Percutaneous coronary intervention

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Abstract

Percutaneous coronary intervention (PCI) is the most common technique to improve myocardial perfusion when treating coronary artery disease. It is very efficacious in improving symptoms for individuals with stable angina, and improves prognosis in acute coronary syndromes, particularly in the emergency treatment of patients presenting with ST elevation myocardial infarction. It is performed via a small intra-arterial sheath. A balloon is used to dilate the coronary stenosis, and a stent is implanted to scaffold the vessel. Re-narrowing at the treated site can occur but has been greatly reduced by drug-eluting stents. Most acute complications of PCI are mediated by platelet activation, so drugs blocking platelet aggregation are pivotal to the procedure's safety. Early complications include haemorrhage from the arterial access site (reduced by a radial approach). Abrupt vessel closure, stroke, vessel perforation and tamponade are rare. The requirement for emergency cardiac surgery is <0.1%, and in-hospital mortality is mainly determined by the indication for PCI – about 0.2% in patients with stable angina, 5% after ST elevation myocardial infarction and 30–50% in the context of cardiogenic shock. Technical advances mean that patients with complex coronary artery disease and co-morbid conditions can now be treated by PCI.

Keywords Angioplasty; clopidogrel; drug-eluting balloon; drug-eluting stent; intravascular lithotripsy; intravascular ultrasound; laser; MRCP; optical coherence tomography; percutaneous coronary intervention; prasugrel; pressure wire; radial; rotablation; stent; thrombectomy; ticagrelor

Key points

- Percutaneous coronary intervention (PCI) is the most frequent method for mechanically improving myocardial perfusion
- Coronary revascularization can be performed by PCI or coronary artery bypass grafting (CABG), depending on the clinical and technical features. Although more complex disease is better treated by CABG, many of these patients have co-morbidity that precludes open surgery, leaving PCI the preferred option
- Drug-eluting stents have become more refined. They have improved short- and long-term outcomes, with a lower risk of late stent thrombosis than bare metal stents
- Antiplatelet agents are key to preventing stent thrombosis, but the benefits must be weighed against the risk of bleeding. A more tailored patient-specific approach is being developed
- Radial artery access reduces bleeding complications and is associated with lower mortality

Introduction

Atherosclerosis in coronary arteries usually manifests clinically by causing stenoses or occlusions that reduce myocardial blood flow. The term 'percutaneous coronary intervention' (PCI) applies to various procedures that reopen obstructed coronary arteries to improve myocardial perfusion without resorting to coronary artery bypass surgery (CABG). PCI usually starts by inflating a balloon within the coronary artery stenosis (percutaneous transluminal coronary angioplasty), followed by implantation of one or more stents. Variations of this basic procedure, described below, are used in some patient subsets. In 2020, >100,000 PCI procedures were performed in the UK, with >5 times more people treated by PCI than by CABG.

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Role of PCI in clinical syndromes

Revascularization in the treatment of coronary artery disease aims to improve symptoms and/or prognosis. Appropriateness is determined by the patient's clinical presentation, symptoms and co-morbidities.

Stable angina

Mechanical revascularization (CABG, PCI) should be considered in individuals who have angina despite medical therapy or who tolerate medication poorly because of adverse effects. PCI is safe and effective in reducing angina in such patients. PCI is associated with better outcomes than medical therapy alone when use is guided by invasive assessment of the functional significance of coronary stenoses (see below).

The choice between PCI and CABG is determined by a combination of clinical and technical considerations. Patients with more extensive atheroma generally have better long-term outcomes with CABG than PCI. A method to score coronary disease complexity in patients with three-vessel disease (SYNTAX score) has been derived from randomized trials, and is often used to inform the decision on optimal revascularization.[1] Individuals in the lowest tertile of the SYNTAX score (score ≤22) are well suited to PCI, with those in the middle and upper tertiles best treated by CABG. PCI is, however, still used in those with higher complexity scores when co-morbidity and frailty mean that open surgery carries a particularly high risk.

Some studies suggest that people with diabetes mellitus are better treated by CABG than PCI, particularly if surgery can include use of the left internal mammary artery to bypass disease

in the left anterior descending coronary artery; this particular bypass graft appears to provide the best long-term benefit. In patients who do not have diabetes mellitus and have less widespread disease, PCI offers equivalent long-term mortality with a lower risk of stroke than CABG, albeit with an increased need for repeat PCI.

Equivalent outcomes for CABG and PCI have been shown for the treatment of left main stem disease, in the absence of disease at the bifurcation or other complex coronary disease. The decision on optimal revascularization strategy for an individual patient can be complex, and should take into account symptoms, coronary anatomy, co-morbidity and patient choice. Guidelines recommend that these decisions be taken by a multidisciplinary 'heart team'.[2]

Acute coronary syndromes

PCI improves survival in patients presenting with acute ST elevation myocardial infarction (STEMI). When PCI is performed as an emergency in this setting, it is called 'primary' PCI. It is safer and more efficacious than thrombolysis and now used throughout the UK. In patients presenting with non-ST elevation myocardial infarction (NSTEMI) and unstable angina, routine early mechanical revascularization (PCI or CABG, determined by clinical and technical considerations), combined with appropriate medical therapy, also reduces later myocardial infarction and cardiovascular mortality.

The mechanics of PCI

When the balloon is inflated in a narrowed coronary artery (Figure 1), the atheromatous plaque is disrupted, deep fissures extend through the intima into the media, and some atheromatous material is displaced outwards into the vessel wall. Any plaque-free segments are stretched. When the balloon is deflated, arterial wall elasticity causes some recoil. If no stent is implanted, there is a 5% risk of acute vessel occlusion in the first 24 hours (acute vessel thrombosis). This is caused by a combination of dissection flaps and platelet-rich thrombosis at the dilated site. Slow blood flow and focal arterial spasm can worsen this. Without a stent, the dilated segment heals over the next 6 months.

Two aspects of healing threaten to re-narrow the newly opened lumen – the external arterial diameter can decrease (negative remodelling), and smooth muscle cells in the media can proliferate and migrate to re-line the damaged arterial lumen with a neo-intimal layer (Figure 2). If the lumen becomes sufficiently re-narrowed to obstruct blood flow ('re-stenosis'), symptoms can recur after an initial angina-free period of a few weeks. After 6 months, cellular proliferation and vessel remodelling become quiescent, so the artery usually remains patent in the long term if re-stenosis has not occurred by then. Re-stenosis rates without stent implantation are 20–50%.

Stents

Stents were introduced in 1990 and revolutionized PCI. The acute results of PCI became much more predictable, with a greatly reduced risk of sudden early vessel occlusion. Stents are now used in >90% of all PCI procedures.

The first stents were made of metal mesh (usually stainless steel or cobalt–chrome alloy). They prevented acute elastic recoil and held back dissection flaps. This reduced the risk of vessel occlusion in the first 24 hours (acute stent thrombosis) to <1%. During healing, the mesh's rigidity prevented negative remodelling, leaving neo-intimal hyperplasia as the only factor causing re-stenosis. This resulted in a re-stenosis rate of about 10–30%. It remained significant not only because of recurrent

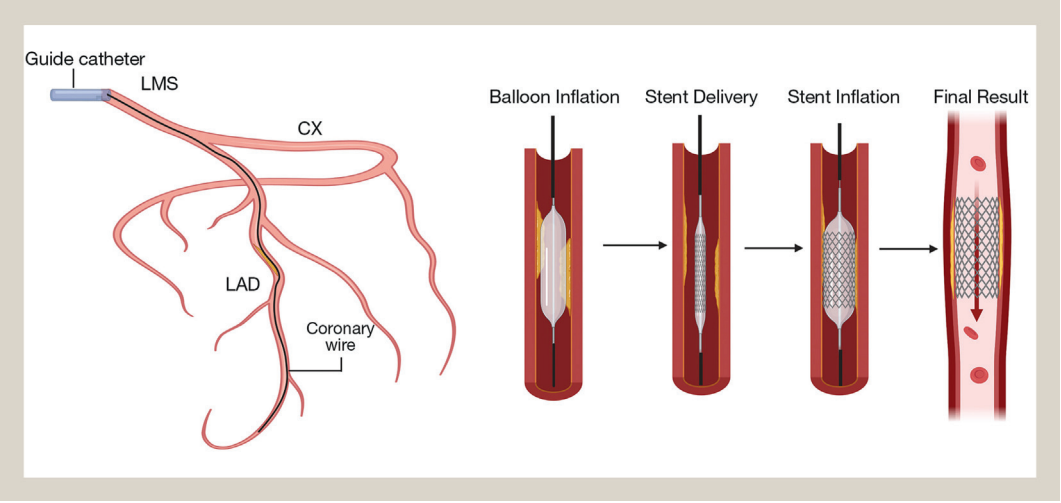

Figure 1 PCI. All equipment is introduced through the guiding catheter, whose soft tip is positioned co-axially within the coronary artery. Intra-arterial pressure can be monitored and radiographic contrast injected through this catheter. Most angioplasty equipment can be introduced through a 6 French gauge guide (external diameter 2 mm), although some more complex procedures can require larger diameter guides. Using X-ray imaging an angioplasty guidewire (0.014 inch diameter) is advanced and steered across the stenosis. A small cross-sectional diameter balloon with a slippery coating can then be advanced over this wire and inflated for 10–30 seconds (occluding coronary blood flow) to prepare the lesion. The balloon is withdrawn and a balloon-mounted stent is then introduced and deployed usually at 10–18 atm. The balloon is withdrawn leaving the stent mesh pressed firmly up against the vessel wall. Further dilatation with a non-compliant balloon is often performed to ensure the stent is fully expanded. Careful angiographic assessment ensures an optimal result, and if a dissection is detected, this requires further stent deployment to tack back the dissection flap. CX, circumflex; LAD, left anterior descending; LMS, left main stem.

symptoms, but also because re-stenosis presents as an acute coronary syndrome in over one-third of patients.

Drug-eluting stents

In the next iteration of therapy, drug-eluting stents were developed, which are now used in most PCI procedures. These stents have been modified to elute antimitotic drugs into the vessel wall for a few weeks after implantation. The stent is usually coated with a polymer that releases the drug. The inhibition of cellular proliferation reduces neo-intimal formation and profoundly decreases the re-stenosis rate to about 5%. This is a relative reduction of about 70% in every patient subgroup so far tested, including situations with a high risk of re-stenosis, such as long lesions and small vessels, and patients with diabetes mellitus.

Nevertheless, these benefits came at a cost. The lack of a proliferative response left some stent struts uncovered. It also became apparent that the polymer itself could promote an inflammatory response. This resulted in occasional episodes of stent thrombosis occurring much later after stent implantation than previously observed. Patients treated with first-generation drug-eluting stents had an annual rate of 'late stent thrombosis' of 0.4–0.6% for at least 3 years after PCI.

Current 'third-generation' drug-eluting stents have a much better long-term safety profile because of changes in design. They have thinner stent struts and a more inert or absorbable polymer coating, which may be restricted to the stent's abluminal surface. Some stents elute drug without needing a polymer coating. Late stent thrombosis rates are now <1% at 6 years and less than those with bare metal stents.[3]

Absorbable stents

Modern drug-eluting stents have allowed PCI to be predictable, efficacious and safe in the short and long term. Nevertheless, they remain as permanent implants in the coronary arteries. Their rigidity alters flow dynamics, abolishes vasoreactivity and can promote continuing inflammation. Bioabsorbable materials have been explored in the hope they will perform like conventional stents but later be fully absorbed, leaving a coronary artery that might be able to regain normal vascular responses. Trial results with polymer-based scaffolds have been disappointing, with higher rates of stent thrombosis. Initial data with magnesium scaffold in a small number of patients have, however, shown good clinical and safety outcomes and this remains an area of continuing research.

Adjunctive pharmacotherapy for PCI

Antiplatelet treatment during PCI

Early work with bare metal stents demonstrated that antiplatelet agents were pivotal to reducing stent thrombosis, both during the procedure and in the first few weeks after implantation. Dual antiplatelet therapy (DAPT) was introduced to minimize stent thrombosis. Aspirin (an irreversible inhibitor of the cyclo-oxygenase pathway) is often combined with clopidogrel (which irreversibly blocks the adenosine diphosphate $P2Y_{12}$ receptor).

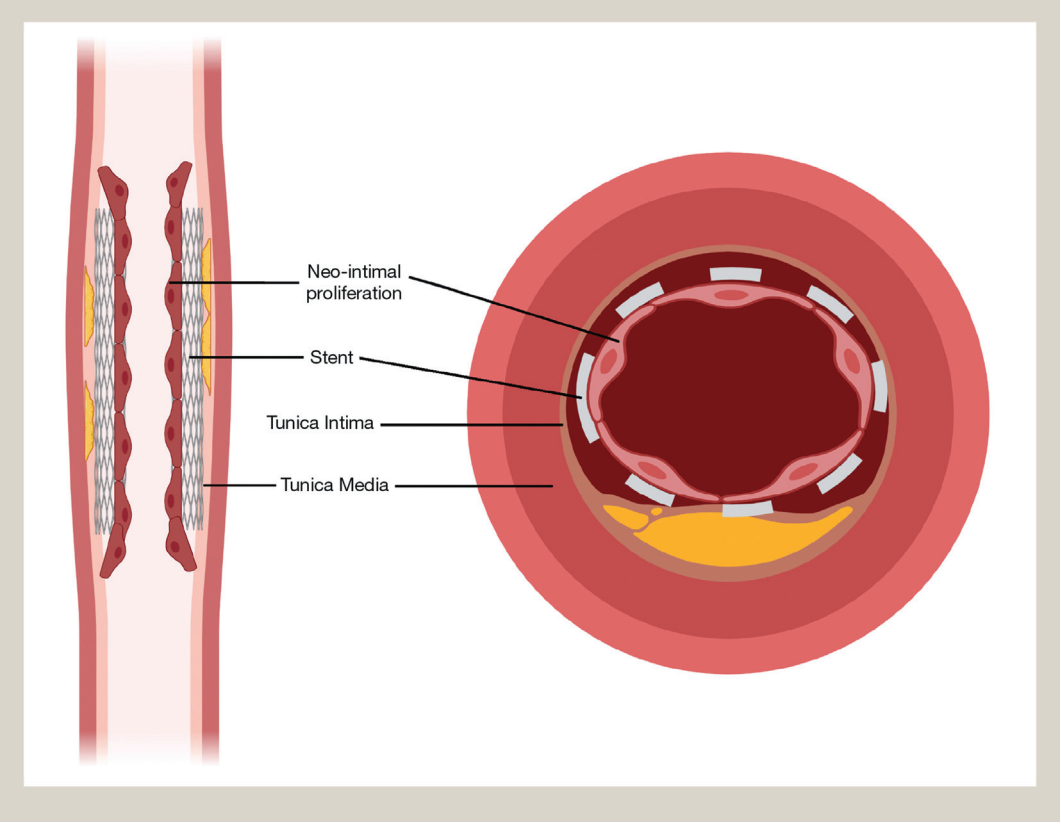

Figure 2 Mechanism of re-stenosis. Stent struts prevent elastic recoil and negative remodelling; however, a neo-intimal response to a stent can occur, leading to re-stenosis in about 5% of cases.

For patients presenting with an acute coronary syndrome, more potent agents have better outcomes, and the newer agents prasugrel and ticagrelor are used instead of clopidogrel. These are also $P2Y_{12}$ inhibitors but show less interindividual variability in antiplatelet response. Prasugrel gives better outcomes in higher risk patients, such as those with diabetes mellitus and those being treated for STEMI, but is contraindicated in patients with a history of transient ischaemic attack or stroke. It is also associated with increased bleeding risk in patients weighing <60 kg or aged 75 years or over.

Ticagrelor is the first oral reversible $P2Y_{12}$ antagonist: existing platelets are rapidly reactivated after its discontinuation. Compared with clopidogrel, ticagrelor is associated with reduced vascular mortality and myocardial infarction, but an increased risk of bleeding. Ticagrelor must be taken twice daily, and although missed doses carry a higher risk of platelet reactivation, there are potential advantages if emergency surgery is required.

A relatively common adverse effect of ticagrelor is dyspnoea (10–20% of patients). It is often a rather sudden onset of unexpected air hunger that can be brief or last up to a few minutes, but widely varying patterns can occur. It usually occurs at rest, not with exertion and is probably caused by increased concentrations of extracellular adenosine because of ticagrelor blockade of adenosine reuptake. When troublesome the drug may need to be switched to a different antiplatelet agent.

There are also intravenous options, which can be of particular benefit in emergency PCI. Rapid and reproducible serum concentrations of drug can be achieved as this route avoids the need for gastric absorption. The final common pathway leading to platelet aggregation involves activation of the glycoprotein (GP) IIb/IIIa receptor. Three inhibitors of this receptor are available: abciximab (a chimaera monoclonal antibody), tirofiban and eptifibatide (both small molecules). GP IIb/IIIa receptor blockade reduces the risk of peri-procedural myocardial necrosis and improves long-term outcome. Evidence of benefit is particularly strong in individuals with diabetes mellitus or an acute coronary syndrome, particularly when serum troponin is elevated (although most of this evidence was accumulated before the routine use of $P2Y_{12}$ antagonists). Cangrelor is a newer, short-acting intravenous $P2Y_{12}$ inhibitor with an evolving role in patients treated by PCI for acute coronary syndromes.

Antiplatelet treatment after PCI

In parallel with developments in stent design, there has been an evolution in the use of adjunctive antiplatelet therapy. Rapid endothelialization of the original bare metal stent struts meant that, after about 4 weeks, the risk of stent thrombosis was low, and it appeared safe to revert to a single agent. Rates were <0.4% up to 1 year and 0.2% at 1–4 years after implantation. However, when drug-eluting stents were first introduced, the devastating complication of late stent thrombosis, carrying high mortality, was observed. This prompted the recommendation to continue DAPT for a year, but this came at the cost of increased bleeding complications, themselves associated with increased mortality. Fortunately, second- and third-generation stents are much less thrombogenic, and, in combination with the more potent antiplatelet drugs (prasugrel, ticagrelor), the optimal duration of differing drug combinations of these drugs is less certain. A more tailored approach to therapy is being developed, taking into account a patient's presenting syndrome (stable, acute) and bleeding risk.

DAPT with aspirin and clopidogrel is currently recommended for 6 months for patients treated with stable angina before reverting to long-term aspirin alone. Shorter courses of DAPT are possible, with data supporting 3 months, and for some stents just 4 weeks, and can be considered in patients who have a high bleeding risk. For patients treated in the context of an acute coronary syndrome, studies suggest that the optimal duration of DAPT may be 12 months (whether treated by PCI or not), so a combination of aspirin and prasugrel, or aspirin and ticagrelor is often used. The latest drug-eluting stents do, however, offer the opportunity for some patients to reduce the duration of DAPT without an increase in stent thrombosis. On the other hand, for some individuals with a high thrombotic risk, particularly if they have tolerated DAPT without bleeding, there may be benefit to extending DAPT beyond a year.[4]

Anticoagulant therapy before and during PCI

NSTEMI patients are usually pre-treated with enoxaparin or fondaparinux before undergoing PCI. Fondaparinux has selective anti Xa activity through its link with antithrombin and has been shown to significantly reduce bleeding complications in comparison to enoxaparin. During a PCI procedure, in addition to antiplatelet drugs, patients are treated with antithrombotic drugs to reduce the risk of thrombus formation at the PCI site and on the intra-arterial equipment. Unfractionated heparin is usually given as an intravenous bolus to maintain anticoagulation during the procedure (monitored by measuring the activated clotting time). Other options include low-molecular-weight heparin (enoxaparin) and bivalirudin (a direct thrombin inhibitor), although recent evidence suggests no additional benefit of bivalirudin over unfractionated heparin.

Anticoagulant therapy after PCI

After PCI, for patients needing anticoagulant therapy (e.g. atrial fibrillation, prosthetic heart valve, previous pulmonary emboli), various combinations of antiplatelet therapy, warfarin and direct oral anticoagulant drugs are used. There are many options, well summarized in the latest European Society of Cardiology guidelines. There should be a low threshold for the use of proton pump inhibitors to reduce the risk of gastrointestinal adverse effects.

The technique of PCI (see Figure 1)

The aim of almost all PCI procedures is to dilate a stenosed coronary artery, or open an occluded one and then implant a stent. This requires access to the patient's arterial system. PCI is performed in an angiography suite with continuous monitoring of the electrocardiogram (ECG), arterial pressure and oxygen saturation. Full resuscitation facilities, including a defibrillator, intra-aortic balloon counterpulsation and anaesthetic back-up, must be readily available, as must access to immediate echocardiography and temporary pacing. Arterial access is usually achieved via the right radial artery or right femoral artery. There

are fewer access site complications if the radial route is used, and this has now become the dominant method in the UK (see 'Arterial access for cardiac procedures' on pages 465–466 of this issue). Patients are often given light sedation (such as an intravenous benzodiazepine with or without fentanyl).

Using X-ray imaging, a guiding catheter is advanced until the tip is at the ostium of the coronary artery to be treated. This serves as a conduit to inject radiographic contrast, measure arterial pressure and introduce equipment into the coronary circulation to perform the procedure. A fine guidewire (commonly 0.014 inch/0.36 mm in diameter) is advanced into the coronary artery and steered across the stenosis to be treated. The distal portion of this wire is very flexible and is positioned in the distal bed of the vessel, leaving the stiffer shaft across the stenosis; here it acts as a rail over which all subsequent equipment is passed.

A balloon is then usually passed over this wire and inflated in the stenosis to enlarge the lumen. This is then deflated and withdrawn, and next a stent, already crimped on a deflated deployment balloon, is fed over the guidewire into the same position at the site of the coronary stenosis. This is inflated, expanding the stent struts against the arterial wall, thus scaffolding it open. The balloon is deflated and removed, leaving the stent in position.

After radial access, the arterial sheath is immediately withdrawn and a compression device positioned over the puncture site. It is important to achieve 'patent haemostasis', whereby sufficient pressure is applied to prevent bleeding but the radial artery remains patent. This reduces the risk of later radial artery occlusion. Patent haemostasis can be achieved by reducing the pressure in the compression device until a palpable radial pulse returns, or by using the Allen test as a guide. In this, an oxygen saturation probe is placed on the index finger. Pressure is applied to the ulnar artery to occlude it, and with the radial compression device also at supra-arterial pressure, the finger desaturates of oxygen. The pressure in the radial compression device is then reduced until radial flow is restored, identified by a return to normal oxygen saturation in the finger.

If femoral access has been used, there is a greater risk of bleeding from the puncture site. This can be mitigated by administering protamine sulfate at the end of the procedure to allow immediate sheath removal, or by delaying sheath removal for 4 hours to allow the effects of peri-procedural heparin to wear off. After sheath removal, haemostasis can be achieved using manual pressure. An arterial closure device (a polypropylene suture or polyethylene glycol sealant plug applied to the hole in the artery during sheath removal) is frequently used to improve the chances of earlier haemostasis.

Post-procedure care

On return to the ward

An ECG is performed on return to the ward, and ECG rhythm is monitored continuously for the first few hours, with frequent haemodynamic observations. After a radial access procedure, it is important to check that the haemostatic pressure device is achieving patent haemostasis (as above); this minimizes the risk of late radial artery occlusion. With femoral closure devices, the puncture site must be inspected regularly for recurrent bleeding, which can usually be treated with a further period of manual pressure. Mobilization is usually possible after 4–6 hours (earlier after a radial procedure), but should be delayed if a haematoma has formed or a GPIIb/IIIa receptor antagonist has been used.

Management of early complications

Patients may notice mild central chest ache after returning to the ward. This can result from stretching of the coronary adventitia or peri-procedural myocardial necrosis (caused by occlusion of a small side branch or microembolic phenomena). It usually resolves gradually over an hour or two.

Any increase or new occurrence of chest pain can signify abrupt occlusion of the treated vessel. The ECG usually shows ST segment elevation or new T wave changes. The person should immediately return to the catheter laboratory so the vessel can be recanalized by emergency repeat PCI.

Hypotension in the absence of myocardial ischaemia usually has one of two causes: haemorrhage or cardiac tamponade. Haemorrhage from the arterial access site may be obvious, but unnoticed bleeding can track from the femoral artery into the retroperitoneal space, so the diagnosis requires emergency abdominal computed tomography. Rarely, cardiac tamponade results from coronary arterial perforation, or right ventricular wall perforation if a temporary pacing wire has been used. There may or may not be pericardial pain, and the patient can rapidly become profoundly hypotensive. Immediate echocardiography is essential to confirm the diagnosis, and treatment is by emergency pericardiocentesis.

Radiographic contrast

Radiographic contrast agents can induce nephropathy. The risk is related to the amount of contrast used and is highest in individuals with significant chronic kidney disease. It can be predicted from other clinical characteristics using a risk score. The rise in serum creatinine usually occurs within 24 hours and peaks at 3–4 days. There is usually spontaneous resolution, but in some cases it progresses and can require renal replacement therapy. Where possible, a second exposure to contrast should be delayed for at least 72 hours.

Pre-procedural hydration remains the cornerstone of prevention. A variety of fluids have been used (including sodium chloride 0.9%, glucose 5% and sodium bicarbonate 1.26%). Care is needed in patients with poor left ventricular function to avoid precipitating pulmonary oedema.

Major complications

The need for emergency cardiac surgery has been greatly reduced by the availability of stents; it now occurs in <0.1% of PCI procedures. A peri-procedural cerebrovascular accident occurs in about 0.3–0.4% of cases. In-hospital mortality is rare (overall about 1%). The bulk of procedural risk is determined by patient and clinical characteristics: patients treated for stable angina have a mortality risk of about 0.2%, those treated for NSTEMI and unstable angina about 0.6%, those treated for STEMI about 5%, and those presenting in cardiogenic shock up to 50%.

Late outcomes

All-cause mortality after PCI in randomized controlled trials is approximately 2% per annum, with no difference between bare metal or drug-eluting stents.

Other important interventional equipment and techniques

Intravascular ultrasound (IVUS)

Early atheroma does not cause luminal narrowing and is therefore invisible at angiography. IVUS produces cross-sectional images of all layers of the arterial wall, providing a complementary imaging method (Figure 3). The miniaturized probe is introduced over the angioplasty guidewire and is particularly helpful in guiding PCI of the left mainstem and when angiographic appearances are ambiguous.

Swept laser imaging (including optical coherence tomography)

This technique uses optical reflections of near infrared light, emitted from a catheter with a fibreoptic core positioned in the coronary artery over a guidewire. Blood is displaced from the artery by injecting clear fluid (contrast, sodium chloride), and a series of cross-sectional optical images is obtained during rapid mechanized catheter withdrawal. This provides much higher image resolution than IVUS (10–15 micrometres compared with 150–200 micrometres) but less depth resolution (Figure 4). It allows details such as the cellular covering of stent struts to be investigated even though the imaging depth is only about 2 mm.

Pressure wire measurement of fractional flow reserve

Although the coronary angiogram provides exquisite anatomical detail of the arterial lumen, it does not provide functional information. A very tight stenosis or occlusion can be assumed to be causing an obstruction, but many stenoses are of intermediate angiographic severity. Their haemodynamic significance can be assessed by measuring the pressure drop across the stenosis while minimizing microvascular resistance with intravenous adenosine.

The measurement is made by comparing pressure in the aortic root (from the guiding catheter) with that measured distal to the stenosis by a miniaturized pressure transducer incorporated into an angioplasty wire (a pressure wire). The ratio of proximal to distal mean pressure is known as the fractional flow reserve. In a normal vessel, the ratio is 1 (i.e. there is no pressure drop), but across functionally significant stenoses the ratio falls to 0.8 or less. Only obstructive lesions cause angina and require PCI. In multivessel disease, pressure wire assessment clarifies which lesions require treatment.[5]

Other methods to assess the haemodynamic effects of stenoses without needing to use adenosine (e.g. instantaneous wave-free ratio) are starting to gain some traction as evidence for their validity strengthens.

Drug-eluting balloons

Concerns about late stent thrombosis with metallic stents have led to an increase in use of drug-eluting balloons. These are coated with lipophilic drugs (usually paclitaxel, although sirolimus balloons are also available) and work by transfer of the drug to the vessel wall despite short contact times. Most of the trial evidence supporting their use is in the arena of re-stenosis although there is interest and ongoing studies relating to *de novo* small vessel disease and bifurcations.

Atherectomy

Balloon dilatation disrupts and moves plaque but does not remove it. In heavily fibrocalcific plaque, the expansion forces

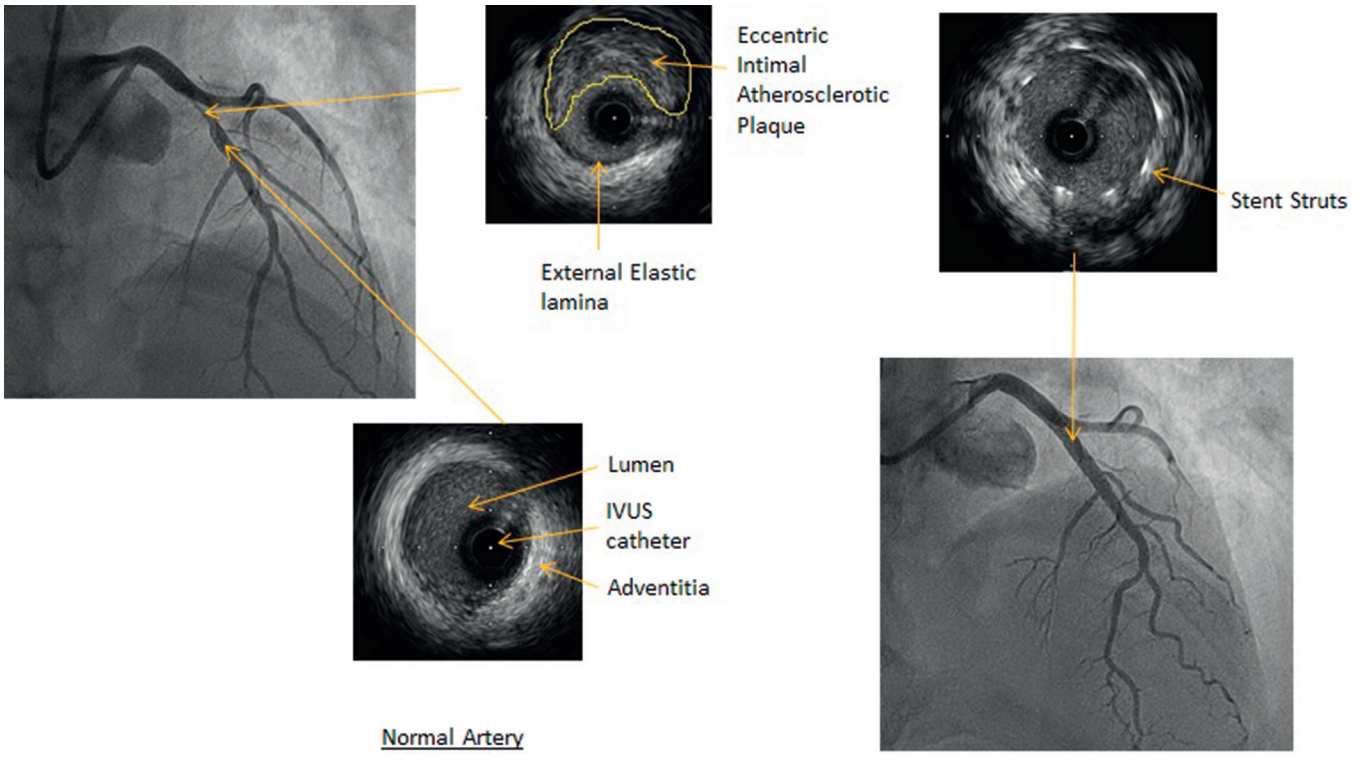

Figure 3 Angiographic and cross-sectional IVUS of an artery obtained before and after stent implantation. The external elastic lamina separates the echo-dense adventitia from the echo-lucent media. The internal elastic lamina separates media from intima. Various features of the arterial wall are demonstrated that are not apparent on the angiographic image (including the intra-coronary stent that has been inserted).

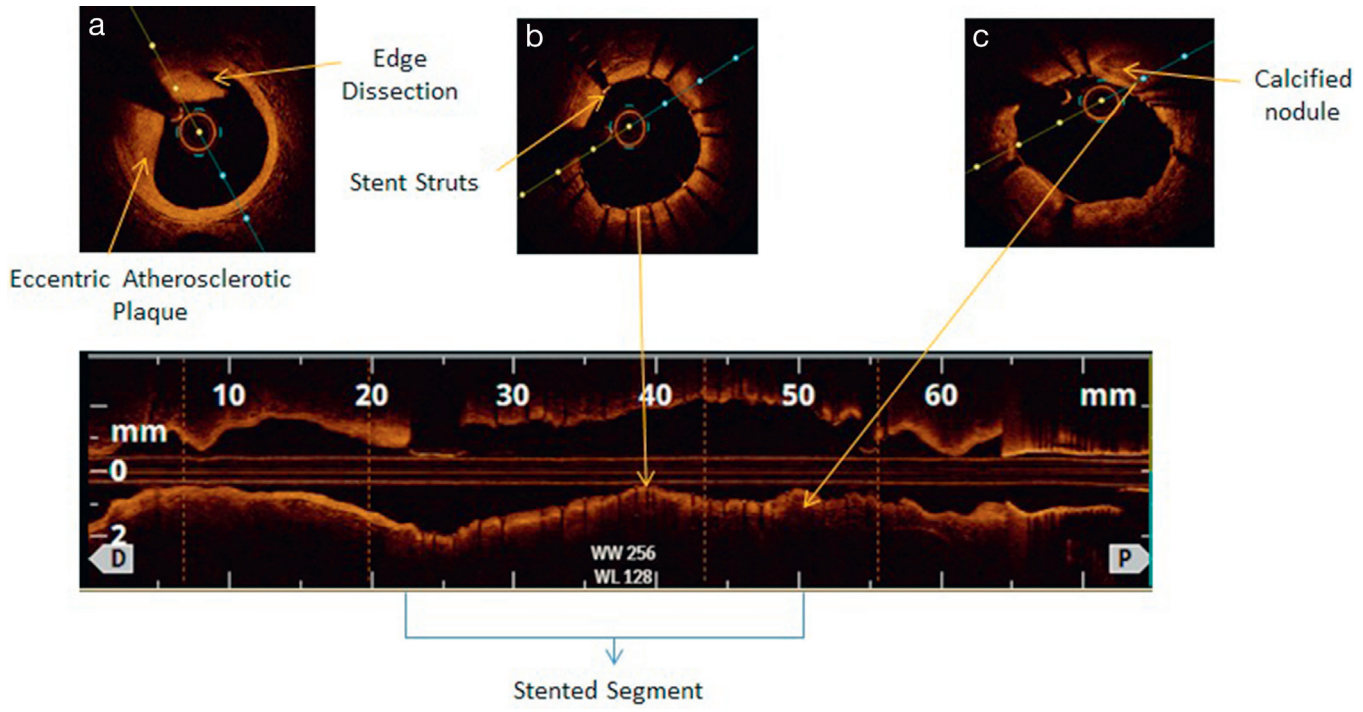

Figure 4 Optical coherence tomography. A longitudinal reconstruction (lower panel) and cross-sectional images (upper panel) acquired using a Dragonfly (Abbott, Santa Clara, CA, USA) optical coherence tomography catheter system at 20 mm per second pullback speed immediately after stent implantation. A 7 cm coronary segment was imaged using a 4-second contrast injection. Several important findings can be seen. (**a**) Distal stent edge dissection within the atherosclerotic plaque. (**b**) Well-expanded and well-apposed stent struts, with the corresponding longitudinal view (arrow). (**c**) Proximal calcified plaque behind the stent stuts, with the corresponding longitudinal view (arrow).

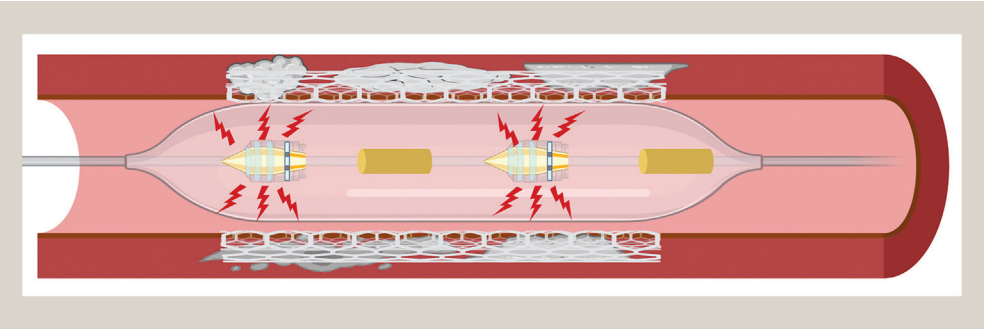

Figure 5 Intravascular lithotripsy balloon. Expanding vapour bubbles create acoustic pressure waves that can pass through soft arterial tissue to cause a localized field effect and disrupt calcified plaque. This allows for vessel modification and adequate stent expansion within the lumen of the vessel.

from a balloon can be insufficient to dilate a stenosis. In these circumstances, successful dilatation can be achieved by first modifying the plaque. Rotational atherectomy involves the use of a spinning diamond-tipped burr advanced along the guidewire to ablate the calcific plaque. Microparticles so created are flushed downstream and pass through the microvasculature. Alternatively, the plaque can be modified using an excimer laser to vaporize non-aqueous components. After both methods, a balloon is still required to dilate the vessels before stent implantation.

Intravascular lithotripsy

This is a novel balloon technique that uses acoustic pressure waves to deliver energy to the vessel wall to allow fracture of superficial and deep calcium. The acoustic energy results in the formation of micro-cracks that then allow for adequate vessel expansion to occur and reduce the risk of having under-expanded stents with the vessel lumen (Figure 5). A particular advantage of this device is that it can be passed over a standard guidewire. ◆

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