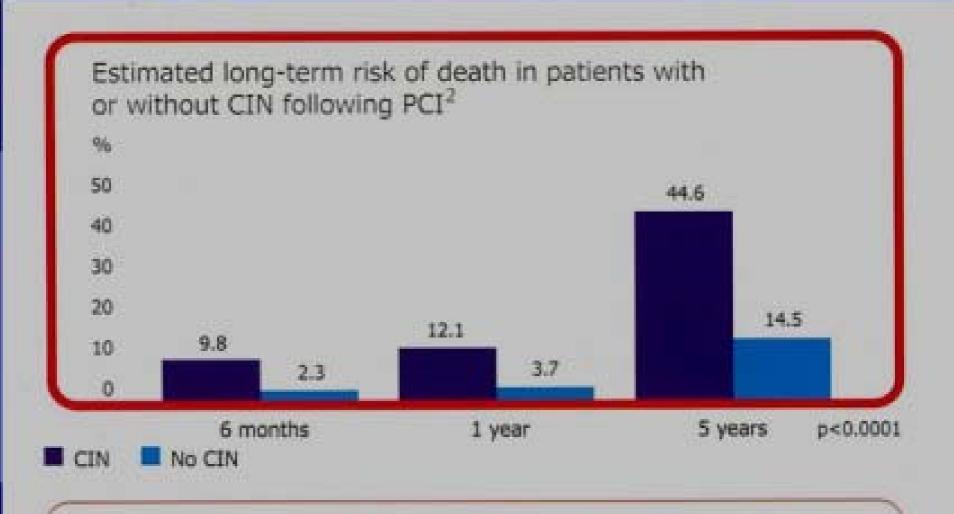
Risk factors for CIN*5

- · pre-existing renal impairment
- diabetic nephropathy
- dehydration
- congestive cardiac failure
- use of nephrotoxic drugs including NSAIDs
- · age over 70 years
- Large doses of contrast media

The sicker the patient, the higher the risk^{6,7}







Retrospective analysis of 7,586 patients who had coronary intervention procedures at the Mayo clinic from January '96 to May '00²

Circulation 2002 CIN manifests usually as ARF and is typically non oliguric.

In oliguric ARF the time course of the oliguria and the rise in serum Cr depend on the preprocedure baseline serum Cr.

Pts with normal renal function or mild renal functional impairment prior to receiving radiocontrast agents usually have oliguria lasting 2 to 5 days with recovery to baseline renal function by day 7.

D_{λ}

- The use of serum Cr solely is not a reliable indicator of renal damage and should not be used to assess the level of kidney function.
- -GFR is the best measure of overall kidney function and is equal to the sum of the filtration rates in all of the functioning nephrons and is a rough measure of the number of functioning nephrons.
- -The normal level of GRF varies according to age, gender, and the body size. (normal GRF in young adults is 120-130m/min)

COMPLICATIONS OF CORONARY INTERVENTION

DISSECTION & ACUTE CLOSURE

A major cause for in-hospital death, MI and emergent CABG following PCI in pre-stent era.

With coronary stents, dissection leading to abrupt closure is unusual but it is still an important cause of ischemic complications, which is edge dissection following stenting that predispose to stent thrombosis.

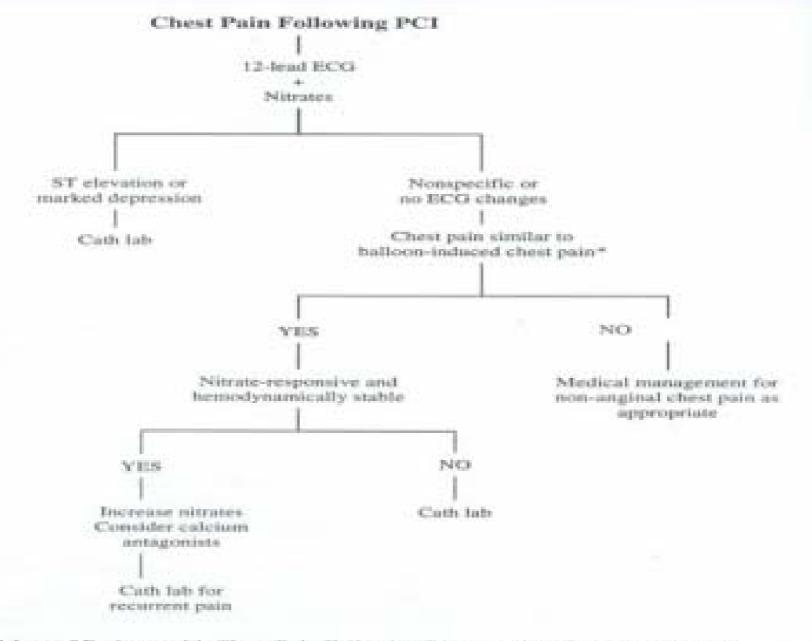


Figure 20.3. Triage of Patients with Chest Pain Following Percutaneous Revascularization

PCI = percutaneous constary intervention

^{*} MPCI was performed on a chronic total occlusion, these patients should return to the eath lab for repeat angiography

Management of Acute Closure

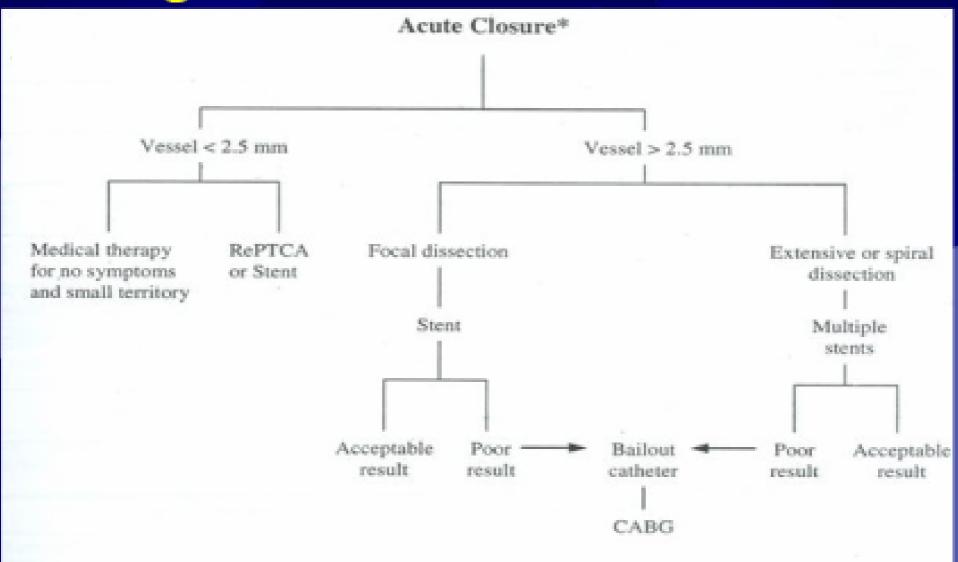


Figure 20.4. Management of Acute Closure

Consider IVUS to guide optimal stenting (Chapter 31)



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