Review Article

Interventional Cardiology

Fundamentals of coronary interventional complications

Abstract

In the practice of interventional cardiology, a comprehensive awareness of the complications associated with Percutaneous Coronary Intervention (PCI), and strategies to both prevent and treat them, is an absolute necessity as one matures into a complete operator. This can be likened to the process that occurs as one develops from a beginning to experienced automobile driver. The vast majority of PCI cases are going to be straightforward and easy to navigate, such as a leisurely drive to the supermarket, with minimal twists and turns. A basic, well-planned PCI procedure will often seem just as comfortable. However, no matter how well a procedure is planned and executed, the reality is that at any point, the unexpected can occur an access site complication, coronary dissection, or even a procedural death. The early career interventionalist will logically be less prepared for this, as a student driver attempting to merge onto the interstate for the first time. These occurrences can be fraught with anxiety and self-doubt. Importantly, with time and experience, one will become quick in recognizing and managing complications. This results in improved patient outcomes, increased breadth of procedural knowledge, and higher physician satisfaction. Strategies to avoid complications will become incorporated into routine practice. This review provides a guideline of common PCI complications, along with measures to both prevent and treat them, should they occur.

Keywords: Coronary complications • Coronary dissection • Coronary perforation • Vascular access • Periprocedural stroke • Contrast-induced nephropathy • Interventional cardiology • Boards review

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Introduction

Any occurrence that results in prolongation of the procedure or causes morbidity or discomfort for the patient can be considered a complication. Types of complications during PCI range from the very general; such as death, stroke, MI, acute renal failure, and access site complication; to issues that are quite specific to coronary procedures such as coronary dissection, perforation, air embolism, vessel closure, etc. As a case transitions from diagnostic to interventional, the risk of complication is further enhanced. This is due to a number of factors including further catheter exchange, use of anticoagulation, prolonged duration, higher contrast load, and instrumentation of the coronary. Although improvements in equipment and techniques have lessened the overall risks, there remains an overall risk of major complication at ~4% for a diagnostic procedure. It can be helpful to communicate to patients in terms of number of patients out of 100 that experience a given issue.

Literature Review

In a diagnostic procedure alone, risks of the basic complications are as follows: Death 0.11% (~1/1000), stroke 0.3% (~1/333), MI 0.1% (~1/1000), significant access complication 1.8% (~2/100), Acute Renal Failure (ARF) 1.7% (~2/100) [1-3]. In

an interventional procedure, risks are as follows: Death 1.27% (~1/100) [4], stroke 0.55% (~1/200) [5], MI 7% (7/100) [6], access complication 4% (4/100) [2], ARF 7% (7/100) [7]. In a meta-analysis of PCI, radial access was found to reduce the risk of access site complications by about 10-fold, from 2.8% down to 0.3% [8]. The risk of contrast-related anaphylactoid reaction during heart procedures is 0.2% (2/1000) with 1 death in 55,000 [9]. The requirement for emergent CABG has improved from 14% in the PTCA era to 0.61% (~1/200) in the stent era [10]. It is imperative prior to beginning a case that the patient has undergone informed consent that allows for a basic understanding of the procedural risks and benefits. A reusable patient handout, written in plain language highlighting the expected benefits and risks as above, can be helpful in this regard as shown in the Table 1.

Table 1: Comparison of procedural risks between diagnostic and interventional cath lab procedures.

Risk of complications in diagnostic and interventional procedures		
	Diagnostic	**Interventional**
Access site complication	1.8% (~2/100)	4% (4/100)
Myocardial infarction	0.1% (~1/1000)	7% (7/100)
Stroke	0.3% (~1/333)	0.55% (4/200)
Acute renal failure	0.11% (~1/1000)	1.25% (~1/100)
Any complication	4% (~4/100)	19% (~19/100)

Focus on prevention of complications overlaps with techniques for procedural efficiency and simplicity. Procedural risk is enhanced based on the clinical factors and angiographic findings (for example use of ACC/AHA classification) [11], which need to be considered prior to moving forward with any case. Clinical prediction scores such as the Mayo Clinic Risk Score (MCRS) and the CHIP score can be informative to determine the risk of periprocedural mortality in PCI [12,13]. These prospectively validated models include clinical features such as age, renal function, LV function, presence/absence of shock, presence/ absence of MI, as well as angiographic features, to determine periprocedural risk.

Recall what the trials (such as COURAGE, ISCHEMIA and BARI 2D among others) are showing us about the safety of up-front medical management [14-16]. It is my practice to strongly advise against heart catheterization procedures in patients that are baseline DNR status, especially in the case of high comorbidity, as the act of rescinding DNR to perform case may not be in keeping with a patient's previously defined goals for less invasive cares. As such, in elderly DNR patients, the discussion "starts with a no" and then discussion can occur. In cases with high complexity, with high risk for complication, avoidance of ad hoc PCI is advisable. This allows for thorough discussion with patient/ family and in-depth procedural planning before returning to the Cath lab.

General principles to avoid complications

Successful procedures are the result of many individual steps performed correctly and safely, however tedious. A mantra from the military rings true: "Slow is steady, and steady is fast". There are many general principles that can be followed. Catheter-based procedures require a baseline level of "gentleness". No piece of equipment should be advanced with high levels of force. Nothing should be advanced or withdrawn within the vessel without fluoroscopy. Vascular access should be given its due–with radial approach should be pursued if possible. This step should also not be rushed. Always use fluoroscopic landmarks. If a vessel cannot be accessed with 2-3 attempts, utilize ultrasound. Avoid back wall femoral sticks. If the finder needle has entered the vessel and access is lost, or if a back wall stick is suspected, a few minutes of pressure should be applied before proceeding again.

The diagnostic coronary angiogram needs to be studied methodically including deliberate consideration of the left main, at-risk side branches, collateral perfusion, and amount of calcium. Procedural views should be selected that maximize simultaneous visualization of the guide, elongated view of the lesion, and the distal vessel. One should utilize the smallest caliber and least aggressive guide catheter and/or guidewire as possible to complete a case. Anticipate the next steps, and have the next bit of kit handy and opened. Do not try to cut corners i.e., if you are considering a procedural step, it should generally be done (for example placement of a buddy wire, predilatation, protection of a side branch). Necessary bailout equipment, as well as circulatory support devices, should be at the ready.

When wiring a vessel, the wire should be "allowed to find its own way", i.e., gently steered, redirected, and advanced with the introducer in place to improve tactile feedback, with constant visual assessment for wire bending or "debacle". The wire tip should always be moving freely during advancement, and allowed to prolapse into a J-shape if possible once the lesion is crossed. Constant redirection of the wire upon wire debacle is as necessary as it can be tedious. Predilatation balloon sizing and pressure should be conservative. Stent sizing should be appropriate to the distal vessel, with post-dilatation matching the proximal vessel, if necessary. If a complication has occurred, and has been stabilized without a perfect PCI result, this may be the time to end the case and consider another attempt later with a revised plan. Once any case is concluded, ensure the blood pressure is neither too high nor too low. It is the experience of the author that use of femoral closure device in the context of multiple blood thinning agents is not advisable, especially GPI medications or recent thrombolytics. These patients need to be allowed the proper "washout" period for the anticoagulant in question, and manual pressure employed for hemostasis.

Prevention and management of complications

Coronary dissection: This is the major cause of acute closure, often identified as a "linear lucency" within the vessel during PCI case. Note that PTCA alone can cause ~60% visible dissection and post-mortem studies confirm some small level of dissection occurs in virtually 100% of PTCA cases [17], which underscores the utility of the stent scaffold. Minor balloon related dissections are therefore commonplace and practically outside the domain of a discussion of complication. Stent edge-dissection was found to be present ~6% of the time in a prospective IVUS study, related to edge-ballooning and "landing in a plaque", and was associated with higher TLR (RR2.67) [18]. However clinically significant Guide Catheter (GC) related dissection is much rarer, occurring in ~0.1% of cases [19]. Dissections can be ranked from A-F according to NHLBI [20], which associates with increasing risk for morbidity/ mortality. A) Minor radiolucent/ hazy areas in the lumen with NO impairment of flow and NO persistent dye staining after contrast; B) Luminal linear flap that is radiolucent and that runs parallel to the vessel wall with contrast injection but still without impairment of flow or persistent dye staining; C) Contrast appears outside of the vessel lumen as an "extraluminal cap". The staining appears even after contrast clears the lumen. Such a persistent stain may be difficult to discern from type A perforation; D) Spiral dissection with persistent staining; E) intraluminal filling defects/ lucency occupying part of the vessel lumen; F) 100% occlusive. A helpful memory aid is to "Never Let One Spiral from Partial to 100%".

Prevention of dissection: The majority of significant dissection is related to the guide catheter (75%), more commonly in the RCA (50%) than left main (45%) [19]. Other risk factors include deep seating of the catheter, complex PCI, and female sex [21]. Selection of the GC should favor using the least aggressive shaping as possible to complete the case. Similarly, the guidewire chosen should be as minimally aggressive as possible as well, favoring use of coil-tipped and blunt-tipped wires over hydrophilic and tapered wires. If the GC is non-coaxial, deep-diving, or dampening, it should be switched out as soon as this is recognized. As soon as a GC engages, review the degree of coaxial fit as well as arterial waveform before injection. Change to a more suitable guide, or add side holes, if dampening present; as poor fitment of a guide is a common cause of dissection. Remember that this may add a few minutes to the case, but a dissection could add hours with substantial morbidity to the patient. Do not inject contrast if the waveform is blunted, and reduce the contrast volume and pressure settings if the vessel is very small (such as a non-dominant RCA). Balloon sizing and inflation pressure should be conservative, used only to open a channel for the stent. Stent sizing should be based on the distal vessel initially. When removing balloons, do so in a view that includes the vessel ostium in an elongated form, and apply counter back-pressure on the GC. Balloons should be pulled out a few millimeters at a time in a staccato fashion while still within the vessel, as they tend to adhere to the wall initially, which will "suck in" the guide. One can also consider forward pressure on a stent balloon first to free it from the device.

Treating coronary dissection: It is rare the case that coronary dissection is managed conservatively, although this strategy is described if flow is maintained [19,22]. It is imperative to maintain wire position to allow for further interventional steps. With minimal or questionable dissection, one can consider use of GPI medications or observation. Any clear dissection type B or higher should be promptly and efficiently covered with a generous length of stent that covers the whole of the dissection [23,24]. Further injections should be minimized to avoid propagation. If the dissection is ostial, this should be addressed first, in an effort to prevent propagation into the aortic root. Be wary that geographical miss of the ostium will have an effect on behavior of the guide. If a dissection has occurred and a wire is not in place, different types of wires may be tried to attain the distal vessel. Preferable are floppy coil tip wires, with strict visual attention to spiraling or debacle of the wire tip during advancement [24]. The wire is more likely to be within true lumen if it remains straight with advancement, or if clearly entering side-branches. If knuckling or spiraling, it is likely in the dissection plane. The Suoh-3 wire is a rope-coil wire with high flexibility and torquability that has been shown to be a good choice in a case series of dissection [25]. If the first wire has entered a dissection plane, leave it in place and use a second wire with differing characteristics. If the dissection is ostial, consider an alternate guide shape than that which caused the dissection. Confirmation of true lumen position can be done with injections through an over the wire balloon, or use of OCT/ IVUS.

Exotic "bailout" re-entry techniques can be applied that come from the CTO world and can include cutting balloon, suction of blood from subintimal space through an OTW balloon followed by re-entry with a puncturing wire with 90-degree bend, ("STRAW technique"), Stingray re-entry, subintimal tracking and spontaneous re-entry ("STAR technique"), use of balloon in the subintimal space to create fenestration ("AFR technique") etc., [26,27]. The STAR technique amounts to advancement of a knuckled wire until it spontaneously re-enters true lumen, which typically occurs at a bifurcation. This can result in loss of important side branches, and is considered a last resort. If the distal true lumen cannot be attained through whatever technique, and the area of at-risk myocardium is high, urgent CABG may be necessary; required in ~6% of cases in a large series [19].

Aortic root dissection: Aortic root dissection occurring as part of a catheter-based procedure is a very rare complication, occurring in ~0.02% of cases, and usually involving the RCA ostium [28]. The most common associated guide is Amplatz shape [29]. Root

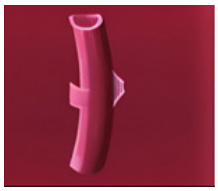
dissections have been described as Class I which involves only the coronary cusp, Class II which extends up the ascending aorta but<4 cm, and Class III which extends ≥ 4 cm. Once identified, this should be addressed promptly with a stent or potentially even a covered stent to seal the aortic root [30]. If sealing is not accomplished, CTA should promptly be done to assess/ document the degree of dissection up the root. TEE can also be considered if contrast is a concern. While there is no guideline, a strategy proposed by Dunning et al, is that dissections that involve only the cusp, or include<4 cm of the root despite stenting, (Class I/II) can be managed with serial imaging and blood pressure control. If dissection extends ≥ 4 cm up the root (Class III), and is not sealed by stenting, surgical aortic root repair should be pursued.

Coronary perforation: Coronary perforation complicates ~0.5% of PCI, i.e., around 1/200 cases, and increases 30-day mortality ~5-fold [31]. Factors that increase the risk of perforation include oversized balloon or stent, atherectomy, cutting balloons, CTO intervention, prior CABG, vessel calcification, female sex, and advanced age [32,33]. Wire perforation appears to account for the majority of events [34]. Perforations can occur at the site of intervention or distally, if related to the wire tip. Perforations are typically classified by the Ellis system as follows: Type I) Extra luminal crater only, similar in appearance to a Type B dissection; Type II) pericardial or myocardial blush without extravasation; Type III) extravasation though a frank jet>1 mm, which is a procedural emergency [35]. Tamponade is rare with type I/ II perforations at 0.3% and ~3% respectively. Tamponade is very common with type III perforation at ~45% with ~21% mortality [36]. Patients may complain of abrupt onset of sharp chest pains, related to pericardial irritation, along with hemodynamic decompensation. Make sure to follow the angiogram to the distal vessel to assess all levels. As little as 100 mL of blood can cause tamponade if accumulating rapidly as shown in the Table 2.

Prevention of perforation: As in prevention of dissection, coil tip workhorse wires should be used if possible. Hydrophilic wires with tapered/penetrating tips should be avoided, or exchanged as soon as possible after crossing the lesion. The tip of the wire should be allowed to prolapse into a J-shape after the lesion is crossed, and should be kept in view at all times during a case. Again, balloon sizing and inflation pressure should be conservative. After any inflation, a quick "puff" of contrast can be done prior to removal of balloon–while this will not prevent perforation per se, it allows for rapid detection. Immediate reinflation and may prevent tamponade proper given earlier identification. If atherectomy is being used, a burr no larger than 1.5 mm should be utilized to essentially facilitate follow-up ballooning and stent passage.

Treatment of perforation: Treatment of perforation depends on

Table 2: Ellis classification of coronary perforation, Type I, Type II, and Type III [34].

Coronary perforation types				
Ellis classification	**Definition**	**Risk of tamponade**	**Balloon only success**	
Ellis type I	Contained extraluminal crater	<1%	High	
Ellis type II	Pericardial blush without gross extravasation	3%	High	
Ellis type III	Frank extravasation with ≥ 1 mm jet	45% with ~20% morality	Low ~50%	

the site and the type involved. A balloon sized 1:1 to the caliber of the vessel should be inflated in practically all cases to "decompress" the vessel and allow for self-sealing. This also allows for the operator to "take a breath" and further planning. Staff should be instructed to summon echocardiography to the bedside, and the pericardiocentesis kit should be identified. IV fluids should be administered rapidly and neosynephrine and/or atropine given if the pressure is dropping. GPI medications should be stopped immediately. If the perforation is proximal or involves a sidebranch that can be excluded, definitive treatment may include a covered stent such as Graftmaster (Abbott Vascular, Abbott Park, IL) or Papyrus (Biotronik, Berlin, Germany) and this device should be identified and brought into the room. If tamponade has occurred, pericardiocentesis should be pursued with the balloon remaining inflated, with use of echo guidance if logistically possible. It is described to immediately reinfuse the pericardial blood through a femoral vein [37], although theoretical clotting complications apply. The pericardial drain should be left in at least overnight. If there is no tamponade and case has been managed expectantly, serial echo studies should be performed.

- The sealing balloon should be inflated over sessions of 10 minutes, with reassessment even up to an hour [38]. A perfusion balloons such as the Ringer balloons (Teleflex; Wayne, PA) can be used uninterrupted. If improvement is not occurring, consideration can be given at that point to reversal of anticoagulation with protamine up to 50 mg (for heparin 1 mg for 100 units or LMWH 1 mg for 1 mg) or recombinant factor VII at 90 mcg/kg (for bivalirudin) [39]. If given, one needs to be wary of vessel and/or guide catheter thrombosis.
- Blood can be withdrawn from the guide or sheath and set aside to clot during balloon inflations.
- If decompression with the balloon fails, proximal or side branch perforations can be addressed with a covered stent. Distal perforations can be addressed by injecting small fat embolus (such as from the groin fat pad), clotted blood, thrombin, or coils through a distally delivered microcatheter.
- Coil designs include detachable or pushable delivery. Pushable coils are advanced through the delivery microcathether with either a proprietary wire or a coronary wire and then tacked down. Detachable coils are equipped with a delivery wire that is attached to the coil until the position is satisfactory, and then released (detached).
- In one series by Meguro, et al., [34], prolonged inflation was effective to seal perforation in 86% of cases, including 73% of perforations caused by stenting, and 55% of type III perforations. The average balloon inflation duration for type I/II was 20 minutes, while for type III was 48 minutes [34]. Covered stenting is associated with 85% success in resolution of grade III perforation [40].
- The PK Papyrus has a single layer of polyurethane covering a cobalt chromium stent, while the GraftMaster has a layer of PTFE within an inner and outer layer of stainless steel stent. Papyrus stents can be delivered through 5F guides, while Graftmaster requires 6F, and even 7F for the devices ≥ 4.5 mm.
- In a registry of patients treated for perforation, Papyrus compared to GraftMaster was associated with lower MACE at 30 days, 3.6 vs 17.6% p=0.02, driven by lower TLR, although equipoise at 1 year [41].

No-reflow phenomenon

No-Reflow Phenomenon (NRF) is defined as either partial or complete loss of blood flow velocity in a coronary artery despite patency of the artery, i.e., not due to thrombus, recoil, or dissection. NRF occurs due to embolized microparticles overwhelming the coronary microcirculation, commonly embolized thrombus or cholesterol crystals, as well as concurrent tissue ischemia and microvascular dysfunction and edema especially during ACS management. No-reflow most commonly occurs during treatment of STEMI, saphenous vein grafts, and after use of atherectomy [39]. IVUS studies also demonstrate higher risk of NRF with large necrotic core [42]. Other risk factors include elderly, diabetes, high CHADS2-Vasc factors, delay in presentation, high thrombus burden, long lesions, and LAD location [43]. The angiographic finding of NRF often occurs simultaneously with a dramatic clinical presentation including severe chest pain, hemodynamic collapse, and bradycardia; bearing rapid treatment. No-reflow is associated with reduced 5-year survival, higher risk of systolic heart failure, and malignant arrhythmia in STEMI [44,45].

Prevention of no-reflow:

- Patients should be ideally being at steady-state on anti-platelet medications before the procedure. Maintenance of therapeutic anticoagulation with appropriate ACT goals cannot be overlooked. Minimizing instrumentation in STEMI is favorable, and aggressive predilatation should be avoided. If thrombus burden is high in ACS, aspiration thrombectomy should be strongly considered. Stent deployment should be done as soon as possible with rapid inflation/ deflation. After thrombus aspiration or low-pressure ballooning, and before stent deployment, consider pre-treating with microvascular dilators in patients with risk factors especially prolonged ischemic time, LAD STEMI, or high thrombus.
- GPI medications can be given in patients with high thrombus burden associated with sluggish flow.
- In all cases, if flow velocity begins to slow, the remainder of

the procedure should be as simple and efficient as possible. High pressure post-dilatation should also be avoided.

- In SVG intervention, embolic protection is universal. Prophylactic treatment with vasodilators at the appropriate doses can be considered [46,47].
- Rotational atherectomy should be done using conservatively sized burrs (<0.7× the reference vessel), using a staccato pecking motion. Runs should be limited to 20 seconds and decelerations>5000 rpm should be avoided.

Treatment of no-reflow:

- The most common medications that are used include adenosine, verapamil, and nitroprosside [48-50]. When in doubt, consider administrations of 100 mcg as this is an acceptable dose for all agents. Each of these medications can result in further drops in blood pressure, and may need to be counteracted with pressors or alternated with intracoronary epinephrine. Medication should be injected as distally as possible in the infarct vessel, such as through a transit catheter or and over the wire balloon.
- Adenosine, nicardipine, nitroprusside, and verapamil have shown similar efficacy for TIMI flow improvement, as well as prevention of MACE compared with no treatment of NRF in a retrospective study [51], and these medications are recommended in the PCI guideline. In one trial, nitroprusside improved TIMI flow by 1 grade in 82% of pts.
- If the patient is severely hypotensive, 1 mL of epinephrine 1:10,000 solutions can be diluted in 10 mL of saline, with 1 mL boluses of the dilution given intracoronary resulting in paradoxical stimulation of B2 receptors. The COAR study of ~100 pts with NRF in ACS actually showed superiority of epinephrine (given at doses 100-600 mcg) vs adenosine (600-1000 mcg) for final TIMI-3 flow (90 vs 78%) and 30 days' ejection fraction [52].
- A mechanical support device and be employed if flow remains<TIMI-3 post PCI. A large registry analysis showed that use of IABP was associated with mortality reduction in STEMI patients with cardiogenic shock and final TIMI flow<2, HR for death 0.72 p=0.002, but not in patients with TIMI flow [53].

Air embolism: Air embolism is estimated to occur ~1/3000 cases, again more commonly during interventional than diagnostic cases [54]. This can occur due to inadequate guide aspiration, faulty connection of manifold tubing, and frequently *via* introduction of air into the system due to suction into the Tuohy during aspiration (such as for checking ACT). If a PCI balloon ruptures and has not been thoroughly de-aired, this can also occur; hence any ultra-high pressure ballooning (such as>18 atm) requires meticulous balloon prep. Small air bubble injection within the coronary is usually a clinically silent event visualized with contrast injection. If the embolism is more substantial, blood flow becomes impaired, and the patient will demonstrate EKG changes including ST elevation, chest pain, arrhythmia, and rarely cardiac arrest.

To prevent this, careful attention is required at multiple steps. All tubing connections on the manifold and auto-injector should be double-checked for tightness. Catheters should be actively aspirated 2-4 mL of blood with every exchange, while in a non-cannulated position. Saline can also be trickled over the exchange wire during removal. If there is another arterial access point for checking ACT, this should be used rather than the guide. If a significant air embolism has occurred, the patient should be immediately administered high flow mask oxygen which improves tissue oxygen delivery and also increases the gradient for the nitrogen-rich room-air bubble to diffuse out of the vessel [55]. Standard ACLS measures obviously apply, as well as placement of a temporary pacemaker if necessary. A wire can be passed into the vessel to deliver a simple aspiration catheter–this can be used to both suction the air and deliver distal injection of blood or contrast medium [56]. Adenosine and/or placement of a balloon pump may be helpful to augment microcirculatory flow. Supportive cares and repetition of aspiration should occur until the patient is stabilized.

Stent dislodgement

Stent dislodgement was more common in the earlier days of PCI, estimated at ~5% prior to 2000 and ~0.3% after 2005 [57], due to improvement in equipment manufacturing processes. Common causes for the stent to dislodge from the balloon include difficult advancement of a stent through a narrowed or calcified vessel, tortuosity/ angulation, poor guide support, delivery through a previous stent, rigid "lead-pipe" coronary vessels, and withdrawal of a "no-cross" device with subsequent dislodgement on the guide or guide-extender edge [57]. Vigilance for stent dislodgement is necessary in all guide-extension cases, and upon removal of an undelivered stent, the device needs to be examined diligently for presence of the stent. Preventative strategies involve recognition of difficult anatomy, liberal predilation, and use of adequate guide caliber and shape and/or buddy wire support prior to placement of a guide-extension. As always, distal-most lesions should be addressed first to avoid crossing a deployed stent.

If stent dislodgement has occurred options include deployment *in situ*, snaring and retrieval, and crushing with another stent [58]. If the coronary wire remains in place, and the device is in an area that is appropriate for stent deployment, re-advancement of graduated sized balloons to deploy the device *in situ* may be the quickest solution. An inflated balloon proximal to the stent can be used to push more distally if necessary. If removal of the stent is

preferable, a smaller sized balloon can be passed distal to the stent and inflated, then withdrawn to the guide to allow for removal of all equipment "as a unit". This may also be assisted by passing a second wire adjacent to or partially through the stent and twisting it multiple times to entangle the stent. Loop snares can also be advanced over the coronary wire, or coronary wire/ balloon, and used to ensnare and remove the equipment "as a unit". Embolic protection baskets can be deployed distal to the stent and used to pull it back into the guide. If these strategies are unsuccessful, another stent can be passed over a second wire and used to crush the dislodged device against the vessel wall, with good outcomes [59]. If a stent has embolized into the peripheral circulation and cannot be ensnared (or even identified), this is typically clinically silent with no identifiable long-term consequence [60].

Vascular complications

Procedural vascular, complications in PCI in descending order of frequency include localized hematoma (7.9%), pseudoaneurysm (1.5%), AV fistulae (0.1%), Retroperitoneal Hematoma (RPH) (<0.5%) [61,62]; with the expectation that that radial access can reduce these occurrences by about 10-fold [8]. Localized hematoma can be recognized by exam and is managed almost universally by compression. Pseudoaneurysm and AV fistulae can be typically diagnosed with ultrasound. Recall that RPH compared with the others is typically quite dramatic clinically, with the patient demonstrating waxing/ waning hypotension, tachycardia, and lower abdominal pain. Risk factors for vascular bleeding complications include low body weight, large bore sheath, female sex, age, kidney disease, ACS, and use of heparin/ GPI as opposed to bivalirudin [63].

Prevention of vascular complications: Meticulous attention to detail when obtaining femoral access is important, with the goal that the arteriotomy should be in the vertical center of the femoral head, which improves the ability to apply compression (as the femoral head provides back-pressure), and also avoids the femoral bifurcation which occurs below this point 98% of the time [64]. Access of the femoral artery outside the optimal zone more than quadruples the risk of vascular complication [65]. Another well-described anatomic landmark is the inferior epigastric artery, the inferior "swooping" aspect of which demarks the border of the inguinal ligament [66]. Typically, a low femoral stick (below the femoral bifurcation) increases risk for hematoma/ pseudoaneurysm, and a high stick (above the inferior epigastric artery) increases risk for RP hemorrhage. One small cohort study revealed that 100% of RP hemorrhage cases occurred with arteriotomy above the inferior epigastric [67].

Commonly, micropuncture kits are utilized based on intuitive safety, although data to show such a benefit is lacking. One analysis comparing micropuncture with standard 18G femoral needle access in PCI showed no reduction in overall complications and higher RP hemorrhage 0.7 vs 0.18%, potentially related to advancement of the small caliber micro wire into side branches [68]. This underscores the importance of advancing the micro wire only under fluoroscopic guidance. Micro puncture affords the logistical advantage that, if a low or high stick has been identified through injection of the micropuncture introducer, it is possible to remove it and apply pressure before another attempt.

"Fluoroscopic technique" entails marking the inferior aspect of the femoral head with a hemostat, entering the skin with the needle at that site, and advancing at a 45-degree angle between the first 2 fingers outlining the maximal femoral pulse. Single anterior anterior wall stick (the modified Seldinger technique) is used. Use of ultrasound access in the FAUST study showed superiority to fluoroscopic guidance for optimal femoral stick and reduced occurrence of hematoma [69]. If a suboptimal stick has been identified at the conclusion of the case, typically the patient should be managed with manual compression upon sheath removal. Use of sandbags post procedurally does not appear to have any effect on development of complication [70].

Vascular closure devices: It is a key concept that Vascular Closure Devices (VCDs) allow for expedited ambulation and improve patient comfort, but are not indicated to reduce vascular complication (Class III no benefit in PCI guideline) [71]. These devices can typically be applied after demonstration on a femoral angiogram of adequate femoral artery caliber>4-6 mm, appropriate site of arteriotomy (i.e., neither high nor low as described above), lack of active groin infection, and lack of severe vascular disease. Multiple types of VCD exist, and this toolbox is always expanding, strategies include suture based (Perclose, Abbott Vascular; Redwood City, CA); epivascular collagen plug with indwelling footplate (Angioseal, Terumo; Somerset, NJ) (Manta, Teleflex; Wayne, PA), balloon-based (Mynx, Cordis; Miami Lakes, FL), collapsible metal disk-based (Vascade, Haemonetics; Boston, MA); epivascular metal closure (Starclose, Abbott) (Celt, Vasorum; Dublin, Ireland) to name but a few. Knowledge of the deployment techniques for several of these devices, and specifics of any leftover intravascular component and device-specific complications, is recommended as part of a broad interventional practice. Based on a very large registry of ~2 million PCI patients including multiple devices, VCDs in general were associated with a significant although small trend towards reduction of vascular complication. The overall access complication was 1.5%, with the absolute risk reduction with VCD 0.4% [72], which provides evidence that these devices can be used safely. It is not recommended to employ VCD in a patient who has received thrombolytics, or if there is ongoing GPI medication infusing–it is better to conclude the infusion and wait the necessary 4 half-lives before applying manual pressure.

Pseudoaneurysm: Pseudoaneurysm (PA) refers to a locally contained hemorrhage occurring epivascularly, related to ongoing pulsatile flow into a hematoma. As the name implies, this is not a true aneurysm, as the walls are comprised of local tissue and components of the clotting cascade including crosslinked platelets/ fibrin. As above, this is the second most common access complication, after hematoma, and is associated with low stick, peripheral arterial disease and larger sheath size [73]. The patient will present with groin swelling and pain. By exam, PA can demonstrate auscalable bruit and painful pulsatile mass. The diagnosis is made with ultrasound with color flow, differentiating from hematoma, showing pulsatile flow and possible "yin-yang" pattern. Pseudoaneurysm>3 cm are at higher risk for rupture and require more proactive management [74]. Management of PA>3.0 cm can include ultrasound-guided compression, localized thrombin injection, and surgical repair, all of which are highly successful [75]. Small PA<2-3 cm may spontaneously resolve without any particular therapy [76], with one series showing ~90% self-resolution of PA<3 cm [77]. Surgical closure of smaller PA is therefore usually reserved for failure of less invasive measures, expanding PA, or if anatomy precludes thrombin injection (such as neck length<2 mm).

Arteriovenous fistula: Arteriovenous Fistula (AVF) is an abnormal communication between adjacent artery and vein, with flow from the artery to vein as dictated by pressure differential. This occurs most commonly after simultaneous arterial and venous cannulation, whether intentional or unintentional. Hence, this tends to be more common in a "low stick", at which point the SFA vessel runs anterior to the vein, allowing for a through and through puncture [78]. The patient may clinically present with pulsatile mass, distal ischemia due to steal, and in rare cases clinical heart failure– but the vast majority of AVF after cardiac procedure are asymptomatic [79,80]. Physical exam may reveal palpable thrill or bruit. Once again, diagnosis is confirmed with ultrasound with color flow, showing communication between the vessels, and "arterialization" and enlargement of a venous vessel [81]. Many AVF close spontaneously, and when confronted with such a patient in practice or on boards exam, the answer for an asymptomatic patient will be watchful waiting. Enlarging fistula, and those causing symptoms of arterial steal or high output heart failure require treatment [82]. Options for treatment include US-guided compression, stent grafting (for simple/ singular fistulae), surgical ligature, and embolization depending upon the anatomy. Consultation with vascular surgeon is advisable.

Retroperitoneal hemorrhage: Retroperitoneal Hemorrhage (RPH) is a rare complication of interventional procedures at<0.5%. Ellis, et al., [83], but is the most dreaded given high case fatality rate at ~10%. RPH represents a true medical emergency that requires high index of suspicion and rapid response. Risk for RPH increases with high femoral stick, back-wall stick, female sex, use of GPI medications, and low body weight [67,83]. RPH is virtually eliminated with radial artery access. An appropriate femoral stick does not completely eliminate this complication, as blood can track though the neurovascular sheath of the femoral artery. The patient presents with hypotension that is often "waxing and waning", which may transiently respond to fluid bolus or pressor medications. The patient may complain of back or flank pain, but not always. Flank discoloration/ bruising may manifest, so-called Grey Turner sign, although uncommonly early in course. Diaphoresis may be present, though in contradistinction to vagal reaction (which is the primary differential diagnosis), the heart rate can be rapid due to intravascular hypovolemia. While a hematoma may be present, do not be falsely reassured, as the lack of an external hematoma does not exclude RPH.

Non-contrast CT imaging can provide fairly rapid diagnosis, however if the suspicion is high, diagnosis and treatment can be immediately provided *via* contralateral femoral access and direct angiography [84]. This strategy is increasingly used. While transporting the patient back to Cath lab, 0-negative trauma blood can be called for. Anticoagulation should be stopped and/ or reversed if possible. Once the bleeding site has been identified, the first option is prolonged balloon inflation with balloon sized to the vessel at the site of bleeding, for increments of 10 minutes. This often stabilizes the patient quickly. With the balloon in place, a covered stent of appropriate size can be identified (sized to vessel or ~1 mm larger), and vascular surgeon can be summoned. If extravasation does not resolve with ballooning or covered stenting, the patient can be transported to the OR with the balloon inflated, for direct vascular repair.

Periprocedural stroke: In the course of an interventional procedure, symptomatic Cerebrovascular Accident (CVA) occurs in approximately 0.5% of cases, a phenomenon on that is likely to be quite memorable for the operator [5,85]. However, subclinical embolic stroke, as diagnosed by post-procedural MRI, occurs as high as 15% [86]. The patient presentation will vary based on anatomic location but can include sudden speech disturbance (MCA), upper extremity or facial weakness or numbness (MCA), lower extremity weakness (ACA), visual abnormalities and/or ocular weakness (posterior circulation), and altered mentation (variable distribution). Visual and motor disturbances appear to be the most common [87]. It is also important to remember that part and parcel with systemic anticoagulation is the risk of hemorrhagic CVA, which occurs nearly as often as embolic CVA, and is treated very differently [85]. Hemorrhagic CVA needs to be considered/ excluded especially prior to administration of lytics; typically, this is accomplished with STAT head CT. A common diagnostic dilemma for the interventionalist is being called to the bedside to assess a somnolent patient-which in and of itself does not necessarily

speak to CVA, and may be related to other causes of delirium.

Case factors associated with higher risk for CVA include advanced patient age, severe vascular disease, totality of cardiac risk factor profile, prior CABG, case duration, volume of contrast, transradial approach (likely related to passage of catheters across carotid and vertebral arteries), crossing of the aortic valve, and the use of thrombus aspiration [88-90]. Valve crossing is one of the few modifiable factors here, and it is good practice to review noninvasive data pertaining to LV function and filling pressures before each case, and avoid crossing the valve if the information is adequate. Sheaths and catheters should be aspirated liberally throughout a case, and all catheters advanced with an atraumatic 0.035" wire in place.

Mortality with periprocedural stroke is high at 25-44% [88]. Deep sedation may mask outward signs and diagnosis is often delayed. Embolized plaques may also result in delayed thrombosis within the embolized intracranial vessel, hence symptoms may not occur for 24-48 hours. Treatment is somewhat controversial without randomized data or consensus guidelines. Solid emboli may be related to intravascular plaque debris rather than thrombus, hence the conceptual role of thrombolytics is questionable. If there is evidence for CVA, neurology should be contacted immediately. If the operator has sufficient experience, intracranial angiography can be done which can help to direct thrombus management. Typically, STAT head CT is ordered as well, to exclude hemorrhagic stroke. Retrospective data supports use of thrombolytics both intra-arterial and IV for embolic stroke [91]. As always, absolute contraindications need be considered. If a neurointerventionist is available, thrombectomy may also be possible.

Transient cortical blindness: One situation that can mimic stroke is transient cortical blindness, i.e., loss of visual perception but maintenance of ocular reflexes, occurring as a result of contrast exposure [92]. This is thought to occur as a result of preferential contrast-related disruption of visual pathways at the occipital cortices, possibly related to supine accumulation of contrast or lessened sympathetic ennervation posteriorly [93]. Cranial imaging may confirm occipital contrast enhancement in these cases. Risk is increased with LIMA injection given proximity to the vertebral artery. Typically, transient cortical blindness is self-limited and requires no specific treatment. If the patient is also demonstrating confusion or is denying the visual deficit, it is deemed as Anton's Syndrome.

Contrast induced nephropathy

The definition of CIN is most commonly reported as Acute Renal Failure (ARF) with increase in Cr>0.5 mg/dL from baseline, or 25% increase from baseline, within 48 hours of receiving iodinated contrast and is present for 2-5 days [94]. The risk for ARF related to an interventional case is ~7% [7], although is dependent on multiple patient factors. CIN is the 3rd most common cause of hospital-acquired ARF behind surgery and hypotension [95]. Multivariable risk factors for ARF include contrast volume, age>75, hypotension, CHF, GFR<60, diabetes, use of balloon pump; these are encompassed the Mehran score which can be used to predict risk for ARF for a given case [96]. GFR<60 is the singular most important predictor.

Typically, the course of CIN is benign, reflected by an asymptomatic "creatine bump" which plateaus around 48-72 h, and may also include reduced urine output [97]. Fewer than ⅓ of patients develop long term change in GFR [98]. A simple calculation for contrast limits comes from a large ~58,000 population study performed by Gurm, et al., [99], the CrCl is multiplied by 2.5, with the goal to keep the contrast volume in mL ≤ that number. For example, if the CrCl is 50, the contrast volume should be kept<125 mL. The risk for CIN became highly significant at a volume of 3 (ClCr) in that study, and below 3 (CrCl) the CIN risk hovers at around only 2% [99]. Rates of dialysis appear to be overall exceptionally low at 0.2%, however if the contrast volume superceded 3.0 (ClCr), the risk tripled to ~0.6%. The risk of in-hospital death goes up to ~20% in the setting of CIN/ARF, as compared with 1.4% no CIN, and progression to dialysis portends 36% in-house mortality and<20% 2-year survival [100].

The mechanism of CIN appears to be injury to the renal medulla due to reduced renal blood flow related to osmotic effects, as well as a smaller component of renal tubular toxicity associated with free radicals. Intrinsic forces likely include vasoconstriction, decreased glomerular Prostaglandin (PG) and/or Nitric Oxide (NO) at afferent arterioles, increased Angiotensin (AT) at efferent arterioles, contrast-induced diuresis, and tubular obstruction [101], all of which contribute to reduction in GFR. Upon exposure of the Juxtoglomerular Apparatus (JGA) to osmotically active contrast, there is transient increase in renal blood flow followed by a more prolonged reduction of flow due to disruption of the regulatory balance of PG/ NO/ AT etc., leading to medullary ischemia and hypoxia. High sodium concentration at the macula densa leads to activation of RAAT system at the efferent arterioles. Data and anecdotal clinical experience reflect that CIN commonly occurs in a background of clinical factors such as emergency procedure, hypotension, baseline CHF, diabetes, and exposure to other nephrotoxins (such as ACE inhibitors or diuretics).

The most effective means for prevention is prehydration of at-risk patients (such as GFR<60 or high Mehran score), with basic Normal Saline (NS) for 3-12 h preprocedurally and continuing 6-24 h post procedurally for at a rate of 1.0-1.5 mL/Kg/hour (for example, a 100 kg patient would receive 100 mL/h for a total of ~1-3 liters) [102]. Standard 0.9% saline is likely more effective

than less concentrated formulations such as 0.45% saline [103], and essentially equivalent to bicarbonate solutions [104]. The POSEIDON study showed that there may be a reduction in CIN by tailoring post-op hydration to the baseline LVEDP using a "sliding scale", with higher LVEDP receiving lower post-op fluid rates and vice versa [105]. There is likely an advantage to use of statin use peri-procedurally [106]. There may be a small positive effect for use of N-Acetylcysteine (NAC), a precursor to glutathione that acts as antioxidant/ free radical scavenger, and also has vaso-dilatory properties. Pannu, et al., [107], meta-analysed several small studies in 2004 and demonstrated a benefit with RR for CIN at 0.64, although of only borderline statistical significance (p=0.049) [108]. Another meta-analysis showed similar findings with a RR 0.43 (p=0.04). Presently, NAC is not recommended for reduction of ARF by the cardiology or nephrology KDIGO guidelines, although is still frequently done given low cost and potential for benefit.

Choice of contrast agent can also affect development of CIN. In meta-analysis, low-Osmolal Contrast (LOC) agents are less likely to result in CIN/ARF compared with High-Osmolal Contrast (HOC) in patients with CKD (OR 0.5) [109]. Third generation Isosmolal Contrast media (IOC) are based on dimeric (two connected) tri-iodinated benzene rings that are presented in solutions that are isosmolal to plasma at ~300 and nonionic, the primary example being iodixanol (Visipaque). Meta-analysis also suggested a reduction of CIN with IOC compared to LOC, risk 1.4 vs 3.5% (p<0.001); though iopamidol (possibly safest of the LOC) was under-represented in this trial [100]. When IOC iodixinol (Visiopaque) was directly compared to LOC iopamidol (Isovue) in the ~400 patient CARE study, findings showed that in pre-hydrated patients there was non-significant 12.4% vs 9.8% CIN (p=NS) with trend to less CIN with LOC iopamidol [108].

It is important to have an awareness that other medication strategies have not been shown to be effective compared to hydration, including mannitol, furosemide, calcium channel blockers, fenoldopam [112-114]. Prophylactic dialysis after contrast exposure was also not shown to be beneficial [115,116]. A systematic review of multiple strategies found a statistical benefit only to hydration, NAC, and statin [104]. Practically speaking, ARF can be mitigated by appropriate hydration, limiting per-procedure contrast administration (for example avoiding LV angiogram), staging interventions with an appropriate delay, etc.

Cholesterol embolization syndrome

Iatrogenic disruption of vascular plaque with embolic obstruction of small arteries and arterioles by cholesterol crystals is known as Cholesterol Embolization Syndrome (CES). This finding can affect lower extremities as well renal vasculature causing Atheroembolic Renal Disease (ARD) [117]. This is a relatively rare phenomenon that is clinically apparent<1% of catheterization procedures, although likely more commonly on a silent basis [118]. Cutaneous clinical signs can include livedo reticularis, blue toe syndrome/trash foot, or frank digital ulceration or gangrene with intact pulse exam. Findings may occur in a delayed fashion even weeks after the procedure. Lab workup may demonstrate leukocytosis, thrombocytopenia, elevated ESR/CRP, and elevated eosinophils. Eosinophiluria can be present in ARD [119]. The standard to diagnose CES/ARD would be skin or renal biopsy, respectively. However, diagnosis is often on a clinical basis, for instance subacute renal failure occurring in a patient status-post PCI procedure with associated skin findings. ARD typically demonstrates a more morbid course than CIN., with dialysis necessary in 28-61% of cases [117]. In-hospital mortality is as high as 16% in those patients with definite CES [118], because multiorgan embolization can often lead to multiorgan failure. In patients requiring dialysis, mortality was quite high at 1 year at>80% [120]. Treatment is largely supportive, as trials involving steroids have been inconclusive.

Anaphylactoid reaction

An immediate systemic reaction to contrast can occur that mimics anaphylaxis, including bronchospasm, hypotension, facial and/ or laryngeal edema, pulmonary edema, arrhythmia, and acute respiratory failure, occurring within 1 hour of exposure. This has been classified as "anaphylactoid" rather than anaphylactic as it is primarily mast-cell and complement mediated release of histamines, rather than an IgE mediated, although the latter can contribute [121]. Reactions are idiosyncratic, i.e., are not dose-responsive. Severe anaphylactoid reactions occur ~0.2% of procedures, i.e., ~2/1000 based on recent estimates, which has lessened over time with the hyposmolar and isosmolar contrast agents [122]. Risk factors for anaphylactoid reactions include history of asthma, atopy, and most importantly, previous reactions [123]. Less intense "delayed" reactions occur far more commonly at 5%-8% of the time, 70% of which involve cutaneous manifestations such as flushing and rash at 1-7 days, also nausea, fever, malaise, and GI distress [124]. It may be difficult to parse out such a reaction from a medication reaction, as the patient has likely been started on a host of new drugs. Pretreatment with a steroid regimen nearly eliminates anaphylactoid reaction. Commonly used would be prednisone 50 mg at 13 h, 7 h, and 1 h before the procedure often concurrently with benadryl and H2 blocker. Such a regimen along with low osmolar agents reduced the occurrence of anaphylactoid reaction to 0.5%, compared to 9.1% in untreated individuals [125]. It is not necessary (Class III no benefit) to pretreat patients with shellfish allergy.

If a severe intraprocedural anaphylactoid reaction occurs, no further contrast should be given. The patient should be

administered 0.3 mg-0.5 mg of epinephrine intramuscular (which is 0.3 mL-0.5 mL of "1:1000" solution which is 1 mg/mL) or 0.1-0.5 mg IV (which is 1-5 mL of "1:10,000" solution which is 1 mg/10 mL) given slowly over 5 minutes, along with IV steroid and diphenhydramine, to be repeated every 15 to 20 minutes if there is no clinical improvement. Epinephrine can be converted to an infusion of 5-20 mcgs/min if symptoms are refractory. If the patient's airway is compromised, emergent intubation will be required [126].

Operator response to complications

It has been said that every proceduralist has his own graveyard of complications and deaths [127]. Historically, medical culture has not been supportive in the face of complications, leading to a reluctance by the operator to disclose to patients or the institution. Adding to this can be the fear of litigation or stigmatization by colleagues. Counterintuitively, expressing remorse to the patient and family with respect to the occurrence, and acknowledging what went wrong, may prevent a lawsuit, or at the very least increase the likelihood to accept a settlement out of court [128,129]. Full disclosure and expression of remorse can also result in maintenance of the physician/ patient relationship. When complications occur, the operator oftentimes experiences a phase of anxiety ("the kick"), followed by a phase of doubt and regret. ("the fall"). During these times, one often examines the case in great detail, and also reviews literature on the matter at hand and discusses with colleagues, which can lead to professional growth. This may take days to weeks, during which it is often harrowing to perform further procedures. Finally, there is a phase of recovery, in which the operator returns to a more normal, undistracted approach, finding comfort in learning something that may prevent a similar future event [130-135].

Conclusion

In conclusion, in the practice of interventional cardiology, an operator will encounter a wide array of complications, with variable frequency. As these issues can arise in procedures that are inherently high risk at the outset, but also when they are least expected, a functional knowledge of prevention, recognition, and treatment is paramount. A baseline level of gentleness in the cardiac Cath lab is important. It should not be overlooked that the operator must also allow him or herself a level of grace. "It is said that "wisdom comes from knowledge, knowledge comes from experience, and experience comes from lack of knowledge". Over time, these emotion-laden occurrences can inform the operators intuition, and lead to avoidance of previous errors, functioning as a key component of lifelong self-improvement. Strategies to avoid complications ultimately become incorporated into routine practice.

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