

STENT PLACEMENT IN SAPHENOUS VEIN CORONARY BYPASS GRAFTS : ITS ROLE IN THE PREVENTION OF RESTENOSIS

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ABSTRACT

The relentless attrition of aortocoronary vein bypass grafts often means a repeat revascularisation procedure is necessary for relief of ischaemia. Repeat coronary artery bypass revascularisation, however, is often associated with a higher mortality and morbidity, is technically more demanding and often yields inferior long-term clinical improvement compared to the initial operation.

Balloon angioplasty for vein grafts is an established alternative revascularisation procedure but is hampered by an unacceptable restenosis rate, particularly for proximal and mid-graft lesions. Furthermore, it carries a significant risk of acute complication when applied to degenerated vein grafts. In contrast, there is considerable observational data documenting a lower risk of distal embolisation, a more favourable, smoother and wider lumen, and a lower risk of restenosis following stent implantation as compared to balloon angioplasty. These encouraging results must, however, await confirmation from prospective randomised trials comparing the 2 treatment strategies in a vein graft setting.

This review article focuses on the promising potentials of intracoronary stenting and provides an update of its role in reducing restenosis in a vein graft setting.

Keywords: saphenous vein grafts, stents, restenosis, balloon angioplasty, coronary atherosclerosis.

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INTRODUCTION

Conventional percutaneous transluminal coronary angioplasty (PTCA) in the treatment of saphenous vein graft stenosis is an established modality, having been shown to be highly effective in relieving ischaemia⁽¹⁾. In comparison with reoperation, PTCA is certainly more appealing as it entails less trauma. Furthermore, reoperation is associated with an enhanced risk of perioperative myocardial infarction, mortality and stroke⁽²⁾. In contrast to the primary operation, repeat bypass surgery is technically more complex and often results in incomplete revascularisation and less symptomatic benefits⁽²⁾. Hence, all efforts should be directed at avoiding another surgical revascularisation procedure. To this end, PTCA is an attractive alternative, particularly when the stenotic lesions are accessible and dilatable with this balloon-based technique.

Unfortunately, although PTCA largely yields excellent short-term results, it is, nevertheless, associated with 2 major drawbacks. First, when applied in the setting of degenerated vein grafts which often contain large friable or thrombus-laden plaques, PTCA may give rise to distal embolisation with resultant myocardial ischaemia, infarction or death⁽³⁻⁵⁾. This disastrous sequela may be averted in part with prolonged intracoronary thrombolytic therapy prior to dilatation^(6, 7) and stenting^(2, 8), by dissolving the thrombus and pressing the friable atheromatous material against the vessel wall respectively.

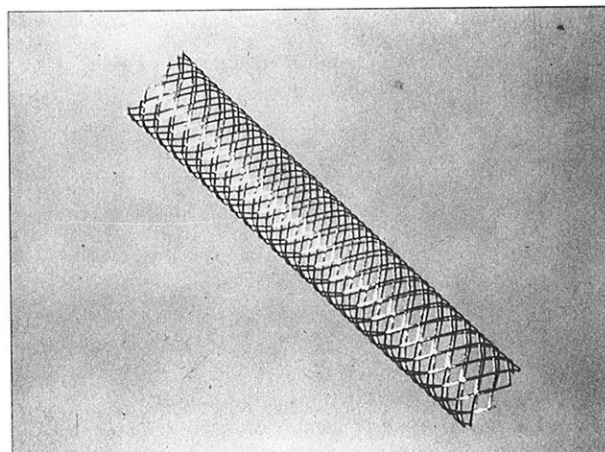
Second, the restenosis rates following PTCA for vein grafts are unacceptably high, especially for stenoses situated in the proximal and mid-graft segments where, on average, about 50% to 60% recurrence rates have been observed⁽²⁾;

rates that are considerably higher than those of native coronary arteries. Currently, no pharmacotherapy, either singly or in combination, has been convincingly shown to reduce restenosis⁽⁹⁾. Because of this major hindrance, various new coronary devices have been extensively tested to address this problem⁽⁹⁻¹¹⁾; one of which is intracoronary stent. The rest of this article will review the role of stents in preventing restenosis in a vein graft scenario.

INTRACORONARY STENTS

Four major stent designs are presently under investigation; the Wallstent (Schneider, Inc, Plymouth, MN), Palmaz-Schatz (Johnson & Johnson, Warren, USA), Gianturco-Roubin (Cook Inc, Bloomington, Indiana) and Wiktor (Medtronic Inc, Minneapolis, Minn., USA) stents (Fig 1 a,b,c & d). All are excellent bailout devices for acute or threatened closures

Fig 1a – Wallstent. A self-expanding mesh stent made of fine (0.06mm diameter) stainless steel filaments. Compared to the other stent models, the Wallstent has a wider range of length and diameter sizes, has more metal and undergoes more longitudinal shortening on deployment. There is, however, no recoil.



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Fig 1b – Palmaz-Schatz stent. A balloon-mounted slotted stainless steel design of two short tubes, each 7 mm in length connected by a 1 mm bridge. Although the articulation has improved its flexibility, the Palmaz-Schatz stent is, nevertheless, more rigid than the Wallstent, the Gianturco-Roubin and Wiktor stent.

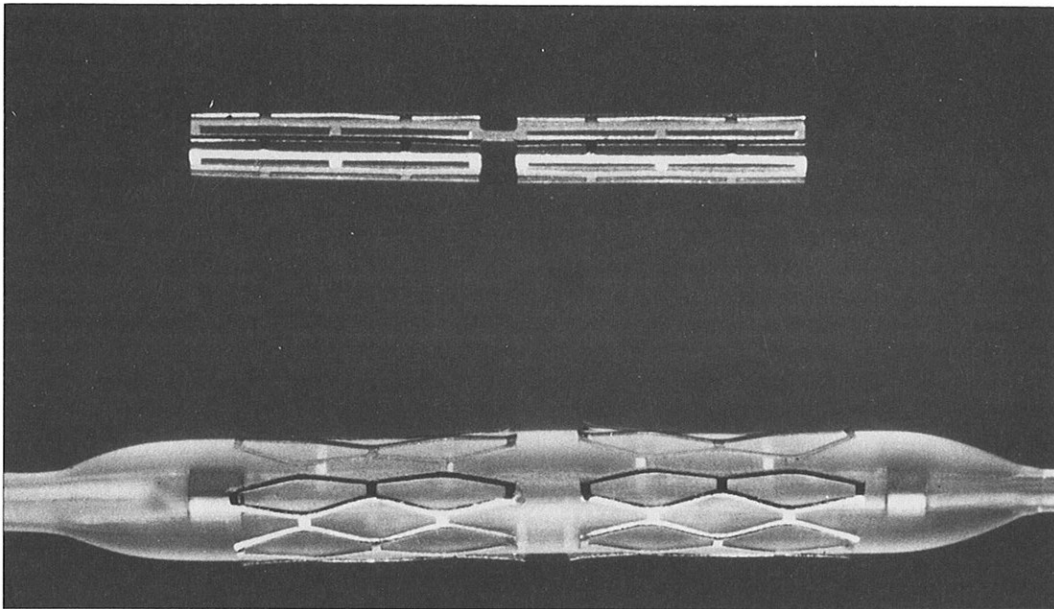
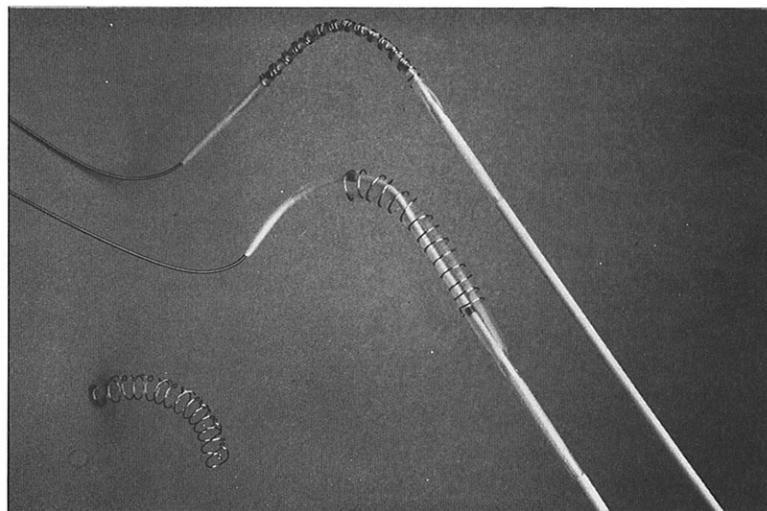


Fig 1c – Gianturco-Roubin stent. A balloon-mounted stainless steel coil stent which is relatively easy to handle and has a loose configuration. It tends to experience more radial recoil than the Wallstent and the Palmaz-Schatz stent.



following conventional balloon dilatation⁽¹⁰⁾. The most vein graft stenting experience accrued are with the first 3 stent models; there is as yet no long-term information available on the Wiktor stent in grafts.

Stenting proposes to prevent restenosis by the following mechanisms. Because PTCA achieves lumen improvement by vessel stretch, atheromatous plaque fracture and vessel wall disruption, thrombogenic subintimal tissues are exposed to the blood elements, thereby stimulating platelet activation, thrombus formation and ultimately, exuberant intimal proliferation from uncontrolled smooth muscle cell proliferation. Recent histologic studies have demonstrated intimal hyperplasia to be the pivotal mechanism of restenosis, regardless of the procedure involved⁽¹²⁾. Proper stent

deployment optimises luminal size by tacking up intimal tears, thus providing a relatively smooth lumen, good forward flow and reduces unfavourable rheologic factors (Fig 2a, b, c, and d); it reduces exposure of the deep wall components, and encourages orderly and limited thrombosis. Some recent studies have indeed demonstrated that suboptimal stent implantation, that is failure of the stent to adequately cover the balloon-traumatised arterial segment, is associated with both an increased risk of early thrombotic stent closure⁽¹³⁾ and a significant risk of restenosis⁽¹⁴⁾. Sigwart's group⁽¹⁴⁾ found that suboptimal stent deployment was the only risk determinant of Wallstent restenosis in their analysis of 15 angiographic, procedural and stent-related variables; all such deployed stents restenosed compared to a more optimistic

Fig 1d – Wiktor stent. A balloon-mounted flexible tantalum stent which undergoes some recoil on deployment. Its salutary advantage over the other stent models (a to c) is its high radioopacity, thereby enabling more precise placement.

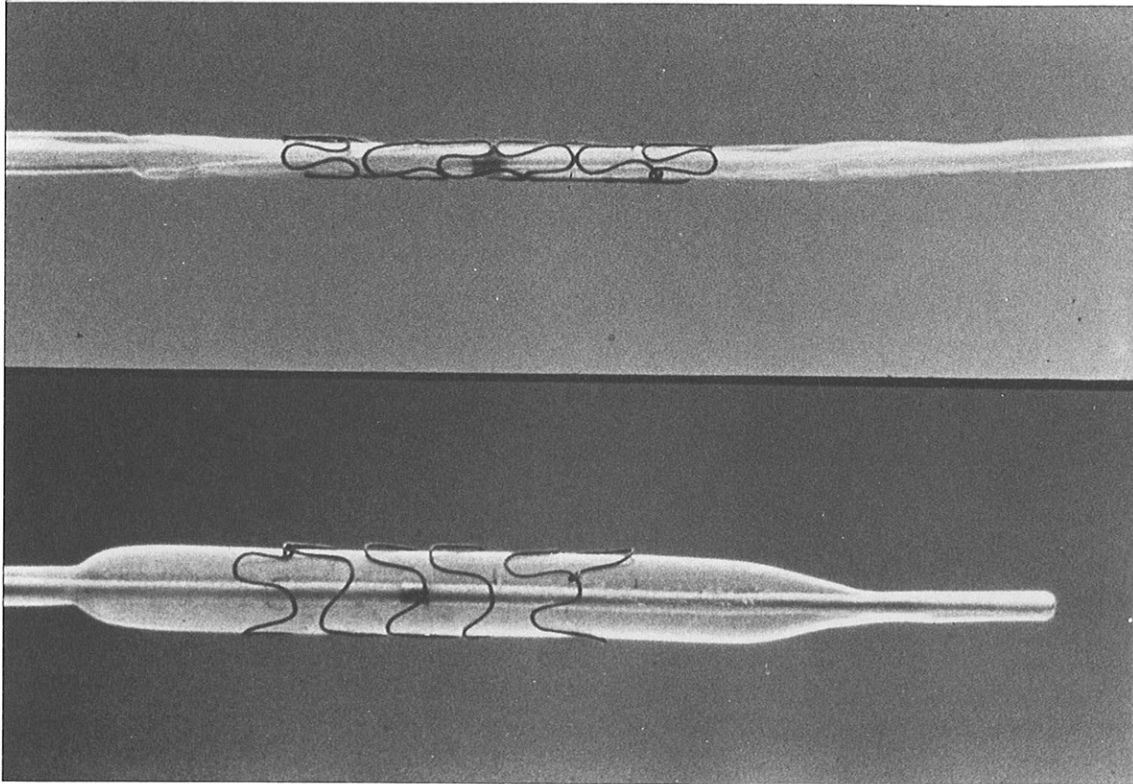


Fig 2a – Angiographic frame demonstrating a severely stenosed and complex lesion situated in the body of the vein graft before intervention.

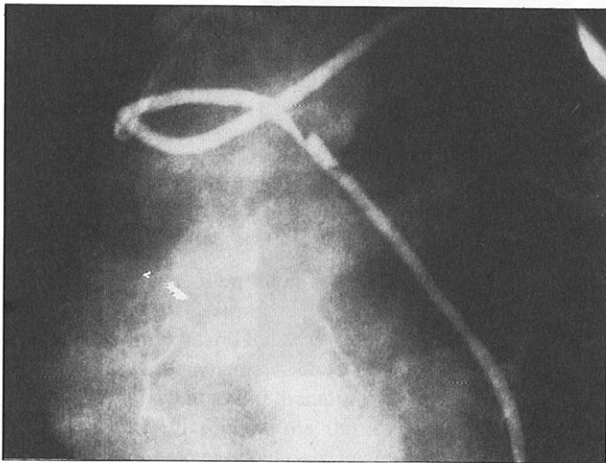
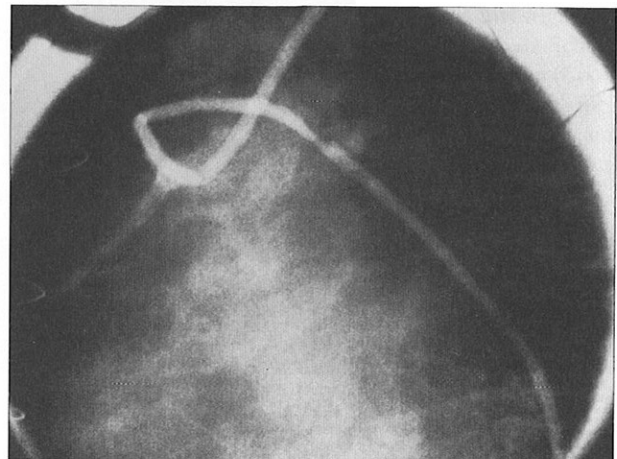


Fig 2b – Angiographic frame demonstrating a residual suboptimal outcome after conventional angioplasty using a balloon.



12% restenosis rate for optimally placed stents ($p=0.0004$).

Second, stenting frequently engenders quantitatively more superior results than PTCA by enlarging the arterial lumen maximally, preventing elastic recoil and scaffolding the lumen with its rigid lattice. Elastic recoil of up to 50% cross-sectional area occurs ubiquitously following PTCA⁽¹⁵⁻¹⁷⁾, being especially more pronounced for eccentric and noncalcified lesions⁽¹⁷⁾. This geometric limitation of PTCA has been implicated as a contributory factor in restenosis⁽¹²⁾. Recently, a number of studies have identified a strong inverse relationship between restenosis, immediate postprocedural luminal diameter and reference vessel size⁽¹⁸⁻²¹⁾, independent of the device used⁽¹⁸⁾, the so-called “bigger is better” concept.

When compared to other interventional devices such as PTCA (which on average leaves behind a 30% residual diameter stenosis)^(18, 20, 21) and laser balloon angioplasty⁽²⁰⁾, the immediate luminal gain attained by stenting was significantly larger, often resulting in a luminal diameter equal to or even greater than that of the reference vessel. Stenting, similarly attains equal or larger luminal outcome compared to directional atherectomy⁽¹⁸⁻²¹⁾. The second generation intracoronary devices, including stents, however, appear to stimulate more intimal hyperplasia, which in turn, is reflected in more late loss in luminal diameter. The greater absolute immediate gain afforded by stenting, fortunately, often more than offsets and compensates for the greater late loss; the

Fig 2c – Angiographic frame demonstrating a smooth and widely patent lumen after implantation of a Palmaz-Schatz stent.

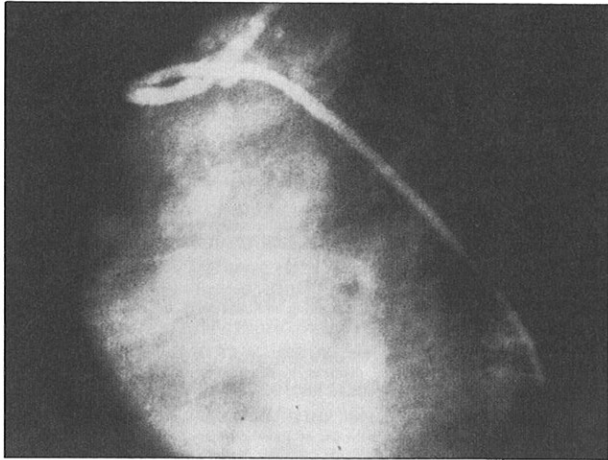
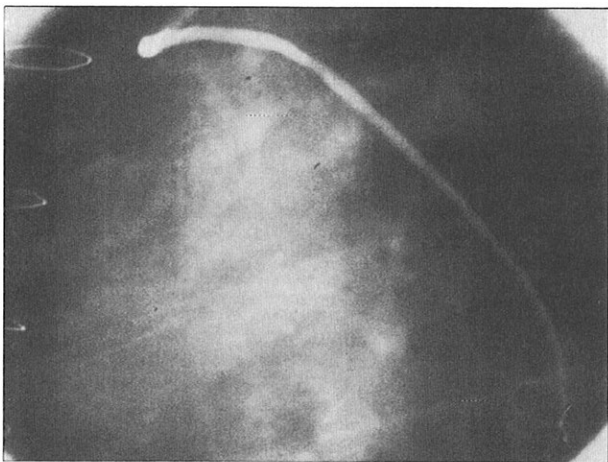


Fig 2d – Angiographic frame demonstrating some insignificant narrowing but no restenosis of the stented segment at 6-month angiographic follow-up.



restenosis risk is thus reduced. And because most vein grafts are large, generally more than native coronary arteries, low restenosis rate may potentially be obtained by proper stenting. The larger luminal size of vein grafts in addition, also imparts a lower risk of early stent thrombosis^(22,23). Thus, stent placements in vein grafts appear promising.

Lau et al⁽¹⁴⁾ reported a restenosis rate of 25% (vs 16% for native coronary arteries, $p=0.5$) for the Wallstent despite the inclusion of a large number of patients with unfavourable lesion morphologies; a result not dissimilar to those published earlier by Urban⁽²⁴⁾, Strauss and co-workers⁽²⁵⁾. Scheerder et al⁽⁸⁾, on the other hand, noted a much higher restenosis rate of 47%; this perhaps could be due to their selection of unfavourable lesions and patients who were poor candidates for bypass surgery. The restenosis rate for the Palmaz-Schatz stent in vein grafts have also been documented to be around 25%^(22,26). A single study by Bilodeau et al⁽²⁷⁾ on the Gianturco-Roubin stents in vein grafts found an overall restenosis rate of 35%. True comparison between the various stents and between stents and PTCA in vein grafts is not possible as all the aforementioned studies currently available are observational in nature. Hence, although preliminary data to date suggest that stent placement in vein grafts is associated

with a lower propensity of lesional recurrence compared to PTCA, this observation should be confirmed by large randomised trials which are currently in progress. Some early data on a randomised trial comparing the Palmaz-Schatz stent and PTCA in de novo native coronary arteries appear to favour stent placement in terms of a lower restenosis rate (18% for stents vs 31% for PTCA; preliminary data from the Benestent Study, presented during the PTCA III course, Singapore General Hospital, July 1993).

CONCLUSION

The central goal in any intracoronary intervention must be to maximise the acute gain in luminal diameter without jeopardising the arterial integrity and the safety of the patient. At the end of the procedure, ideally, the interventionist should strive to leave behind a smooth and widely patent lumen that approximates the reference size of the artery. By doing so, it is hoped the restenosis rate may be reduced. Stenting appears to be able to reduce restenosis by (1) providing a smooth and large lumen, thereby, attenuating adverse rheologic factors that promote intimal hyperplasia, and (2) allowing the lumen to accommodate more late loss before the onset of a haemodynamically significant lesion. However, stents should not be excessively oversized and attempts should be made not to overlap multiple stents as these factors have been shown to be markers of stent restenosis^(25, 28).

Certainly, there is no question about its efficacy as a bailout device in suitable lesions with acute or threatened closure, either as a permanent strategy or as a bridge to surgery. In such circumstance, the immediate aim is to decrease myocardial injury and circumvent emergent bypass surgery, and not the prevention of restenosis. In contrast, the current indications for elective stenting of grafts to prevent restenosis are not so clear-cut although the relative ease of implanting stents and the high success rate (> 95%) is definitely advantageous (and may also provide the impetus for indiscrete and unjustifiable routine application of stents). Of major concern is the 2% to 8% risk of early stent thrombosis, albeit lower in vein conduits than native coronary arteries^(23, 29), and its serious clinical consequences. Hence, until such time when stent thrombosis can be effectively averted and more data become available from prospective randomised trials comparing the various intracoronary interventional devices, perhaps the current clinical utility of elective stenting for the prevention of restenosis should be more selective and targeted at specific adverse graft lesion morphologies and circumstances where PTCA is expected to yield inferior early and long-term results. Potential lesional niches include: (1) suboptimal luminal outcome after PTCA and recurrent restenosis (after multiple previous balloon angioplasties) where the restenosis rate after conventional PTCA is considerable, and (2) complex (ulcerated or degenerated) lesions where the risk of acute complication and restenosis associated with stand-alone PTCA is high⁽¹²⁾. Importantly, the graft lesion selected for elective stenting should also have a large reference size (and thus, larger stent size), be optimally dilated and adequately covered in order to reduce the risk of stent thrombosis and restenosis^(2, 13, 14, 22, 28, 30, 31).

The other caveat of stenting is that of vascular access site complications⁽¹⁰⁾. The latter, the result of the stringent regimes in antithrombotic-antiplatelet therapy mandatory in the immediate poststent period, however, do not appear to be a major issue and are manageable with the application of various haemostatic devices such as collagen plugs and groin

compressors, and appropriate adherence to careful anticoagulation titration and a graduated mobilisation protocol^(13, 32).

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ADDENDUM

Since the writing of this article, 2 moderate-sized randomised trials comparing PTCA and elective single Palmaz-Schatz stent implantation in de novo native coronary arteries have been published. In the Benestent trial (N Engl J Med 1994; 331: 489-95) which enrolled 520 patients, the 6-month restenosis rate (based on the binary criteria) was significantly lower after stenting (22% vs 32%, $p=0.02$). Similar results were obtained in the STRESS trial (N Engl J Med 1994; 331: 496-501); the 6-month restenosis for the stent group was 30% compared with 43% in the PTCA group ($p=0.016$). This reduced restenosis rate was translated into a superior long-term outcome after stent implantation, mainly in terms of the need for repeat PTCA for restenosis-induced ischaemia.

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