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CATH LAB
PRACTICALS

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Dedicated to

My Father, Mr OP Mishra who has been my “Role Model” and my
Mother Mrs Shakuntala Mishra who is my
“Spiritual Mentor”
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Although percutaneous coronary intervention (PCI) is considered as a safe procedure, it is not free of complications. Complications in the cardiac catheterization laboratory can be attributed to the condition of the patient upon arrival to the catheterization laboratory (cath lab) or to the technical aspects of the actual procedure. The patient-level complications can be attributed to the state of the disease, for example, patients with cardiogenic shock, acute myocardial infarction, or stent thrombosis pose a higher risk for procedural complications, such as cardiac death, bleeding, and arrhythmias, when compared to stable patients undergoing elective PCI. This complex patient subset frequently requires more intense monitoring, often an anesthesia consult and hemodynamic support with pressors or devices, such as an intra-aortic balloon pump or Impella, to secure a safe procedure.

Other measures that need to be addressed prior to the PCI to avoid complication are the optimization of the anticoagulation protocol; appropriate selection of access for the procedure, either radial or femoral; and the equipment that will be used during the PCI. Procedural PCI complications are often related to the operator technique and skills; to the nature of the lesion, calcified, torturous, or thrombotic; and to the device performance, including malfunction or misuse. These complications can lead to catastrophic events in the cath lab, such as vessel perforation, spiral dissection, distal embolization, and the no-reflow phenomenon. Device-related complications could be attributed to broken wires, deformed stents, stripping stents from the balloon, stuck balloons, rotablator burrs, etc.

Clearly planning ahead of the procedure and having the right equipment and back-up to perform the procedure are essential to minimizing complication rates. Bailout of complications in the cath lab is an art in itself, and although one complication during the procedure can be forgiven, two or more sequential complications cannot. The manual on cath lab complications focuses primarily on the procedural-related complications and is a useful guide to gain familiarity with the options and the modalities to reduce the complication rate and to treat the complication safely if it occurs. The best way to take care of complications is to avoid them, and this can be achieved with proper preparation of the procedure components—the operator, device, patient, and lesion.

Among the most common complications in the cath lab are access site complications, which result in a higher bleeding rate. But with the migration
of access from femoral to radial, the rate of vascular-related complications has been declining. Radial access, however, is not free of complications. Once the complication occurs, it is imperative to identify the complication and to treat it as soon as possible, even at the expense of differing the planned procedure, and even if the complication does not seem to be life threatening. Each device has its own potential complications, and the operator needs to be familiar not only with the use of the device but also with managing the complications that the device may cause.

One approach to minimize PCI complications is to shorten the procedure time. Staging the procedure should be considered, and it is important to know when to stop if things are not going as planned. Usually when one strategy or device does not perform as planned, it is important to not force it on the vessel and to change the device or strategy or to abort the procedure, which is still better than experiencing the complication. Managing complications is a team effort, and therefore once a complication is encountered, it is wise to call for help and the rest of the team, including nurses, technologists, experienced operators, and an anesthesiologist. Time is of the essence when treating complications, and the more time that passes, the worse the outcome. Other noncardiac but procedural-related complications that may impair patient outcomes are induced contrast nephropathy, radiation exposure, and burns. Risk assessments of the procedure and risk adjustment are essential for planning and reporting the rate of complication per institution, especially as we move to public reporting.

Finally, we should remember that as long as we perform PCI, we will encounter complications. Therefore, learning how to bailout from these complications, and how to manage them safely is as important as knowing how to perform safe PCI. This manual is a useful educational tool to get you and your patient safely through the procedure, even if gets complicated.

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I fly frequently. Dividing my time between the Eternal Heart Care Centre (Jaipur) and the Mount Sinai Hospital (New York), I rely on commercial airlines. Boarding each flight, I expect to lift-off, proceed to my destination, and land successfully, with minimal discomfort and no complications. Whatever turbulence may arise in the course of travel, I expect my plane’s captain and crew to have the proper training, technology and temperament to manage it.

Our patients expect no less from us. As percutaneous coronary intervention has matured, procedural success has soared toward 98–99% and complication rates have plummeted for even the most complex cases. As experience grows and equipment further evolves, ambitions are similar.

The difference between good and ideal is measured in how we handle the tough cases. In the *Cath Lab Practicals*, Professor Sundeep Mishra and his team have taken on the ambitious task of preparing interventional cardiologists for the quandaries, challenges and emergencies that can imperil success and safety in the cardiac cath lab.

With wit, savvy, clinical examples and a touch of philosophy, Professor Sundeep Mishra and his colleagues cover a broad array of potential problems in the course of coronary and structural intervention. Interventional cardiologists in practice and in training, nurses, technicians and staff would do well to digest these highly-readable chapters, which detail solutions to challenges ranging from the rare (device embolization, Chapter 2) to the routine (problems with guide support, Chapter 7). By meeting percutaneous coronary intervention’s most feared complications head on, the text helps demystify its most technically demanding procedures. In review of complications of rotational atherectomy (Chapter 10), for example, the authors comprehensively explore the terrifying event of burr entrapment and provide practical options for management.

“The key is not just to know,” the authors write, “but to know that you know.” The confidence to embark on the most complex interventional procedures grows from a comfort in one’s ability to manage even the most dire complications. With such confidence, we are more likely to give the best, we have to offer to the patients who need it most. Professor Mishra and colleagues are to be congratulated on this textbook that will help all members of the interventional team feel more confident that they
can deliver the best possible interventional care, even when the going gets tough.

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Since renaissance science has evolved as a body of empirical, theoretical, and practical knowledge about the natural world, produced by scientists who emphasize the observation, explanation, and prediction of real world phenomena. The method applied is differentiation and randomized controlled trials (RCTs) have now become the gold standard for causal inference in medicine. However, to observe a difference, there are essentially 3 requirements: presence of control, sufficient number of participants to bring out a meaningful difference, and finally, a stable environment to study the difference. However, what to do when none of these conditions are satisfied? For example, cath lab complications: neither there is a control, nor sufficient numbers occurring predictably over a course of time, nor a stable environment!

The situation is somewhat akin to a “Fighter Pilot” although the essential difference here is that operator’s life is not at risk (unlike the fighter pilot). Thus, to take a cue from aviation profession where procedural know-how has been over the years extensively externalized, verbalized and documented. It is verbalized not only in clearly structured instruction manuals formulated over declarative knowledge, i.e. on technical and scientific aeronautical data but also incorporated into virtual reality aviation simulators equipped with sophisticated board computers, FMS, programmed to mimic variety of real-life scenarios. Thus, throughout the training pilots learn to master the knowledge; proceeding from simple to routine to unexpected scenarios. These established teaching processes assure objective assessments of achieved levels of professional competence by all trainees across the board independently from local circumstances and dispositions. Recently, based on cognitive research, it has been shown that acquisition of know-how may be enhanced by providing the trainees with additional contextual data embedded into concrete tasks.

However, in contrast to field of aviation the percutaneous coronary interventions (PCI) procedural knowledge has not been systematically verbalized, and has remained so despite over 30 years of clinical PCI practice (perhaps because of rapid changes in PCI technology). Thus, while textbooks on PCI typically beam with evidence-based data derived from numerous devices-driven trials, the fundamental cognitive processes required for the actual PCI performance are scanty; however, the “tips and tricks” which seem essential to procedural skill transfer, cover only a tiny corner in the huge PCI decision space and are by far not enough to provide for the needs. The efficacy of traditional “trainee-mentor” knowledge transfer is highly dependent on ability of trainees to perceive and mentors to explain and to demonstrate; marked heterogeneity of professional PCI competence result. With this view, we embarked upon this manual to build up the collective memory of cases, to
develop cognitive teaching programs based on retrieval of expert knowledge rather than mere mentor-guided empirical approach, to try build a mental library of cases, to try to begin to close the gap between the training modus in aviation and PCI. In other words, develop a “Cath Lab Manual” akin to aviation manual.

Sundeep Mishra

You take the red pill – you stay in Wonderland, and I show you how deep the rabbit hole goes
First of all, I would like to thank all my teachers who brought me to a level, where I am able to write a manual. In particular Professor VK Bahl who is also my mentor and my guide as also Professors SC Manchanda, SS Kothari and of course Ron Waksman who played down my limitations, my mistakes and my weaknesses to guide me all along. I would also like to acknowledge all the contributors to the cases and chapters. I would like to thank the Director of AIIMS, Professor MC Misra (Center Chief) Professor Balram Airan (Head), all my colleagues and staff, All India Institute of Medical Sciences (AIIMS), New Delhi, India, Cardiological Society of India-National Interventional Council (CSI-NIC) and all my well-wishers. I am also deeply indebted and grateful to my wife Deepti Mishra, who not only sacrificed herself so that I could complete this monumental task but also encouraged me to do it, and gave me practical advice all along. Without her help, this work would not have seen “Light of the Day.” I would also like to thank my kids—Gargi Aditi and Badri Vinayak who allowed me to “sit on the computer”.
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INTRODUCTION

Extremely sick patient is someone whom an experienced physician can easily identify standing at the foot end of the bed. Extremely sick patient is usually cold, clammy, in cardiogenic shock and in respiratory distress from congestive heart failure. High risk is defined as the probability and more importantly, the consequence, of abrupt closure of the dilated site, occlusion of large side or distal branches or widespread microvascular obstruction with spasm. Abrupt closure of a large epicardial artery or of a side or distal branch is recognized by standard angiogram while occlusion at the microvascular level is seen as slow flow or no-reflow secondary to showers of material from ruptured plaques. The risk of cardiogenic shock is higher in patients with left ventricular dysfunction (ejection fraction <30%), target vessel supplying more than 50% viable myocardium, circulation to both papillary muscles compromised and a high jeopardy score >3. Other conditions that can cause cardiogenic shock are incessant ventricular arrhythmias or severe aortic stenosis.

Cardiogenic shock (CS) is a state of inadequate end-organ perfusion primarily due to cardiac pump failure. It is characterized by persistent hypotension with a SBP less than 80–90 mm Hg or a MAP less than 30 mm Hg below baseline with a CI less than 1.8 L/minute/m² without support or less than 2.0–2.2 L/minute/m² with support with an adequate or elevated filling pressure.1 Acute MI is the most common cause of CS. Approximately 5–8% of STEMI and 2.5% of non-STEMI are associated with CS. CS is the leading cause of death in patients with AMI with mortality rate of ≈50%.1,3 Early revascularization, compared to initial medical stabilization, improves long term survival and is the recommended strategy for AMI patients presenting with CS due to LV failure.2

Percutaneous cardiac assist devices have been historically employed in AMI with CS for mechanical hemodynamic support.3 In the setting of CS complicating AMI, Intra-aortic Balloon Pump (IABP) counter-pulsation is the most widely used form of mechanical hemodynamic support. IABP is a simple and safe device to insert. It relies on left ventricular function and a stable cardiac rhythm, which may not always be present in CS, to achieve its full potential. The estimated augmentation of cardiac output by 0.5 L/minute may not be sufficient to meet the demands of sick patients in CS without the additional use of deleterious vasoactive agents.3 Randomized clinical trials
have not shown either 30 day or 1-year mortality benefit with IABP in patients with CS complicating AMI.\textsuperscript{2,4,5}

Newer percutaneous ventricular assist devices (PVADs) that provide emergent and effective hemodynamic support in high risk patient population that overcome the limitations of IABP are increasingly being utilized.\textsuperscript{1,3,6} The Tandem Heart\textsuperscript{TM} and Impella 2.5/CP\textsuperscript{TM} or extracorporeal membrane oxygenator (ECMO) are the more frequently used PVADs. These devices differ in their insertion technique and mechanism of action. Potential major complications include limb ischemia, bleeding, and transfusion requirements.\textsuperscript{3,6}

The Tandem Heart\textsuperscript{TM} is a left atrial to femoral artery bypass which can provide up to 3.5–4 L/Minute forward flow and active hemodynamic support. Device insertion, management and discontinuation require experienced staff. Clinical studies have shown that the Tandem Heart\textsuperscript{TM} provides significantly better hemodynamic support compared to IABP in CS but at no difference in 30 day mortality.\textsuperscript{6} Anecdotally several cases have shown effective utility of Tandem Heart\textsuperscript{TM} in providing effective support in refractory CS and during high-risk cardiac interventions.\textsuperscript{7}

The Impella 2.5\textsuperscript{TM} device provides partial hemodynamic support that directly unloads the left ventricle. It requires one femoral artery access and, unlike the Tandem Heart, provides a maximal flow rate of $\approx$2.5 L/minute. It is considered a safe and easy to use, providing effective hemodynamic support during high-risk PCI\textsuperscript{8} but has not shown superior outcome compared to IABP at 30 days.\textsuperscript{9} Recently, Impella CP\textsuperscript{TM} catheter has been introduced and it can provide flows averaging 3 L/minute.

Extracorporeal membrane oxygenation can assist both right and left ventricles. It can be used either venovenous or venoarterial depending on the placement of the cannulas and the ventricle that needs the support. ECMO has been used in various settings, including cardiogenic shock and postcardiac and respiratory arrest.\textsuperscript{1}

Despite the lack of randomized clinical trials, higher rates of adverse events, and no proven survival benefits, the continued use of PVADs in CS is recommended.\textsuperscript{6} The ability to initiate and maintain timely and effective hemodynamic support using PVADs in the catheterization laboratory has changed the way CS is approached in certain patient populations and is considered a major advancement in interventional cardiology. More clinical trials and studies are needed to further evaluate their effectiveness, comparative merits and adverse effects.\textsuperscript{3,6,7}

**CASE EXAMPLES**

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**Case 1**

*Critical Aortic Stenosis Presenting in Cardiogenic Shock*

*Introduction:* Aortic stenosis is one of the most common valvular abnormalities encountered in our practice. Once aortic stenosis becomes
symptomatic, the mortality rate is approximately 50% in the first 2 years. The treatment is typically valve replacement surgery, however in the setting of advanced age or multiple co-morbidities patients may be denied, secondary to increased surgical risk. Transcatheter aortic valve replacement (TAVR) has been proven to be beneficial in this subset of patients. That being said TAVI itself has certain contraindications including LVEF <20% and cardiogenic shock requiring inotropes.1

An 82-year-old male first presented to clinic with symptomatic aortic stenosis (Fig. 11.1). His significant past medical history included: paroxysmal atrial fibrillation, chronic kidney disease stage I, prostate cancer in remission and GI bleed. He was initially randomized to the surgical arm of PARTNER II trial. Prior to scheduled surgery, he presented to the emergency department in cardiogenic shock. The patient was placed on mechanical ventilation, inotropes and vasopressors initially. Despite this, he remained in shock with multisystem organ failure. A repeat echocardiogram demonstrated and LVEF of less than 20% down from 55% to 60% a few months prior. Soon after a Tandem Heart™ was placed in the cardiac catheterization laboratory (Fig. 11.2). A balloon aortic valvuloplasty was considered, however, since his cardiac output drastically improved with the Tandem Heart™ this was deferred. The patient was withdrawn from the PARTNER trial and received #26 Edwards Sapien™ valve via transfemoral approach. There was residual mild perivalvular leak shown on postdeployment transesophageal echocardiogram. Postprocedure, he was able to be weaned off of inotropes, vasopressors as well as the Tandem Heart™. At discharge, his organ function had returned to normal. He was seen in clinic several months after completion of cardiac rehabilitation and his symptoms had improved to NYHA class I.

Fig. 11.1 Computed tomography scan of the chest showing severely calcified, trileaflet aortic valve with severe aortic stenosis
Analysis of the case: We present a case of a patient with aortic stenosis in cardiogenic shock secondary to acute left ventricular systolic failure who ultimately went on to receive a TAVI and demonstrated good functional recovery. There is little data in the literature in this subset of patients. Fudim et al. all presented 2 case reports where TAVI was used successfully as a bailout in patients with cardiogenic shock secondary to bioprosthetic valve failure. The most published data on the subject comes from D’Ancona et al. demonstrated technical feasibility and 19%, 30 day mortality in 21 patients with cardiogenic shock undergoing TAVI. The Tandem Heart™ percutaneous ventricular assist device is a left atrial to femoral artery bypass system using a centrifugal pump that can deliver flow rates up to 4L/min. It is particularly useful in this subset of patients since unlike Impella™, it does not involve crossing the aortic valve. Traditionally, balloon valvuloplasty has been the bailout procedure for patients in refractory cardiogenic shock with severe aortic stenosis. There is little data on performing TAVI in patients with cardiogenic shock, but with ventricular assist device placement this could be the next frontier for the procedure.

Case 2

Introduction: Hemodynamic support has been shown to be useful in recurrent arrhythmias secondary to STEMI.

A 52-year-old male with past history of CAD dyslipidemia, HTN, presented to a primary center with substernal chest pain and diaphoresis. He was on chronic clopidogrel therapy for a bare metal stent placed 4 months prior, and he discontinued it several days prior to presentation for an upcoming orthopedic surgery. His initial EKG showed an inferior STEMI (Fig. 11.3). He was given thrombolytics and transferred to a tertiary care
facility. He underwent defibrillation for unstable ventricular tachycardia en route. Emergent angiography was performed with five defibrillations during the diagnostic portion of the procedure. Lidocaine and Amiodarone therapy was initiated without full resolution of his electrical instability. The RCA showed a mid-90% stenosis (Fig. 11.4), and he underwent successful drug-eluting stenting to his RCA. Despite revascularization of his culprit vessel,
and antiarrhythmic therapy, he continued to have episodes of ventricular tachycardia requiring defibrillations. At this point, an Impella 2.5™ along with a Swan-Ganz catheter was placed (Fig. 11.5) for hemodynamic support and electrical stability. At this point, he did not have any more episodes of ventricular arrhythmias, but his ST-elevations did not fully resolve. A repeat angiography revealed a thrombus and dissection in his mid-RCA. After aspiration of the thrombus, IVUS showed the stent to be underdeployed with a small distal dissection. At this point, a second stent was placed in his RCA with resolution of ECG changes (Fig. 11.6). He was transferred to the cardiac intensive care unit. His Impella™ was weaned and removed in 24 hours without any recurrence of arrhythmias. He was discharged by hospital day 5.

Analysis of the case: Impella for hemodynamic support is a useful modality for electrical stability in unstable patients, which is illustrated in this case.

Introduction

This case describes the successful use of an Impella CP™ device in a 60-year-old male who presented with acute ST-elevation myocardial infarction with cardiogenic shock.

A 60-year-old male with a past medical history of HTN, dyslipidemia, peripheral vascular disease, and CAD (CABG 20 years prior with known occluded vein grafts and patent LIMA-LAD) who presented to the emergency department at an outside facility with complaints of substernal chest pain. Initial ECGs did not show any signs of ischemia. He was given nitroglycerin

Fig. 11.5 Angiography of the right coronary artery status postplacement of drug-eluting stent in middle segment with good angiographic results. Also seen is the Impella™ device in situ
initially and became hypotensive. Repeat ECG showed ST-elevations in the inferior leads (Fig. 11.7). He then developed ventricular fibrillation followed by 20 minutes of advanced resuscitation. He was started on dopamine and levophed for cardiogenic shock with persistent hypotension of 80/50 mm Hg. Patient was given TNKase prior to emergent transfer to a tertiary care facility.
Emergent angiogram showed severe native vessel disease (90% occluded left main, 100% proximally occluded left anterior descending with a small diagonal branch, 100% proximal left circumflex artery, and a 100% proximally occluded right coronary artery) and occlusion of 3 vein grafts with a patent LIMA to LAD. The native LAD after touchdown of the graft was a small vessel. An Impella CPTM catheter was then placed through the right femoral artery for hemodynamic support along with a cooling catheter in the right femoral vein. A Swan-Ganz catheter was placed in the left femoral vein for hemodynamic monitoring. Hypothermic protocol was initiated and the patient was transferred to the cardiac intensive care unit (Fig. 11.8). Initial echocardiogram upon admission revealed an ejection fraction of 30–35% with mainly inferior akinesis. Initially, he exhibited decorticate posturing and was started on paralytic due to shivering on the hypothermic protocol. Within several hours of his CCU stay, re-warming protocol was initiated because he awakened and was able to follow commands. LevophedTM was used for vasopressor support upon arrival to the tertiary care facility and was weaned off by hospital day 2.

The use of the ImpellaTM and vasopressor support continued through hospital day 1. An echocardiogram was repeated on hospital day 2 which showed an improved EF of 45%. At this point, his vaspressors were weaned off while his ImpellaTM was weaned to P5. His ImpellaTM was removed by the end of hospital day 2 due to hemodynamic recovery. Echocardiogram on hospital day 6 showed an ejection fraction of 55–60%. He was extubated on hospital day 3. He was discharged to home on hospital day 10 with full recovery.

**Fig. 11.8** Electrocardiogram showing sinus tachycardia with improved ST-segment deviations in inferior and lateral leads after Impella placement.
Analysis of the case: When one is taking care of an extremely sick patient, there is sparse evidence-based medicine to decide the choice of cardiac device to be used. ABCDs of resuscitation efforts have to be followed first that include airway, breathing, circulation and defibrillation, if needed. Once patient’s respiratory status is stabilized, attention should be turned to stabilize the patient hemodynamic status. Cardiogenic shock is a spectrum, still carrying high mortality in the range of 40–50% with all currently available left ventricular support devices. One should first assess the patient and classify shock into mild, moderate or severe shock.

LEVELS OF CARDIOGENIC SHOCK

- **Mild:** Low levels of inotropic support (Dopamine ≤5 µg/kg/min, epinephrine or norepinephrine ≤0.03 µg/kg/min)
- **Moderate:** Modest levels of inotropic support (Dopamine ≤10 µg/kg/min, epinephrine or norepinephrine ≤0.05 µg/kg/min)
- **Severe:** Maximal inotropic support (Dopamine >10 µg/kg/min, epinephrine or norepinephrine >0.05 µg/kg/min, vasopressin at any dose.

Suggested Devices

**Mild Shock:** Intra-aortic balloon pump  
**Moderate Shock:** Impella CP™ catheter  
**Severe Shock:** Impella CP/Impella™ 5.0, Tandem Heart™ or ECMO.

The choice of device depends on local expertise and comfort of the operator. The right ventricular support devices such as the Impella RP™ and Tandem Heart™ right ventricular support cannula are available now. However, there have been no randomized studies to support one device over the other to improve mortality. In our own registry of 42 patients with acute myocardial infarction and cardiogenic shock, quick escalation protocol of devices has reduced in-hospital mortality from 44% to 24% (Flow chart 11.1 and Table 11.1). These results have to be further confirmed in larger sample size. The role of cardiac assist devices in extremely sick patients is a changing landscape due to the paucity of data, cost of the devices, patient factors and support from insurance companies. Extracorporeal membrane oxygenation (ECMO) has been used in refractory cardiogenic shock patients for decades with and without cardiac or respiratory arrest. However, survival in most ECMO trials or series is less than 40%. ECMO can be performed at bedside with femoral venous and arterial access. Complications include bleeding, ischemia and venous thrombosis. Poor predictors of mortality with ECMO include age >60 years, pH >7.30, inotropic score >20, recent history of CPR, ECMO implantation during CPR and oligoanuria.
Flow chart 11.1 Suggested cardiogenic shock protocol at East Carolina Heart Institute, Greenville, NC, USA

*Cardiac power catheter can pump up to 3.5 L/min and is inserted percutaneously in the lab.
**Table 11.1** Outcomes of East Carolina Heart Institute Cardiogenic Shock Registry

<table>
<thead>
<tr>
<th>Cardiogenic shock</th>
<th>Group I (18)</th>
<th>Group II (24)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>7.7%</td>
<td>8.4%</td>
</tr>
<tr>
<td>Mortality</td>
<td>44.8%</td>
<td>24.4%</td>
</tr>
<tr>
<td>Impella insertion</td>
<td>5.5%</td>
<td>85%</td>
</tr>
<tr>
<td>Tandem heart</td>
<td>11%</td>
<td>21%</td>
</tr>
</tbody>
</table>

**REFERENCES**


“One size does not fit all”
Fig. 1.2  Circumferential rupture of the balloon catheter

Courtesy: Dr Shantanu Deshpande

Fig. 1.3  Balloon pinhole rupture
Fig. 2.1 Retrieval devices in catheterization laboratory

Fig. 2.4 Embolized stent extracted with a snare

Courtesy: Ranjit K Nath
Fig. 9.1 Thrombus extracted after thrombosuction with eliminate device