STRUCTURAL DESIGN, CLINICAL EXPERIENCE, AND CURRENT INDICATIONS OF THE CORONARY WALLSTENT

David Keane, MB, Peter de Jaegere, MD, and Patrick W. Serruys, MD

STRUCTURAL DESIGN

The Wallstent (Schneider, Bülach, Switzerland) has a stainless steel tubular mesh design with longitudinal flexibility. A total of 18 to 20 monofilaments 0.07- to 0.10-mm thick are used to fabricate each coronary Wallstent, resulting in a metallic surface area in the expanded state of approximately 20%[6] and a metallic cross-sectional area 0.062 mm^2.[15] This relatively high metallic surface area (compared with 8% for the new advanced cardiovascular system [ACS] stent) may be of theoretical importance as it appears likely that stents of lower metallic surface area are less thrombogenic and cause less inflammatory response in the vessel wall. The intersections of the filaments of the mesh structure are not fixed or soldered together, thereby affording greater longitudinal flexibility in comparison with that of the Palmaz-Schatz mesh design. Vessel splinting is, thus, not prominent in either native or vein graft coronary vessels following Wallstent implantation. Early concerns of friction from metal surfaces rubbing together at points of filament intersection appear to be unwarranted, and signs of such complications have not emerged from clinical studies.

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The stainless steel alloy is cobalt-based, and energy dispersion spectrometric studies have identified elements of chromium, iron, nickel, and molybdenum in addition to cobalt,[15] thus indicating that the Wallstent consists of a more heterogenous alloy than many other metallic coronary stents. The radiopacity of the Wallstent during conventional cineangiography is relatively low (compared with that of the tantalum Wiktor and Strecker stents); however, with advances in digital pulsed fluoroscopy, the Wallstent mesh can usually be identified (at 50 kV) even after removal of the radiopaque markers of the delivery system.[15] Unlike some other stainless steel stents (e.g., Palmaz stent) but similar to tantalum stents (e.g. Strecker stent), the Wallstent has been found not to move or deflect during MR imaging, even under high magnetic field strengths.[13, 19] However, as stents become incorporated into the vessel wall after several weeks, MR imaging of patients with ferromagnetic stents is probably safe after a suitable period has elapsed to ensure stable positioning of the device.[13]

The Wallstent is self-expanding and, therefore, does not require a balloon for deployment. This may be particularly advantageous in friable, diffusely diseased bypass grafts at high risk for embolization with balloon angioplasty. Instead, the Wallstent is mounted on a

From the Department of Interventional Cardiology, Thoraxcenter, Erasmus University, Rotterdam, The Netherlands

low-profile catheter and is maintained in a collapsed state until it is progressively released by the retraction of its rolling membrane (Fig. 1). The doubled-over restraining membrane and delivery mechanism prevent accidental release of the stent, and a partially deployed Wallstent can often be withdrawn proximally or removed entirely.[6]

Dilatation of a balloon within the Wallstent is normally performed after its release to accelerate early expansion, to ensure symmetrical and adequate apposition of the stent against the vessel wall, and to dissipate thrombus within the stent (Fig. 2). The Wallstent is thought to continue to expand until an equilibrium is reached between the elastic recoil of the vessel wall and the radial force of the stent.[1]

The low profile of the Wallstent compares favorably with that of stents which require balloon mounting. The outer diameter of the coronary stent–mounted delivery catheter of the Wallstent is 1.57 mm (Schneider, Bulach, Switzerland) in comparison with 1.65 mm for the preballoon-mounted Palmaz-Schatz mesh stent (Johnson & Johnson, Paris, France) and 2.13 mm for the balloon-mounted Gianturco-Roubin coil stent (Cook, Bloomington, IN). This feature increases the versatility of the Wallstent and, coupled with its longitudinal flexibility, results in its aptitude to negotiate proximal vessel tortuosities and points of small luminal diameter. Furthermore, its low profile permits introduction of the Wallstent through an 8-F guiding catheter (0.077-inch internal diameter), and, thus, exchange of the femoral sheath and guiding catheter during the bailout management of an acute occlusive

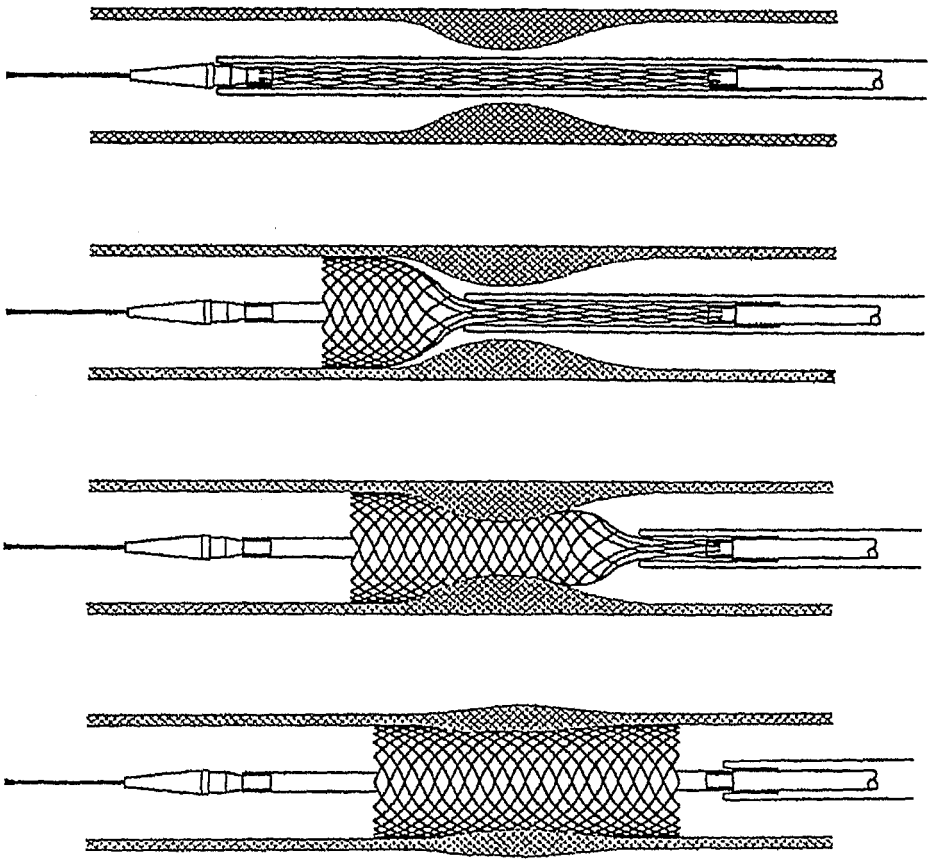

Figure 1. Deployment of the Wallstent requires the advancement of the Wallstent over a 0.014-in or 0.018-in guidewire across the lesion. The distal marker band should be positioned slightly beyond the distal edge of the stenosis to account for distal shortening of the stent during expansion. The central and proximal markers approximately define the final position of the stent. The rolling membrane is inflated with contrast medium to 4 atmospheres prior to its gradual retraction to facilitate the smooth and progressive release of the self-expanding stent.

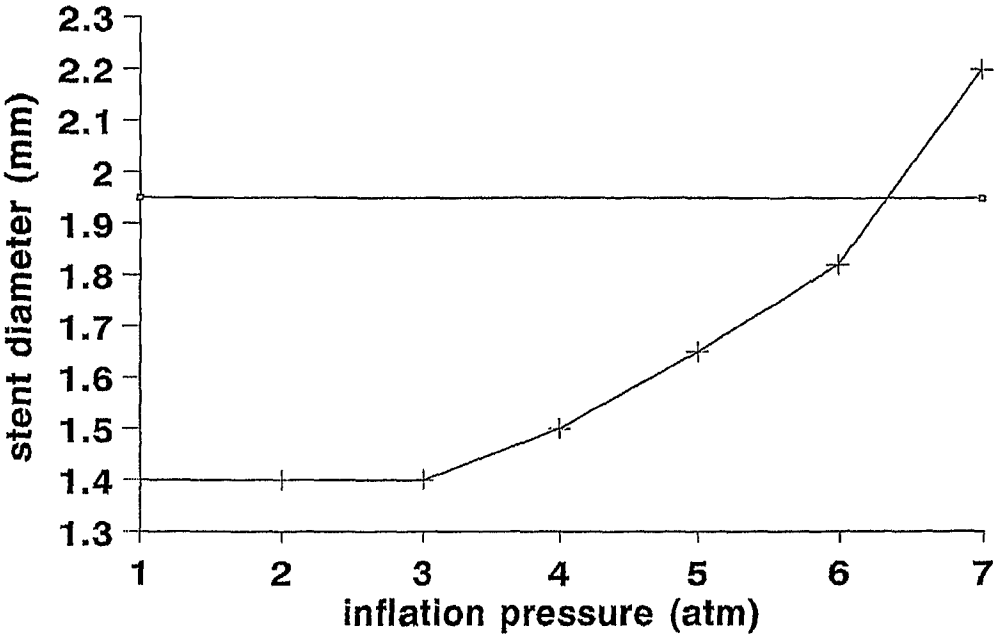

Figure 2. A balloon dilatation is normally performed within the Wallstent after its release to optimize deployment. This plot of inflation pressure against the diameter of the Wallstent in vitro indicates that inflation pressures should be kept under 4.5 atmospheres to avoid significant alteration of the mesh configuration of the expanded Wallstent.

dissection with the Wallstent is rarely necessary. This compares favorably with the Gianturco-Roubin coil stent for which, normally, a 9-F guiding catheter (internal diameter 0.089 inches) is required for introduction of a Flexstent larger than 3.0 mm [this undesirable exchange of guiding catheters and sheaths during bailout management might be overcome by the recent development of the Lumax 8-F guiding catheter (Cook), which has a relatively large internal lumen of 0.086 inches and allows delivery of the 4.0-mm Flexstent stat].

An additional feature of the Wallstent is the wide range of sizes available for coronary vessels, ranging from 2.5 to 6.0 mm in diameter in the expanded state with lengths of 15 to 30 mm. This range of diameters is adequate for two reasons. First, stenting of vessels 2.5 mm or less in diameter appears to carry an unacceptable risk of subacute thrombosis, and a Wallstent size of 0.5 mm greater than the vessel size is required for optimal results to ensure that sufficient radial force is exerted on the vessel wall to prevent stent migration and to overcome elastic recoil. Second, few vessels are of greater diameter than 5.5 mm, even among coronary vein grafts. Wallstents of greater than 6.0 mm in diameter are available, but they are usually deployed in peripheral vessels. The large range of lengths available for the Wallstent may be particularly advantageous. It is helpful for the treatment of long dissections and to avoid the deployment of more than one stent for long dissections, a factor known to increase the risk for early thrombosis[3] and subsequent late restenosis (particularly at the site of stent overlap where the metallic burden on the vessel wall is highest.[16]) Also, the large range of lengths permits the selection of a Wallstent of greater length than the lesion, thus reducing the risk of failing to cover the entire lesion length.

The length of the Wallstent may shorten significantly upon expansion. This may result in a failure to cover a lesion and necessitate the deployment of a telescoping second Wallstent to cover a stenosis or dissection. To overcome the difficulty of unpredictable longitudinal shortening following radial expansion, the design of the coronary Wallstent has recently been modified to produce a device which is less prone to shortening (the less shortening Wallstent). This is achieved at a cost of some loss of radial strength. Its technical features are depicted in Figure 3. The

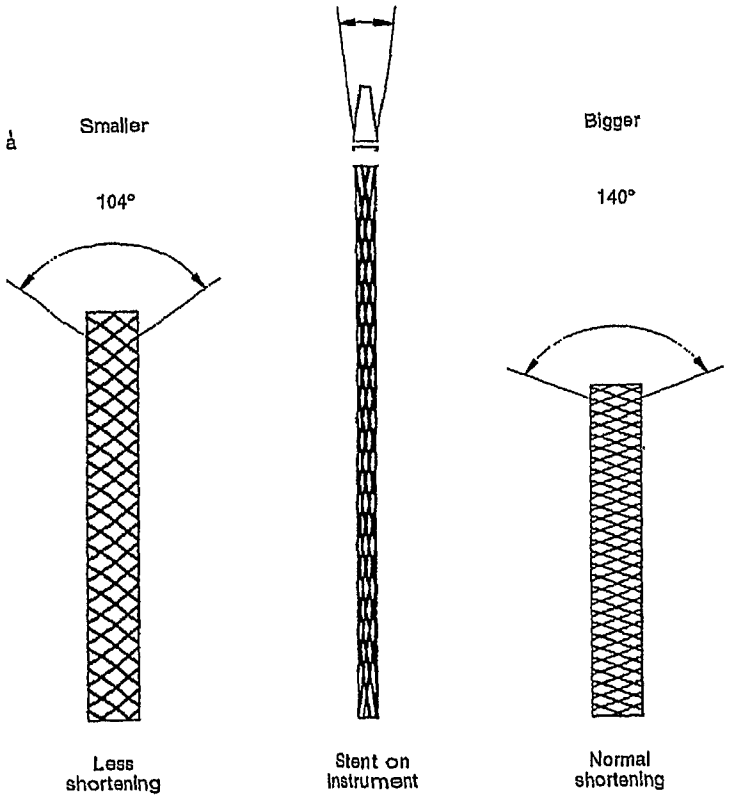

Figure 3. To overcome the problem of marked shortening of the Wallstent upon expansion, the design of the mesh has been modified to produce the Less Shortening Wallstent. The braiding angle of the mesh has been been reduced from 140° to 104°. This is associated with an overall lower density of metal struts per unit length but carries a cost of lower radial support.

less shortening Wallstent undergoes approximately 25% shortening upon expansion, and the braiding angle of its mesh is 104 degrees in comparison with 140 degrees for the normal shortening Wallstent. The follow-up phase of the first clinical study to evaluate this modified design has just been completed. All previously reported experience with the Wallstent has involved the earlier normal shortening design.

EXPERIMENTAL STUDIES OF THROMBOGENICITY AND POLYMERIC COATING

The primary reservation concerning clinical use of the Wallstent has been a relatively high incidence of acute thrombosis. This most likely relates to its high stent-to-vessel surface area. It is unlikely that the stainless steel alloy of Wallstents is more thrombogenic than the composition of other metallic stents.

Among the stainless steel alloy stents, differences in thrombogenicity have not been found. In an exteriorized arteriovenous silicone shunt model in baboons, the thrombogenicity of the chromium-free stainless steel Palmaz-Schatz stent was compared with that of the chromium-based stainless steel Wallstent. The Palmaz-Schatz stent accumulated $4.37 \pm 0.68 \times 10^9$ platelets/cm, which was not statistically different from the Wallstent, which accumulated $3.91 \pm 0.42 \times 10^9$ platelets/cm.[8]

Although no thrombotic occlusions were observed within 4 weeks in a noncomparative study of a tantalum stent (Wiktor) in the coronary arteries of 10 pigs,[20] evidence for lower thrombogenicity of tantalum in comparison with stainless steel stents has not been found in comparative in vitro and in vivo studies despite theoretical differences in ionic charge between tantalum and stainless steel. In an in vitro study by Hearn et al,[5] no significant difference in thrombogenicity was found between noncoated tantalum and stainless steel stents exposed to human blood. Further work from Emory University in both extracorporeal baboon and intracoronary porcine models has confirmed that stainless steel stents are not more thrombogenic than tantalum stents.[14n]

In an effort to reduce the incidence of stent thrombosis, the surface of the Wallstent was

modified in 1989 to incorporate a polymer coating (BioGold). In vivo research at the Thoraxcenter has indicated that this polymeric coating may reduce the thrombogenicity of the Wallstent.[21] In this comparative study in porcine coronary arteries, thrombogenicity (by angiography) at 1, 4, and 12 weeks and vessel wall histology at 12 weeks were studied in 15 polymer-coated Wallstents placed with no medication and 48 uncoated Wallstents placed with the administration of acenocoumarol (16 stents), aspirin (16 stents), and no medication (16 stents). A 38% thrombotic occlusion rate within 1 week (uncoated Wallstents with no medication) was prevented by polymeric coating and by acenocoumarol but not by aspirin. Histologic analysis showed no difference in the thickness of the neointimal hyperplasia between the groups, and the BioGold polymer did not induce an inflammatory reaction. This experimental histologic finding is supported by the retrospective angiographic comparison of those patients who received an uncoated Wallstent before August 1989 versus those patients in whom a polymeric-coated stent was implanted after August 1989, in which the BioGold coating was found to have no significant relationship on late restenosis.[16] The efficacy of this coating in the reduction of subacute thrombosis, however, has yet to be demonstrated clinically.

EARLY CLINICAL EXPERIENCE WITH THE WALLSTENT

Valuable clinical experience with coronary stenting was gained from a six-center European study of the Wallstent which was undertaken in the 1980s.[12, 14] Between March 1986 and January 1988, 117 Wallstents were implanted in the native coronary arteries (94 stents) or saphenous vein bypass grafts (23 stents) of 105 patients. Seventy-one stents were implanted after dilatation of a restenotic lesion, 14 were placed as bailout therapy for an acute occlusion following balloon angioplasty, 5 were deployed after angioplasty for chronic occlusion, and 27 were included as an adjunct procedure to primary balloon angioplasty.

It is not surprising that this early experience was fraught with the controversy of anticoagulant management. Regimens ranged from the use of subcutaneous heparin to routine intracoronary urokinase. Many of the initial patients in this early experience were not started on an oral anticoagulant regimen post-stenting, and a few patients did not receive aspirin, whereas others were maintained on coumadin, aspirin, dipyridamole, and sulfinpyrazole. Even today, 8 years later, the strategies proposed for anticoagulant therapy post-stenting continue to differ widely without international consensus.[7]

The overall mortality rate for the group at 1 year was 7.6% (eight deaths). Of these deaths, six were most likely the result of stent occlusion. Stent occlusion was angiographically confirmed at implantation in two of the six patients and after 24 hours in one patient, whereas in the other three, sudden death occurred at 48 hours, 11 days, and 6 weeks, respectively, after stent implantation.

Quantitative angiography (cardiovascular angiography analysis system [CAAS]) revealed that, overall, the minimum luminal diameter improved from 1.21 ± 0.56 mm to 1.88 ± 0.43 mm after balloon angioplasty and then further to 2.48 ± 0.51 mm immediately after stent implantation. Angiographic follow-up was obtained in 90% of patients at a mean of 5.7 ± 4.4 months. At follow-up, the diameter for the whole group (95 patients) had decreased to 1.68 ± 1.20 mm. The validity of including or excluding total occlusions from an angiographic follow-up analysis of restenosis following balloon angioplasty is unclear in view of the unconfirmed mechanisms of total occlusion.[7, 22] With coronary stenting, it seems more likely that total occlusions may represent a different phenomenon than typical restenosis in view of the frequently documented occurrence of thrombotic occlusions in the early period following stent implantation. When the total occlusions (27 stents in 25 patients) were removed from the angiographic follow-up group, the minimal luminal diameter at follow-up was 2.26 ± 0.78 mm. Ten stents in nine patients of this group and one segment proximal to a stent had a diameter stenosis of more than 50% at follow-up for a total restenosis rate of 14% of patients in whom the Wallstent was patent at follow-up.

LATER CLINICAL EXPERIENCE WITH THE WALLSTENT

The previously described results from the early experience, which was pioneering not only for the Wallstent but also for all coronary stenting, were compared with the subsequent

results of the period from February 1988 to March 1990.[16, 17] In addition, the cumulative clinical experience gained from 265 patients (308 lesions) from March 1986 to March 1990 was analyzed according to whether the Wallstent was implanted in a native coronary artery or bypass vein graft (Fig. 4).[16, 17] The selection of cases during the early and late periods differed significantly, with a higher proportion of bypass vein grafts and primary stenoses in the later period (1988 to 1990).

Early occlusions following Wallstent implantation were less frequent in later years (10% of stents) than in the early experience (18% of stents). This may relate to the adoption of a more effective and standardized anticoagulant regimen, increased operator experience, and refined patient/lesion selection in the later years. In the combined early and later experience, early occlusions were documented in 18% of native coronary arteries compared with 7% of bypass vein grafts. The lower incidence of thrombotic occlusion in vein grafts may reflect, in part, that most of these vein graft stents were implanted during the later period when the anticoagulation period was more aggressive and, in part, the greater vessel size (recent studies indicate that thrombotic occlusion is less likely to occur in vessels larger than 3.25 mm compared with vessels smaller than 3.25 mm[3]).

Quantitative angiographic follow-up was obtained in 82% of all patients. The outcomes for native and bypass vessels are compared in Table 1. It has been proposed that the higher late restenosis rate in vein grafts may be the price for lower early occlusion rates.[17] By diminishing the formation of early occlusive thrombus with more effective anticoagulation, the residual nonoccluding thrombus could form the substrate for late restenosis.[17] This hypothesis is not supported by a more recent observation that the presence of thrombus at the time of balloon angioplasty is not associated with greater luminal loss at follow-up if total occlusions at follow-up are excluded from the analysis.[22]

To gain a perspective of these relative results of the Wallstent in bypass vein grafts and native coronary arteries, it is worth, for comparative purposes, to consider the results of a provisional report of early ischemic events in bypass vein grafts and coronary arteries following implantation of the Gian-

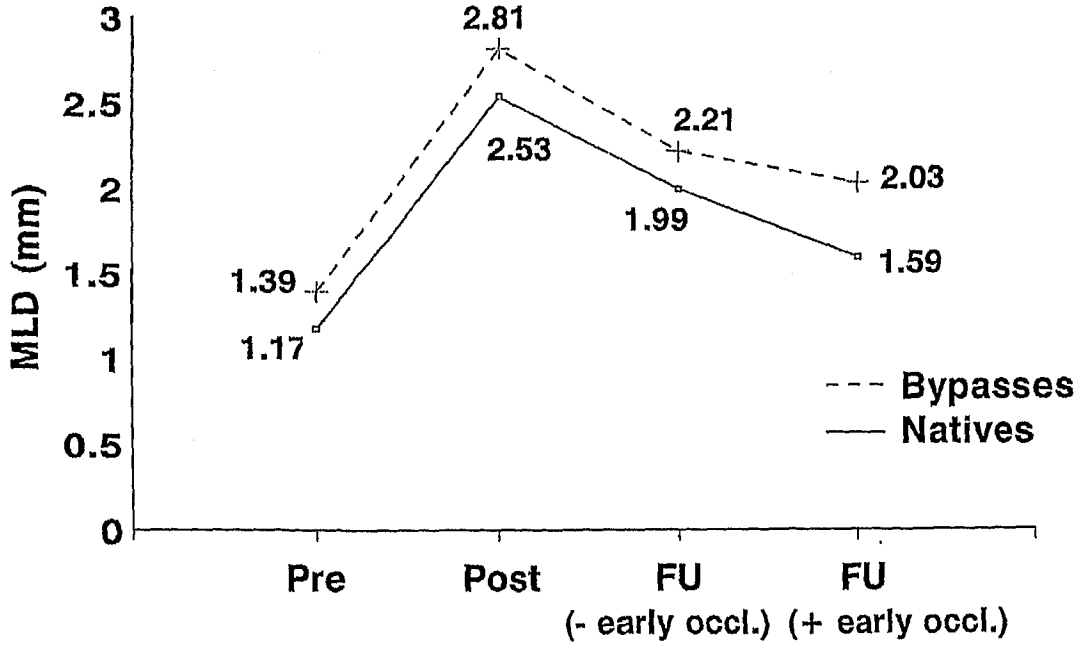

Figure 4. Minimal luminal diameter (MLD) of native coronary arteries and bypass vein grafts preprocedure, postprocedure and at follow up. The mean values have been calculated with and without the inclusion of the early in-hospital occlusions. (*From* Strauss BH, Serruys PW, Bertrand ME, et al: Quantitative angiographic follow-up of the coronary wallstent in native vessels and bypass grafts (European experience—March 1986 to March 1990). Am J Cardiol 69:475–481, 1992; with permission.)

Table 1. EARLY AND LATE RESULTS FOLLOWING WALLSTENT IMPLANTATION*

Vessel	Percent Early Occlusion	Percent Restenosis
Native coronary artery	19	39
Bypass vein graft	8	18

*Although the early occlusion rate for vein grafts was lower than that for native coronary arteries, the restenosis rate (>50% diameter stenosis) per patient was higher in bypass vein grafts than in native coronary arteries. There was no difference in the late occlusion rate between the two vessel types (16 lesions.)[17]

turco-Roubin coil stent.[18] Periprocedural (<24 hours) ischemic events occurred in 2% of vein grafts compared within 8% of native arteries ($P=0.07$), and the rate of later ischemic events at 1 to 90 days was at least as favorable for vein grafts as it was for native arteries.[18] Interpretation of these results, however, is confounded by differing clinical indications and lesional characteristics between the two vessel types and by the subsequent greater requirement for the deployment of multiple stents in bypass vein grafts.

CLINICAL EVALUATION OF THE LESS SHORTENING WALLSTENT

One of the factors thought to have contributed to the high rate of stent occlusion in the European registry of the coronary Wallstent in the 1980s was the frequent deployment of more than one stent to cover the target lesion. This factor attributed to the high degree of shortening of the Wallstent upon expansion. To overcome this limitation, the design of the Wallstent was modified to reduce the degree of shortening with the development of the Less Shortening Wallstent. A small study was undertaken to determine the safety and efficacy of implanting this new Wallstent for the management of saphenous vein coronary bypass graft stenoses. Between November 1991 and March 1993, the Less Shortening Wallstent was implanted in 29 patients at 6 centers in the Netherlands, Germany, and Belgium.[6n]

Thirty-five Wallstents were electively deployed in aorto-coronary vein grafts in 29 patients. Stent deployment was successful in 35 of 36 attempts in 30 lesions. In 5 of the 30 lesions, a second stent was required to cover the proximal portion of the lesion. Angiographic success (< 50% residual diameter stenosis as determined by offline quantitative coronary angiography) was achieved in all 29 patients. During the in-hospital phase, no major adverse cardiac event occurred (reintervention, re-CABG, myocardial infarction, or death), and 5 patients had hemorrhagic complications. Following hospital discharge, one patient had a subacute stent occlusion associated with symptoms and elevated cardiac enzymes at 11 days, another patient had symptoms and elevated cardiac enzymes (CK 300 U/l) at 22 days with a patent stent, five patients required balloon angioplasty within the 6 month follow-up period (four for restenosis and one for stent occlusion), and one patient underwent re-CABG for a native artery stenosis distal to the anastmosis of the patent stented vein graft. The results of this introductory study suggest that the new Less Shortening Wallstent may be associated with a reduction in the requirement for multiple stent deployment and a lower rate of thrombotic occlusion in comparison to its pioneering prototype. On the basis of these results, a number of larger multicenter clinical trials have been initiated to further evaluate the new coronary Wallstent.

CURRENT INDICATIONS

Based on the data described previously the Wallstent is a versatile stent which, by virtue of its longitudinal flexibility and low profile, can be deployed with a high degree of success in complex lesions of both native coronary arteries and bypass vein grafts.

Despite strict clinical indications that have been proved by prospective randomized trials, it could be proposed that there are, as yet, no absolute indications for coronary stenting. Indeed, traditional balloon angioplasty itself, the gold standard with which stenting frequently is compared, 17 years after its introduction[1] has yet to establish unequivocally its current indications in randomized, comparative morbidity and mortality trials in preference to medical therapy and bypass surgery.[2,9–11]

Thus, it may be more appropriate in this relatively early era of coronary stent evolution to refer to clinical applications and contraindications. Fewer patients are required and randomization is not essential to detect contraindications to coronary stenting, whereas absolute indications for coronary stenting are more difficult to establish. Currently, these contraindications include persistent intracoronary thrombus (after intracoronary administration of thrombolytic therapy

and, when necessary, confirmation with angioscopy) and contraindications to anticoagulant therapy. Relative contraindications include a vessel size of less than 3.0 mm, excessive proximal tortuosity where introduction of the stent would be unlikely to succeed, deployment of a stent in an unprotected left main coronary artery, and the absence of in-house cardiac surgical back-up.

It is unclear if an indication is established by a large randomized trial for one stent, whether the same results can be expected from a stent of similar design. In view of the relatively early stage of the clinical evaluation (8 years) of coronary stenting and the fundamental differences in metal surface area, strut composition (with or without coating), stent profile, delivery system, expansile mechanism, longitudinal flexibility, radial strength, and design among mesh stents (Wallstent, Palmaz-Schatz, and Strecker) and among coil stents (Wiktor, Gianturco-Roubin, and ACS), it seems that, for at least the present, each stent will have to prove its efficacy and safety for each clinical indication. Furthermore, although the coating of currently available stents continues to necessitate systemic anticoagulant therapy, the absolute indications for coronary stenting are likely to remain limited.

The clinical conditions in which the coronary Wallstent has been applied (usually as an adjunct to balloon angioplasty) during observational studies include the elective treatment of primary stenoses, restenoses, and chronic occlusions in native coronary arteries, the elective treatment of primary stenoses in vein grafts, and the bailout management of acute vessel closure following coronary intervention. By virtue of the high early thrombotic rate in native vessels and the high restenosis rate in vein grafts, it seems that, currently, the only condition in which implantation of a coronary Wallstent can be justified outside of a randomized trial are the bailout management of acute nonthrombotic occlusion (dissection) following interventional procedures in native vessels of 2.5 mm or greater diameter and, possibly, the elective treatment of bypass vein grafts with friable lesions. In light of its unique mechanical properties, the Wallstent might be expected to excel over stents of different design in long lesions in large-diameter vessels associated with proximal tortuosity.

Upcoming prospective randomized trials with the new less shortening Wallstent should determine whether the Wallstent has been a victim of the pioneering era of clinical evaluation during the early learning phase of coronary stenting associated with inadequate anticoagulation regimens, inappropriate patient selection, and multiple stent placement (on account of stent shortening), or whether exposure of the coronary artery to the relatively large stent surface area of the Wallstent presents an unacceptably high early thrombogenic burden or, possibly, late excessive stimulus to vessel wall hyperplasia in bypass vein grafts.

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Address reprint requests to

Patrick W. Serruys, MD, PhD
Dept Interventional Cardiology
Thoraxcenter, Ee 2332
Erasmus University
PO Box 1738
300 DR Rotterdam
The Netherlands