Percutaneous coronary intervention

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Abstract

Percutaneous coronary intervention (PCI) is now the dominant method for mechanically improving myocardial perfusion in the treatment of coronary artery disease. The procedure is performed via a small intra-arterial sheath and usually involves a single overnight stay in hospital. Day-case treatment is now possible in many cases. A balloon is used to dilate the coronary stenosis and a stent then implanted to scaffold the vessel. Renarrowing at the treated site may occur owing to neointimal proliferation but stents with drug-eluting coatings have profoundly reduced this problem. Most acute complications of PCI are mediated by platelet activation, so that appropriate drug combinations that block platelet aggregation are critical to the safety of the procedure and later outcomes. Early complications include haemorrhage from the arterial access site, abrupt vessel closure and cardiac tamponade. The requirement for emergency CABG is now about 0.1% and in hospital mortality about 1%. Technical advances mean that patients with ever more complex coronary artery disease can now be safely treated by PCI.

Keywords aspirin; atherectomy; clopidogrel; distal protection; drug-eluting stents; glycoprotein IIb/IIIa inhibitor; intravascular ultrasound; optical coherence tomography; percutaneous coronary intervention; percutaneous transluminal coronary angioplasty; prasugrel; stents; pressure wire; rotablation

The term 'percutaneous coronary intervention' (PCI) applies to various procedures that mechanically improve myocardial perfusion without resorting to surgery. The most common procedure is percutaneous transluminal coronary angioplasty (PTCA), involving inflation of a balloon within the coronary artery to enlarge the lumen and allow implantation of an intracoronary stent. Other methods may be appropriate in small subsets of patients. More than 80,000 PCI procedures were performed in the UK in 2008.[1]

The role of PCI

PCI is now the dominant method for achieving myocardial revascularization, with fewer patients needing coronary artery bypass grafting (CABG).

Acute coronary syndromes

PCI improves the prognosis and symptoms in patients presenting with acute coronary syndromes, particularly ST-elevation myocardial infarction (STEMI). PCI for STEMI (so called 'primary PCI') is more effective and safer than thrombolysis.[2] In non-ST-elevation myocardial infarction (NSTEMI) and unstable angina (UA), a strategy of early mechanical revascularization (PCI or CABG, choice determined by technical considerations) in combination with appropriate medical therapy also reduces later coronary events and mortality.[3]

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Stable angina

Mechanical revascularization (CABG or PCI) should be considered in patients with angina despite medical therapy (or those in whom medication is poorly tolerated because of adverse effects). PCI is both safe and effective in reducing angina in such patients,[4] and may improve prognosis where high risk features are present on non-invasive testing.[5] The choice between PCI and CABG is determined mainly by technical considerations. For example, totally occluded vessels are more easily treated by surgery (but see below for new PCI techniques). In patients, suitable for either technique, long-term mortality outcomes are similar; PCI is associated with fewer strokes but involves more repeated procedures.[6] The Syntax trial [7] has provided important insights into the relative roles of PCI and CABG for the optimal treatment of left main stem or complex multivessel disease. Those without diabetes and with less extensive disease far better with PCI, whereas CABG provides better outcomes for those with diabetes and very extensive disease.[8] In some patients (particularly diabetics with extensive coronary artery disease and impaired left ventricular function) CABG may offer prognostic benefit and should be considered, even if patients are asymptomatic.

Response of the artery to PCI

Balloon inflation causes vessel dilatation by several mechanisms. The atheromatous plaque is disrupted, deep fissures extend through the intima into the media, some atheromatous material is displaced outward into the vessel wall, and any plaque-free segments are stretched.

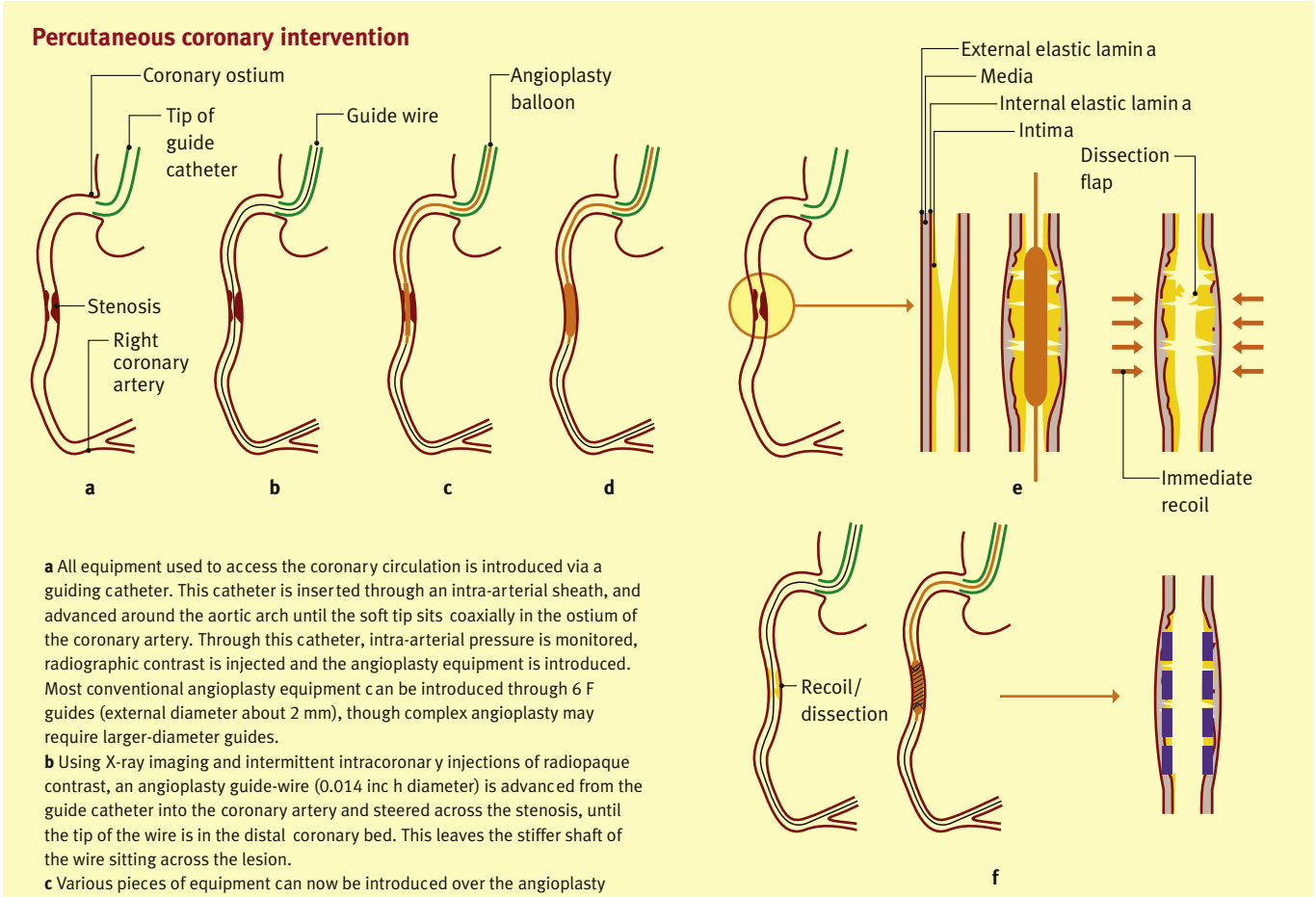
Plain balloon angioplasty

Early response: when the balloon is deflated, the elasticity of the arterial wall causes some recoil. In the first 24 h, there is a 5% risk of acute vessel occlusion caused by a combination of dissection flaps and platelet-rich thrombosis at the dilated site (Figure 1e). Slow blood flow and focal arterial spasm may exacerbate the problem.

Late response: during the next 6 months, the dilated segment heals. Two aspects of healing threaten to narrow the newly opened lumen — the external arterial diameter may decrease (negative remodelling), and smooth muscle cells in the media proliferate and migrate to reline the damaged arterial lumen with a neo-intimal layer (Figure 2). If the lumen becomes sufficiently re-narrowed to obstruct blood flow ('restenosis'), symptoms may recur after an initial angina-free period of a few weeks. If restenosis does not occur in the first 6 months, the artery will remain patent in the long term. Restenosis rates are 20–50%.

Stents

Bare metal stents: stent implantation has greatly improved outcome (both by improving acute safety and later restenosis)



a All equipment used to access the coronary circulation is introduced via a guiding catheter. This catheter is inserted through an intra-arterial sheath, and advanced around the aortic arch until the soft tip sits coaxially in the ostium of the coronary artery. Through this catheter, intra-arterial pressure is monitored, radiographic contrast is injected and the angioplasty equipment is introduced. Most conventional angioplasty equipment can be introduced through 6 F guides (external diameter about 2 mm), though complex angioplasty may require larger-diameter guides.

b Using X-ray imaging and intermittent intracoronary injections of radiopaque contrast, an angioplasty guide-wire (0.014 inch diameter) is advanced from the guide catheter into the coronary artery and steered across the stenosis, until the tip of the wire is in the distal coronary bed. This leaves the stiffer shaft of the wire sitting across the lesion.

c Various pieces of equipment can now be introduced over the angioplasty guide-wire and across the lesion. If balloon dilatation is planned, an angioplasty balloon is introduced onto the guide-wire and advanced so that it sits in the coronary artery at the site of the stenosis. Before this balloon is inflated, it has a very small cross-sectional diameter, which, in conjunction with a slippery outer coating, allows it to be manipulated across even extremely tight and tortuous stenoses.

d and **e** Once positioned, the balloon is inflated for about 10 to 30 seconds (occluding coronary flow). The balloon is then deflated and withdrawn from the coronary circulation into the guiding catheter. Injection of contrast into the coronary artery during cine acquisition enables assessment of the result.

f In most procedures, an intracoronary stent (a cylindrical steel mesh) is then deployed. Inflation pressures used for stent deployment are usually higher (12–20 atmospheres). After about 15–30 seconds, the balloon is deflated and withdrawn into the guiding catheter, leaving the stent mesh pressed firmly against the walls of the coronary artery. Advances in stent design are such that it is now often possible to position a stent across a tight stenosis without pre-dilating the lesion (so-called 'primary stent implantation').

After stent deployment, careful angiographic assessment ensures that an optimal result has been achieved. The stent should be fully expanded, with no dissection at the stent edges (which would increase the risk of subacute stent thrombosis and later restenosis). Further balloon dilatation to a higher pressure or use of balloons of larger diameter may be required, and further stents may need to be implanted to tack back dissection flaps.

Figure 1

and is now used in over 90% of all PCI procedures. The metal mesh (usually stainless steel or cobalt–chrome alloy) prevents acute elastic recoil and holds back dissection flaps (Figure 3) to reduce the risk of vessel occlusion in the first 24 h to <1%. During healing, the rigidity of the mesh prevents negative remodelling. Neointimal hyperplasia is the only factor causing restenosis. Although this is less common when stents are deployed, restenosis remains a limitation (10–30% of cases). Sudden occlusion can occur, as a result of platelet aggregation on the bare stent struts (acute and subacute thrombosis), especially during the first 4 weeks after implantation. Antiplatelet drugs and close attention to the technical aspects of stent implantation have reduced incidence to 1–2%. The stent struts are covered with new endothelial cells after 3–4 weeks and stent thrombosis is then very rare.

Drug-eluting stents: in order to reduce the problem of restenosis, the original 'bare metal' stents have been be modified to elute an antiproliferative drug into the vessel wall for a few weeks after implantation. This inhibits cellular proliferation, to reduce neointimal formation and profoundly inhibit the restenotic process (Figure 4). The first two drugs to be used were sirolimus (rapamycin; a cell cycle inhibitor, used in the Cypher™ stent) and paclitaxel (an inhibitor of cell microtubular function, used in the Taxus™ stent) (Figure 5). A wide variety of different stents have since been designed, using different stent mesh designs, different drugs, and different methods to control drug release into the vessel wall. Stents made entirely from biodegradable polymer are now also being developed – if successful, these will leave no residual material in the treated coronary artery.