In conclusion, rotational atherectomy definitively has a place in high volume interventional cardiology centres, in the era of stenting. It requires technical training and experience, and special patient care in the prevention and treatment of complications, mainly the slow-flow phenomenon. It can increase procedural success in a subset of complex lesions and in small vessels, such as those described in the COBRA trial, and can always be tried in cases when the balloon or a stent cannot reach or cross a lesion (‘rota-rescue’). The strategy of debulking prior to balloon dilatation or stenting (‘rotastenting’) to enhance long-term results and reduce restenosis, still needs to be proven, and the same holds true with the current knowledge of rotablation for in-stent restenosis.

R. SEABRA-GOMES
Hospital Santa Cruz,
Lisbon, Portugal

References

Intracoronary drug delivery: mechanically too rough, pharmacologically too weak?

See page 1767 for the article to which this Editorial refers

Restenosis is a major problem in coronary interventions. Despite considerable research and clinical effort, despite a large volume of detailed knowledge about its mechanisms, cardiologists are still partially failing in this battle. Stents have succeeded in decreasing angiographic as well as clinical restenosis rates, but they have also introduced a new and more difficult problem: in-stent restenosis. A large number of pharmacological agents have been tested in the battle with restenosis: all of them have failed. Conceptually, local delivery of these drugs should be effective. Drug concentration at the target site is up to 1000 × higher than systemic concentration[1].

Meneveau et al.[2] in this issue present the results of the IMPRESS trial. Local intracoronary intramural delivery of androprin after stent implantation showed no benefit in this well designed, clinically very well performed study. The detailed analysis of these data may even show a trend (not significant) towards more angiographic complications (2 × more filling defects, 5 × more haziness, 3 × more side branch occlusion and 2 vs 0 cases with the decrease of TIMI-flow). The net clinical and angiographic benefit was zero.

Why did this sophisticated approach fail despite its theoretical advantages? The authors present several possible explanations in their discussion. The most probable is that this approach is pharmacologically too weak. There is insufficient drug concentration in the wall and even more important — after several hours this decreases to zero. Camenzind et al.[3] showed that only 1–8% of locally infused 99mTc-labelled heparin is locally detectable and that the mean retention time is 50 hours (ranging between 12–90 h). Clearly, even an ideal drug is at high risk for failure when it remains at the target site for only hours or days, while restenosis is taking weeks or even months to develop. The story of local intracoronary drug delivery may well resemble the story of intracoronary thrombolysis in acute myocardial infarction: it will help greatly to improve our knowledge, but it will be no more effective than systemic administration of the same drug.

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A second limitation may be that our mechanical methods (balloons with jets, leaks or even needles, coated stents, etc.) are too rough for the tiny endothelial and subendothelial tissues. Despite reports about the feasibility and safety of different local drug delivery devices \cite{4-7}, their clinical effectiveness has yet to be proved. Increased risk of arterial thrombosis after local delivery of some drugs is one major issue \cite{8}, but probably the most important limitation is the greater extent of tissue injury by local drug delivery devices. This additional injury may stimulate more neointimal hyperplasia and thus counterbalance the potential benefit of locally delivered drug.

The fight against restenosis is a 20-year vicious circle. Up to now, local intracoronary drug delivery seems not to have broken this circle. New, entirely different approaches — intrapericardial drug delivery \cite{9}, radioactive stents and intracoronary radiation therapy may help break this circle in near future.

**P. WIDIMSKÝ**

Cardiocenter University Hospital Vinohrady,

Prague, Czech Republic

References


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Collateral flow and restenosis: appreciating hydraulics and outcomes of percutaneous coronary intervention

See page 1776 for the article to which this Editorial refers

Wahl et al.\cite{1} have addressed the question whether patients with restenosis after angioplasty had higher collateral flow to the recipient vessel than patients without restenosis. Two hundred patients were examined and a collateral flow index was derived during balloon occlusion using aortic, distal coronary and central venous pressures. Sixty-four patients had angiographic follow-up 2 months later and were divided into two groups; 34 patients with and 30 patients without restenosis. Patients with restenosis had a higher collateral flow index at the time of coronary angioplasty than patients without restenosis. These data suggested that patients with restenosis after angioplasty had a more extensive collateral supply beforehand and that well-developed collaterals were a risk factor for restenosis.

This paper comes from a laboratory with a superior track record in coronary physiology and an established publication history for the coronary collateral flow velocity and pressure indices derived from patients undergoing intervention. The investigators are to be complimented for delving further into the haemodynamics of coronary responses to