Editorial

Percutaneous transluminal coronary angioplasty: what next?

The concept of treating heart disease by means of intravascular catheters was first suggested by Werner Forssmann in 1929.1 These principles, however, lay dormant for almost half a century until 1978 when Andreas Gruentzig introduced percutaneous transluminal coronary angioplasty.2 Gruentzig was an imaginative and daring physician who envisaged that coronary recanalisation could be achieved by a minor non-surgical procedure if miniaturised balloons were used to dilate coronary artery stenoses.

Over the past decade the use of percutaneous transluminal coronary angioplasty has grown rapidly. This year about a quarter of a million percutaneous transluminal coronary angioplasty procedures will be performed in the United States alone. Percutaneous transluminal coronary angioplasty can successfully treat single, double, and triple vessel disease and, in properly selected patients, can achieve similar results to open chest surgery. There are now more percutaneous transluminal coronary angioplasty procedures than coronary bypass operations in western countries.

Despite the success of percutaneous transluminal coronary angioplasty during the past decade, several problems remain to be solved. Although most lesions, even the tightest ones and those found in the most remote sites, can be crossed and satisfactorily dilated by modern low profile angioplasty balloons, the short term and long term results remain unpredictable. About one in 20 percutaneous transluminal coronary angioplasty procedures result in occlusion (often abrupt) of the vessel,3 sometimes requiring emergency surgery. Of those patients who have an initially successful percutaneous transluminal coronary angioplasty, one in three has only temporary relief of symptoms; the lesion, for reasons that are not yet wholly known, recurs within a few weeks or months.4 In consequence, about 40% of patients undergoing percutaneous transluminal coronary angioplasty will not achieve a long term benefit from this "minor" procedure.

In the early years the enthusiasm for revascularisation of the myocardium via the natural routes of the circulation and without the need for surgery overwhelmed any scepticism. With increasing experience, however, many cardiologists became concerned about the high rate of restenosis and the need for repeated dilatations. Not infrequently three, four, or more dilatations would be required before recurrence was finally prevented, and this reduced substantially the effectiveness of this otherwise elegant procedure. Health authorities became aware of the cost of repetitive interventions, patients worried about excessive morbidity, and physicians became frustrated. Various solutions to these problems were proposed. Because thrombosis was believed to be an important factor, several expensive trials of different antithrombotic regimens were undertaken. Although these agents were effective in reducing the acute problems, none reduced the restenosis.5 Anti-inflammatory drugs, intended to reduce tissue proliferation, were also ineffective.6 The possibility that vasomotor tone affected the genesis of restenosis led to the use of calcium channel blockers, but no significant reduction of restenosis was shown.7 The results of giving dietary supplements of fish oil were conflicting. Currently, several large clinical trials with different enzyme inhibitors are either under way or about to be launched.

Mechanical approaches have included longer inflations and over-inflations intended to optimise the initial result, the removal of plaque material with the help of complex catheter systems (fluid jets, drills, shavers, lasers), and the permanent implantation of miniaturised endoluminal support devices (stents) made of metal or other biologically compatible substances.

Though removal of atheromatous material gives a fascinating insight into the histological composition of restenoses, it does not seem to reduce the rate of restenosis.8 Atherectomy may help to direct further research but so far it has not improved results. Laser ablation too has been beset by technical and safety problems for many years; preliminary clinical observations indicated few acute problems but the restenosis rate, at least for lasers that produce local heat, was disappointingly high.9,10 The restenosis rates after excimer laser ablation are as yet unknown.

The results with stents are more encouraging. If the acute phase passes without thrombosis then restenosis seems to be rare.11,12 The incidence of restenosis varies slightly according to the type of stent implanted, but can be well below the usual 33%. For self expanding mesh stents we found restenosis rates of under 10% in native arteries and only slightly higher in bypass grafts,13 which without stenting tend to produce many restenoses.14 If the incidence of percutaneous transluminal coronary angioplasty procedures remains stable, such a reduction in restenosis rate would produce substantial savings—about 2 billion dollars per year in the United States. Stents were also effective in the management of acute occlusion after percutaneous transluminal coronary angioplasty15 and restenosis is rare in patients who have stents for this indication.

Physicians are reviewing their attitudes to percutaneous transluminal coronary angioplasty. Marketing reports indicate that the growth of percutaneous transluminal coronary angioplasty in the United States is slowing; for 1989 the increase over 1988 is estimated to be 9%—a considerably smaller increase than in earlier years. The reasons for this change are complex; certainly if the demand for percutaneous transluminal coronary angioplasty is about to level out competition among centres should encourage better quality control and should deter operators with a weak performance record. This change in attitude, however, largely results from the increasing appreciation of the limitations of percutaneous transluminal coronary angioplasty. The results of studies such as TIMI II have clearly added to this trend.16

For the first 10 years of experience with the procedure, percutaneous transluminal coronary angioplasty was a rather crude way of treating diseased arteries. I am
convinced that the best is yet to come. In future percutaneous transluminal coronary angioplasty is likely to focus on hybrid therapeutic strategies: pressure remodelling with balloons; removal of atherosclerotic material if necessary (possibly under angioscopic vision) by cutters, drills, or lasers that analyse their target before destroying it; and last but not least, supporting the artery with non-thrombogenic, possibly biodegradable stents. These stents must be easily implantable and must limit proliferation. The technology exists: we have to direct our imagination and research towards the choice of implantable materials and local delivery systems for drugs that modulate the control of vasomotor tone and promote optimal healing of the vessel wall. Most importantly, we need to identify and eliminate the stimuli that lead to atherosclerosis and restenosis.

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