TIPS & TRICKS

3rd Edition

TIPS & TRICKS *in* Interventional Cardiology

Shuvanan Ray

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Contents

CHAPTER 2

Percutaneous Coronary Intervention: Workstation (The Unsung Heroics)

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INTRODUCTION

This chapter was written by the Pre-CathLab junior doctor, and it was for the CathLab juniors, doctors, nurses, technicians and the peripheral personnel involved. Though there is a tendency to rate percutaneous coronary intervention (PCI) to almost a procedure such as sebaceous cyst excision or any outdoor surgery, it is actually not so. It is a procedure which can turn ugly at any moment and can produce severe consequences, even death. A survey (Institute of Medicine Report, 1999) showed that 44,000–98,000 deaths annually happened from adverse events during procedure which is equivalent to one airplane crash each day. Most of the cases were related to either a mistake in picking up a cue before sending to catheterization laboratory or missing out a drug before or during the procedure (like antiplatelets/heparin). Patients and relatives do not understand the heroics in catheterization laboratory, but they remember the welcoming environment, prompt professional work-up, and a transparent billing related to the procedure and this requires a team rather than an individual. A dedicated team working with a definite protocol and checklist can significantly reduce morbidity and mortality in patients undergoing any surgical procedures including percutaneous cardiovascular intervention.

COMPONENTS OF CATHETERIZATION LABORATORY TEAM

- Physician [primary operator, (PO)]
- *Secondary operators*: Assistant to PO
- Physicians assistants
- *Nurse supervisor*: This person must be familiar with the overall function of the laboratory, have strong management skills, help set tone of patient surroundings, and be in charge of preprocedure and postprocedure holding areas. Nurse supervisor should ensure that institutional guidelines for patient monitoring, drug administration, and protocols for patient care (including protocols for handling potential complications) are established and that all catheterization laboratory nurses are properly trained.
- *Catheterization laboratory nurses*: Background of such nurses should be from critical care experience, including knowledge of cardiovascular medications, intravenous (IV) infusion, and sterile techniques. Experienced with vascular catheter

instrumentation, especially with identification, cleaning, sterilization, and storage.

- *Non-nursing personnel*:
  - Technologist: Should have proper radiology and angiography training, experience about X-ray generators, cine pulse systems, image intensification, cine and digital imaging and storage, pressure injection system, and radiation safety principles
  - Additional administrative personnel:
    - Scheduler/case manager
    - Inventory manager
    - Database or administrative staff
  - Billing and customer care officer

1. Duty of catheterization laboratory team—pre-PCI
   C: Check (point by point of a checklist)
   A: Avoid (offending drugs and adverse laboratory/hemodynamic irregularities)
   R: Reset: Blood sugar
   Drugs: [Dual-antiplatelets therapy (DAPT), IV fluid, sedation, premedications]
   E: Explain to patient and relatives
2. Checklist—components
   - Patient identification, consent confirmed
   - Patient's recent clinical status/potential complication reviewed [electrocardiogram (ECG), ejection fraction (EF), congestive heart failure (CHF), and shock]
   - Procedure, indication
   - Equipment needed available
   - Access site planned
   - Allergies (especially contrast) and premedications
   - Antibiotic prophylaxis for implants
   - Laboratories reviewed
3. Most important laboratory reports
   - Hemoglobin% (Hb%), hematocrit (>30/stable)
   - Potassium—3.5–5 mEq/L.
   - Creatinine/blood urea nitrogen (BUN)/albumin
   - International normalized ratio (INR)
   - Platelets > 50,000/dL
   - Serology
4. Calculations
   - Estimated glomerular filtration rate (eGFR) [modification of diet in renal disease (MDRD)]
   - Bleeding score
   - Overall mortality score
   - SYNTAX score

CARE—C: CHECK

The following checklist should give an idea of the vital points. It is only for guidance purpose and kindly modify as per local protocols.

Catheterization laboratory checklist is shown in **Table 2.1** and postcatheterization checklist is shown in **Table 2.2**.

TABLE 2.1: Catheterization laboratory checklist.

C (Check)	*A (Avoid)*	*R (Reset)*	*E (Explain)*
Identity check	Nephrotoxics off	Aspirin loading	Consent
Procedure	Metformin	Prasugrel/ ticagrelor/ clopidogrel	
Indication	Gliflozins (SGLT-2 inhibitors)	Statins	
Laboratories	ACEi/ARB	Hydrocortisone	
Hb%, hematocrit	Diuretics	Chlorpheniramine	
Potassium 3.5–5 mEq/L		Other drugs	
Platelets > 50,000/dL			
Creatinine/BUN/ eGFR			
INR			
Serology			
Albumin		Hydration (IV fluids)	
Equipment	Oral anticoagulant	NAC	
Allergies	Bridging LMWH	Famotidine	
ECG		Cyclizine/ ondansetron	
Bedside echo		Sedation	

(ACEi: angiotensin converting enzyme inhibitor; ARB: angiotensin II receptor blocker; BUN: blood urea nitrogen; ECG: electrocardiogram; eGFR: estimated glomerular filtration rate; Hb: hemoglobin; INR: international normalized ratio; IV: intravenous; LMWH: low-molecular-weight heparin; NAC: N-acetylcysteine; SGLT-2: sodium-glucose cotransporter-2)

Electrocardiogram

There is no way to overemphasize the importance of an ECG—preprocedure ECG, postprocedure ECG, and an ECG from historical notes to compare, if available.

A list (no way exhaustive) of vital items to look for on ECG:

- New ST elevation myocardial infarction (STEMI)/nonspecific changes, before and after the procedure
- Arrhythmia (brady/tachy/heart blocks)
- Signs of electrolyte imbalance
- Identification of old infarcts, hypertrophy, atrial fibrillation (AF)—which could influence the management in catheterization laboratory and after
- Outcome and success of PCI (especially primary PCI)

TABLE 2.2: Postcatheterization checklist.

C (Check)	*R (Reset)*	*E (Explain)*
Symptoms	*Supine position*	
Hemodynamics	Premedications before sheath removal	Postcatheterization laboratory care • Hematoma • Skin changes • Allergic reaction • Bleeding • Fall in urine output • Chest pain/ arrhythmia/blackouts • Breathlessness/ankle swelling
Heart rate	Atropine	
Blood pressure	Midazolam	
Respiratory rate	IV fluids	
SpO_2	Lignocaine	
Temperature	Fentanyl	
Groin (femoral access)	Other	Life after PCI
Hematoma		Stop smoking
Bleeding		
Measure thigh		Diet
Lungs (crepitus = heart failure)		Medications
IV lines	Sheath removal	Clopidogrel/ticagrelor/ prasugrel
Fluids	Vascular closure device	Aspirin
IV drugs	Mechanical compression device	Statin
Urine output	Manual compression	ACEi/ARB
CBG	Radial bandaging	Beta-blocker
Oral drugs prescribed	TR band	Spironolactone
Aspirin	Inflation	Nitrates/sorbitrate GTN/other antianginals
	Deflation 15 minutes	
	30 minutes 45 minutes	
	60 minutes 75 minutes	
Clopidogrel/ ticagrelor/ prasugrel		Oral hypoglycemics (diabetes medications)
Statin		Restarting nephrotoxics

(ACEi: angiotensin-I converting enzyme inhibitor; ARB: angiotensin II receptor blocker; CBG: capillary blood glucose; GTN: glyceryl trinitrate; IV: intravenous; PCI: percutaneous coronary intervention; SpO_2: oxygen saturation)

Bedside Echo

A complete discussion on echocardiogram (with or without stress) and the pivotal role it plays in a patient undergoing PCI is way beyond the scope of this book.

There is no way a patient should be enlisted for an elective PCI or catheterization laboratory procedure without a prior echocardiogram.

But this discussion is about two issues beyond that as follows:

1. A bedside echocardiogram before elective case
2. A bedside echocardiogram before emergency or primary PCI (PPCI) in a STEMI.

A bedside echo essentially should be done by the catheterization laboratory doctors—cardiologists/juniors and does not need to be a detailed echocardiogram. The vital role of this bedside echo is to rule out any acute changes in the heart since the last echo for an elective case.

Although in emergency case, the bedside echo might really be the only heart image available and no comparison might be possible.

The essential points that should be looked for at the bedside and be evaluated and if found may need further discussion/consideration before undergoing PCI are as follows:

- New valvular heart defect—mitral regurgitation (MR) is common in acute anterior wall myocardial infarction (MI)
- Ventricular septal defect (VSD)—another common occurrence in acute MI
- EF, acute heart failure, and fluid overload
- Acute changes in regional wall motion abnormalities
- Left ventricular (LV) clot if detectable (another occurrence in acute MI)
- Post PCI—a pericardial tamponade from a coronary artery dissection is another important finding to not miss.

This should outline the importance of a bedside echocardiogram machine at the catheterization laboratory.

CARE—A: AVOID

Medications to Avoid

An integral and vital part of the preprocedure checklist is medication list to avoid to minimize the risk of complications.

The list of medications that should be avoided or stopped before the procedure can be extremely long, but here are a few which should not be overlooked:

- Nephrotoxic drugs to be discontinued (ideally) 48 hours prior to contrast injection [metformin, angiotensin-I converting enzyme inhibitor (ACEi), angiotensin II receptor blocker (ARB), nonsteroidal anti-inflammatory drugs (NSAIDs), and aminoglycosides].
- Diuretics [depending on heart failure and stability vs. risk of contrast-induced nephropathy (CIN)]
- Oral anticoagulants (OAC)
- All oral hypoglycemics (see diabetic patient before PCI mentioned further).

Nephrotoxic Drugs and Estimated Glomerular Filtration Rate Calculation and Prevention of Contrast-induced Nephropathy

The development of CIN also referred to as contrast-induced acute kidney injury, is a significant complication of intravascular contrast medium—use of which is linked with excessive morbidity and mortality. Although the serum level of creatinine is widely used in the diagnosis of renal impairment in CIN, the serum level is an unreliable indicator of kidney function because creatinine is not a real-time biomarker. Both glomerular filtration rate (GFR) and creatinine clearance reflect filtration of creatinine. However, GFR is considered a more accurate index of kidney function. GFR provides a more accurate account of working nephrons that is based on glomerular filtration. The National Kidney Foundation Kidney Disease Outcome Quality Initiative recommends that clinicians should use eGFR calculated on the basis of the serum level of creatinine. Patients with a GFR < 60 mL/min/1.73 m^2 have considerable loss of nephron units.

Risk score for CIN: In an attempt to provide an aid toward a rapid bedside identification of patients at increased risk for CIN, several risk prediction tools have been recently developed which can be used also for institution of quality improvement interventions aimed toward the reduction of contrast nephropathy and nephropathy requiring dialysis, in such patients **(Tables 2.3 and 2.4)**.

Calculation

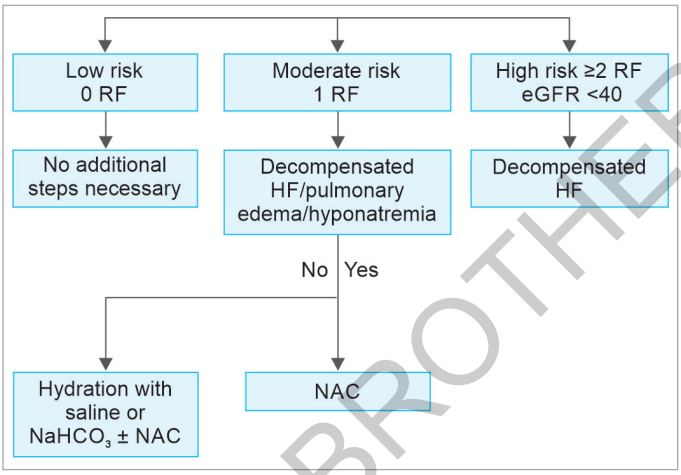
- Percent risk of CIN can be roughly calculated by multiplying serum creatinine concentration in milligrams per deciliter by 10.
- *Volume of contrast media:* Some studies found a correlation between the volume of contrast given and the risk of

TABLE 2.3: Cardiovascular research foundation risk score for contrast-induced nephropathy.

Risk factors	*Integer score*
Hypotension	5
Intra-aortic balloon pump	5
Congestive heart failure	5
Age > 75 years	4
Anemia	3
Diabetes	3
Contrast media volume	1
Serum creatinine > 1.5 mg/dL or eGFR < 60 mL/min/1.73 m^2	4 2 → 40–60 4 → 20–40 6 → <20

TABLE 2.4: Risk score for risks of contrast-induced nephropathy (CIN) and dialysis.

Risk score	*Risk of CIN*	*Risk of dialysis*
≤5	7.5%	0.04%
6–10	14%	0.12%
11–16	26.1%	1.09%
>16	57.3%	12.6%



Flowchart 2.1: Prevention of contrast-induced nephropathy.

(eGFR: estimated glomerular filtration rate; HF: heart failure; NAC: N-acetylcysteine; $NaHCO_3$: sodium bicarbonate; RF: risk factor)

nephropathy. The limit was 5 mL of contrast per kg body weight up to a maximum of 300 mL divided by serum creatinine concentration in mg/dL. Nephropathy developed in 21% of the patients in whom the total volume of contrast exceeded the formula amount, compared with 2% ($p < 0.001$) of patients in whom the contrast volume fell within the prescribed limit.

- *Types of contrast media:* Low osmolar agents reduce the incidence of CIN in comparison to high osmolar agents. So, low osmolar agents (iohexol, ioxaglate—osmolality 600–900 mOsm/L) are used frequently. The newest agent is isosmolar (iodixanole, 300 mOsm/L). It was seen to reduce the incidence of CIN in high-risk patients, but larger randomized controlled trials (RCTs) are needed to verify this encouraging result.

Prevention (Flowchart 2.1)

- Avoid dehydration
- Contrast volume and frequency of administration should be minimized. Avoid repeat injection within 72 hours.
- Low osmolar/isosmolar contrast
- Nephrotoxic drugs are to be discontinued 48 hours prior to contrast injection (metformin, ACEi, ARB, NSAIDs, and aminoglycosides).

Hydration: The experimental and clinical studies support the use of IV hydration to prevent CIN, especially in patients with azotemia at high risk. IV sodium chloride 1 mL/kg/h (max 100 mL/h) 12 hours pre- and postcontrast (24 hours total infusion duration) can be given as a protocol in every patient who needs intervention. If there is CHF or low LVEF (<40%), IV fluid should be infused 0.5 mL/kg/h (maximum 50 mL/h) 12 hours pre- and postcontrast (24 hours total infusion duration).

In emergency procedure—0.9% normal saline (NS): 3 mL/kg/h bolus infusion 1 hour before contrast administration followed by an infusion of 1.5 mL/ kg/h during the procedure and for 4 hours thereafter.

Sodium Bicarbonate Preparation

Sodium bicarbonate in some small studies has been observed to reduce incidence to CIN compared to NS. Add 6.5 amps of 7.5% w/v sodium bicarbonate (150 mEq) in 1 liter of 5% distilled water, 3 mL/kg/h of this solution for 1 hour before contrast administration, and then for 6 hours afterward. (In diabetics—mixing sodium bicarbonate in 1 liter of sterile water.)

N-acetylcysteine (NAC): Its value in preventing CIN is controversial. Dose—NAC

- 600–1,200 mg capsules PO every 12 hours × 4 doses.
  Two doses precontrast and two doses postcontrast is optimal.
- *Emergent procedure*: One dose before and three doses postcatheterization procedure is acceptable (every 12 hours × 4 doses total).

Hemodialysis and Hemofiltration

Most people can safely wait 24–36 hours after contrast exposure until next hemodialysis treatment.

Further Discussion

There should be a further discussion regarding this topic. The risks and benefits of the aforementioned strategies to prevent contrast-induced nephropathy (CIN) remain a subject of debate. Let us consider a patient with STEMI who would need a primary PCI to survive—in this condition, the risks of CIN and the preventive methods are massively trumped over by the benefits of PCI.

Even in elective circumstances, we need to think about the detrimental effects of the preventive strategies.

- Hydration—although a very important step to prevent CIN, it can have extremely dangerous effects to precipitate or worsen heart failure in a patient with certain/suspected coronary artery compromise.
- Nephrotoxic drugs are a long list and although stopping most of them for 1–2 days, including metformin, can be harmless, stopping diuretics, ACEi, and ARBs can be difficult in some situations of significant heart failure—a risk versus benefit needs to be considered in every individual case.

- The benefit of NAC is still under controversy and leaves a scope for further research in this area.

Oral Anticoagulants

The next major complication to avoid is bleeding, and the major culprit to avoid is OAC. In this context, we should start by clearing off, that anti-platelets are a vital component of PCI (page 19 of this chapter) and it cannot fall under the group of avoid drugs. Antiplatelets are necessary evil in PCI, while OAC are the difficult enigma. Also note, a rescue PCI after a failed thrombolysis within 90 minutes of thrombolytic agent and heparin [or low-molecular-weight heparin (LMWH) is also a necessary risk taken in life-threatening situation.

This leads us to consider—in elective/stable situation, it is good (or recommended) to avoid probleeding agents such as OAC, but it is not an absolute step in emergency settings or with moderate–high risk of thrombotic complications of stopping OAC. Let us elaborate.

In order to reduce bleeding (both at access and nonaccess sites) during and shortly after cardiac catheterization, it is generally advised to stop taking OAC therapy (warfarin or direct OAC). However, the drawbacks of stopping OAC include the possibility of ischemic challenges, while the medicine is held and the extended time required to recover to a therapeutic INR after resumption (with warfarin). It is a tug of war between thrombotic risk of withholding OAC and bleeding risk of the procedure (increased in emergency, femoral access, complicated and long procedure, rotablation, previous bleeding history, low platelets, and bleeding risk score mentioned in **Table 2.5**).

The subset of patient with moderate–high risk of thrombotic complications include:

- Previous metallic valve
- AF with previous embolic event such as stroke
- Previous unprovoked venous thromboembolism (VTE)
- LV clot (new due to MI or prior)

Strategies for moderate–high thrombotic risk patients:

- Continue OAC and attempt for transradial route. Transradial access is much superior to transfemoral access (even with uninterrupted OAC). Contralateral transradial or transulnar access is also superior to transfemoral access to prevent bleeding complications in these subset of patients, if transradial access is difficult.
- If OAC is interrupted, bridging with LMWH is often recommended (consider bleeding risk).
- Hold warfarin for 48–72 hours and bridge with LWMH. Restart warfarin after 24 hours and continue LMWH till INR in therapeutic range.
- Hold direct OAC for 24 hours, bridge with LMWH, and then restart after 24 hours.
- If supratherapeutic INR before procedure—can consider postponing till INR < 1.8 if elective procedure (bridge with LMWH, if necessary). If emergency, then can consider prothrombin complex concentrate.

TABLE 2.5: NCDR cathPCI bleeding risk score.

Variable	*Score*			
STEMI	No	Yes		
	0	15		
Age (years)	<60	60–70	71–79	>80
	0	10	15	20
BMI (kg/m^2)	<20	20–30	31–39	>40
	15	5	0	5
Previous PCI	No	Yes		
	10	0		
Chronic kidney disease	No	Mild	Moderate	Dialysis
	0	10	25	30
Shock	No	Yes		
	0	35		
Cardiac arrest <24 hours	No	Yes		
	0	15		
Female	No	Yes		
	0	20		
Hemoglobin	<13	13–15	>15	
	5	0	10	
PCI status	Elective	Urgent	Emergency/ salvage	
	0	20	40	

(BMI: body mass index; NCDR: National Cardiovascular Data Registry; PCI: percutaneous coronary intervention; STEMI: ST elevation myocardial infarction)

Evidence from multiple studies have shown that these strategies individually and together have reduced bleeding/vascular and major adverse cardiovascular event (MACE) or cerebrovascular events.

To summarize, the ideal strategy for thrombotic high-risk and bleeding low-risk patients—continue OAC, attempt for transradial access route, and monitor vascular complications.

For situations in which the bleeding risk is high and the ischemic risk of withholding OAC is low, the optimal timing for withholding OAC, if at all needed, is mentioned further:

- *Warfarin*: 3 days before the procedure for a target INR < 1.8 for transfemoral procedures and < 2.2 for transradial procedures
- *Dabigatran*: 24 hours (eGFR ≥ 80), 36 hours (eGFR 50–79 mL/min/1.73 m^2), and 48 hours (eGFR 30–49 mL/min/1.73 m^2). (Dabigatran is contraindicated below eGFR 30 mL/min/1.73 m^2.)
- *Rivaroxaban/edoxaban/apixaban*: 24 hours (eGFR ≥ 30 mL/min/1.73 m^2) and 36 hours (eGFR 15–29 mL/min/1.73 m^2).

Restarting Oral Anticoagulants after Percutaneous Coronary Intervention

Difficult decisions do not end with OACs once the patient leaves the catheterization laboratory in stable condition. A difficult question comes back haunting (usually from the juniors on the wards)—now what? Which to continue and which to stop?

If the indication of OAC is unavoidable—previous metallic valve, AF, previous unprovoked VTE or LV clot (new due to MI or prior), then the use of "triple therapy" (DAPT plus anticoagulation) is an unavoidable situation. There are a lot of guidelines, but generally, it is not recommended for most patients due to an increased risk of bleeding. If triple therapy is initiated after PCI, then:

- OAC + clopidogrel + aspirin (<100 mg daily) for first 30 days
- OAC + clopidogrel for 6–12 months
- OAC to continue after that (lifelong, if indicated)

Bleeding

As mentioned in the context of OAC, bleeding risk of the procedure depends on multiple factors and is increased with the use of OAC, in emergency, femoral access, complicated and long procedure, rotablation, previous bleeding history, low platelets, and factors in catheterization PCI bleeding risk score mentioned in **Table 2.5**. A careful consideration and calculation of bleeding risk score should lead to a judicious approach before a PCI (especially in elective cases).

Bleeding is the most common complication following PCI and is associated with an increased risk of other adverse outcomes. Bleeding was defined as occurring at percutaneous entry site, during or after catheterization laboratory visit until discharge, which may be external or a hematoma >10 cm for femoral, >5 cm for brachial, or >2 cm for radial access or retroperitoneal or gastrointestinal or genitourinary or other unknown origin during or after catheterization until discharge and required a transfusion, prolonged hospital stay, and/or a drop of hemoglobin >3.0 g/dL [National Cardiovascular Data Registry (NCDR), cathPCI data). There are other definitions of bleeding in PCI **(Table 2.6)**.

TABLE 2.6: TIMI bleeding definitions.

Major	ICH ≥5 g/dL, decrease in Hb% concentration ≥15% absolute decrease in hematocrit
Minor	Observed blood loss ≥3 g/dL decrease in Hb% concentration ≥10% decrease in the hematocrit No observed blood loss ≥4 g/dL decrease in Hb% concentration ≥12% decrease in the hematocrit
Minimal	Any clinically overt sign of hemorrhage associated with <3 g/dL decrease in Hb% concentration <9% decrease in hematocrit

(Hb: hemoglobin; ICH: intracerebral hemorrhage; TIMI: thrombolysis in myocardial infarction)

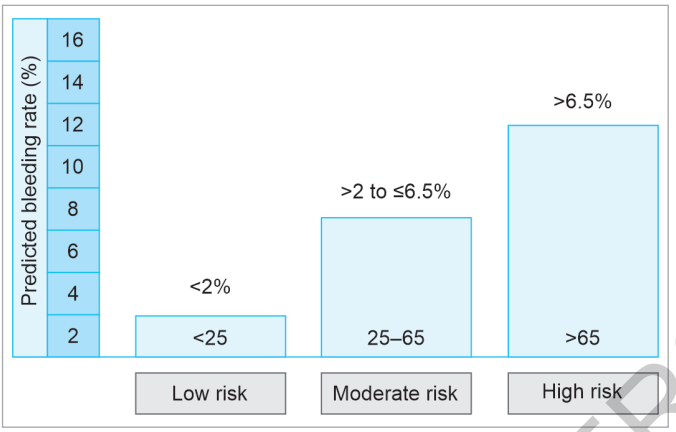


Fig. 2.1: Risk of post-percutaneous coronary intervention (PCI) bleeding.

Bleeding Scores

National Cardiovascular Data Registry data has derived a bleeding score to predict bleeding after PCI which includes both acute and chronic cases, (i.e., in ACS or chronic stable patients), whereas there are others which predict bleeding in acute situations or after use of specific agents. A bleeding score calculation is necessary to calculate the bleeding risk and to use agents during PCI accordingly to avoid post-PCI bleeding which is correlated with increased mortality **(Table 2.5 and Fig. 2.1)**.

Strategies to Reduce Bleeding Risk

- *Activated clotting time (ACT) to desired level:* ACT should be kept between 200 and 250, if glycoprotein IIb (GPIIb)/IIIa inhibitors are used, otherwise between 250 and 300, if it is not used. ACT above 350 has not only been demonstrated to increase the risk of bleeding but also increase the ischemic complications such as MI, death, or revascularization.
- Addition of GPIIb/IIIa inhibitors with unfractionated heparin (UFH) increases the risk of access site bleeding in patients with high bleeding score. It seems reasonable that appropriate dosing of antithrombotic agents based on weight and renal clearance is an important step to reduce bleeding risk.
- Bivalirudin was shown to reduce bleeding in the earlier trials but has shown to increase acute stent thrombosis in recent trials. Its use is debated but in high bleeding score patients, it still remains a choice.
- Change of access from femoral to radial in high bleeding score patients may help.

CARE—R: RESET

Medications to Reset

- DAPT
- Statins

Tips & Tricks in Interventional Cardiology

Salient Features

- Presents a detailed exposition of the concepts using a simple and student-friendly approach
- Includes an introductory discussion on who needs percutaneous coronary intervention (PCI)—backed up by current evidences and data sources
- Contain an overview of cardiological vascular anatomy and radiological views
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- Overall an up-to-date and comprehensive book.

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Over the years of practice in the field of cardiac intervention, he has gathered countless experiences and immense reputation. Through this book, he ventures to share a part of his eternal knowledge for all.

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