Percutaneous coronary intervention

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Abstract

Percutaneous coronary intervention (PCI) is 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 not infrequent. A balloon is used to dilate the coronary stenosis and a stent is then implanted to scaffold the vessel. Re-narrowing at the treated site may occur but has been greatly reduced by drug-eluting stents. Most acute complications of PCI are mediated by platelet activation, so that drugs blocking platelet aggregation are pivotal to the safety of the procedure. Early complications include haemorrhage from the arterial access site (reduced by a radial approach). Abrupt vessel closure, CVA and tamponade are very rare. The requirement for emergency cardiac surgery is less than 0.1% and in-hospital mortality is mainly determined by the indication for PCI — about 0.2% in those with stable angina, 5% following STEMI and 30% to 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; aspirin; atherectomy; bivalirudin; clopidogrel; distal protection; drug-eluting stent; enoxaparin; glycoprotein IIb/IIIa inhibitor; heparin; intravascular ultrasound; laser; optical coherence tomography; percutaneous coronary intervention; prasugrel; pressure wire; rotablation; stent; thrombectomy; ticagrelor

Introduction

Atherosclerosis in coronary arteries usually becomes manifest clinically by causing stenosis and occlusions that reduce myocardial blood flow. The term 'percutaneous coronary intervention' (PCI) applies to various procedures that address obstructed coronary arteries to improve myocardial perfusion without the need to resort to coronary artery bypass surgery (CABG). The most common form of PCI starts with the inflation of a balloon within the stenosis of the coronary artery (called a percutaneous transluminal coronary angioplasty or PTCA), and then the implantation of one or more stents. Variations of this basic procedure are used in some subsets of patients and are described below. More than 92,000 PCI procedures were performed in the UK in 2012, with over five times as many patients being treated by PCI rather than by CABG.[1]

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What's new?

- Drug-eluting stents are more refined and safer with improved short- and long-term outcomes
- Fully absorbable stents are being developed, with encouraging early results
- Radial artery access reduces complications and is associated with lower mortality and is therefore now the dominant route for PCI in the UK
- Primary PCI has almost completely replaced thrombolysis as the preferred treatment for STEMI in the UK

Role of PCI in clinical syndromes

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

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,[2] and may improve prognosis where high-risk features are present on non-invasive testing.[3] PCI is associated with better outcomes than medical therapy alone when its use is guided by an invasive assessment of the functional significance of coronary stenoses (see below 'pressure wire').[4]

If revascularization is indicated in patients with complex multi-vessel disease, the choice between PCI and CABG is determined by a combination of clinical and technical considerations. In patients with diabetes mellitus, CABG is associated with better long-term survival than PCI.[5,6] In patients who do not have diabetes and have less widespread disease, PCI offers equivalent long-term mortality with a lower risk of CVA than CABG, albeit with an increased need for repeat PCI. Equivalent outcomes between CABG and PCI have been shown for the treatment of left main stem disease, providing patients do not have other very complex coronary disease.[5] To decide the optimal revascularization strategy for an individual patient can be complex, and needs to take into account symptoms, coronary anatomy, co-morbidity and personal choices. These decisions should be taken by a multidisciplinary 'heart team'.[7]

Acute coronary syndromes

PCI improves survival in patients presenting with acute ST-elevation MI (STEMI), and when a PCI is performed as an emergency treatment in this setting it is called a 'primary' PCI. It is safer and more efficacious than thrombolysis,[8] and has now almost completely replaced thrombolytic treatment in the UK. In patients presenting with non-ST-elevation myocardial infarction (NSTEMI) and unstable angina (UA), a strategy of routine early mechanical revascularization (PCI or CABG, choice determined by technical considerations) in combination with appropriate medical therapy also reduces later myocardial infarction and cardiovascular mortality.[3,9]

The mechanics of PCI

When a balloon is inflated in a narrowed coronary artery, the atheromatous plaque is disrupted, deep fissures extend through the intima into the media and some atheromatous material is displaced outward into the vessel wall. Any plaque-free segments are stretched. When the balloon is deflated, the elasticity of the arterial wall 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 (Figure 1e). Slow blood flow and focal arterial spasm may exacerbate the problem. In the absence of a stent, the dilated segment heals over the next six months. Two aspects of healing threaten to re-narrow the newly opened lumen – the external arterial diameter may decrease (negative re-modelling), and smooth muscle cells in the media 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 (a process called 're-stenosis'), symptoms may recur after an initial angina-free period of a few weeks. After 6 months, cellular proliferation and vessel re-modelling become quiescent, so that if re-stenosis has not occurred by this time, the artery usually remains patent in the long term. Re-stenosis rates without stent implantation are 20–50%.

Stents

Stents were introduced in 1990 and revolutionized the technique of PCI. The acute results of PCI became much more predicable with risk of sudden early vessel occlusion greatly reduced. Stents are now used in over 90% of all PCI procedures. The metal mesh

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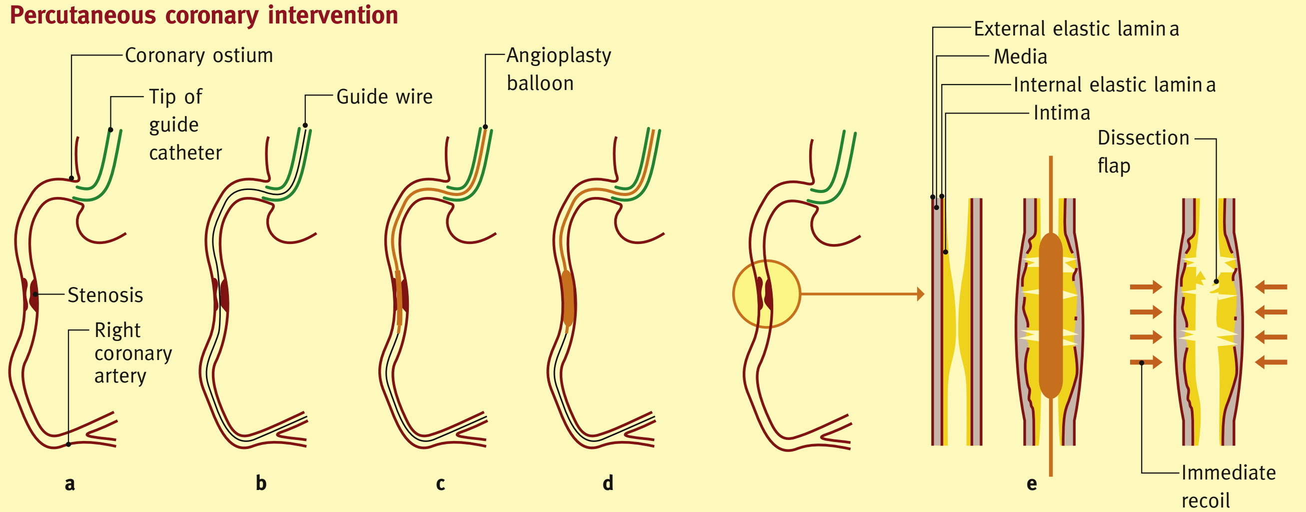

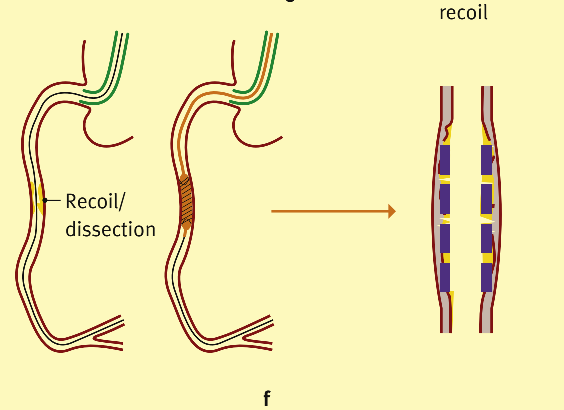

a All equipment used to access the coronary circulation is introduced via a guiding catheter. This catheter is inserted through an intra-arterial sheath, and advanced around the aortic arch until the soft tip sits coaxially in the ostium of the coronary artery. Through this catheter, intra-arterial pressure is monitored, radiographic contrast is injected and the angioplasty equipment is introduced. Most conventional angioplasty equipment can be introduced through 6 Fr 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