# THE CURRENT STATUS OF INTRACORONARY STENT: AN OVERVIEW

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*ABSTRACT*

***Intracoronary stenting is a relatively new therapeutic interventional modality conceived to address the 2 major pitfalls of conventional balloon angioplasty, namely that of acute closure and restenosis. Much progress has been made since its clinical application in 1986. Its place amongst the multitude of novel devices currently available in the treatment of coronary obstructive disease seems secure. The challenge for investigators now is to develop a stent without the inherent problem of acute thrombosis.***

*Keywords : Stent, angioplasty, acute closure, restenosis*

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## INTRODUCTION

Percutaneous Transluminal Coronary Angioplasty (PTCA) since its introduction by Andreas Gruentzig in 1977[1], has revolutionised cardiology, opening up a whole new vista of interventional therapeutic cardiology. It is now a firmly established modality in the treatment of obstructive coronary artery disease, offering an alternative to bypass surgery. Experienced operators and further refinement in angioplasty technology have enhanced its success rate and decreased its major complication rate despite the inclusion of more high risk lesions and patients[2,3]. However, PTCA-related acute closures and restenosis have remained blemishes in the short- and long-term follow-up results of PTCA. In an attempt to circumvent these problems and improve the results of PTCA, at the same time maintaining a percutaneous approach, 3 broad categories of techniques have been developed and tested in clinical practice.

1. Plaque removal: Atherectomy and laser vapourisation.
2. Welding: Laser balloon angioplasty (LBA)
3. Scaffolding: Intracoronary stent

The present review will focus on the current status of intracoronary stenting.

## INDICATIONS FOR STENTING

### A. Post-PTCA acute closure

Approximately 5% of PTCA procedures ends up with acute closure. Although a large number of predisposing risk factors of acute closure have been described eg complex lesions (type B and C lesions), presence of intracoronary thrombus, major dissection, insufficient antiplatelet therapy and inadequate intraprocedural anticoagulation, acute closure remains largely unpredictable[3-5]. This dreaded complication often results in emergent CABG in about 40-50% of the time with the result that saphenous vein grafts rather than internal mammary arteries are used, a high infarct rate of 30-70% and up to 15% inhospital mortality (average 5%)[4,6]. The initial conventional approach to such patients usually involves repeat balloon angioplasty using a slightly oversized balloon inflated for longer periods (either with an autoperfusion or standard balloon catheter) with or without the use of intracoronary thrombolytic agent. This is however only successful in about 50% of patients. Irrespective of the management, periprocedural occlusion is associated with far worst short and long term outcomes as compared to those without this complication[4].

The pathomechanisms of acute closure are postulated to be due to either a major dissection or flap, an occluding thrombus, intramural haematoma or coronary vasospasm[7]. Theoretically, stent implantation in such a situation would be able to tack down any dissection or flap, provide a mechanical buttress against any elastic recoil or vasospasm and possibly prevent thrombus formation by maintaining patency and good antegrade flow.

### B. Prevention of Restenosis

Restenosis is truly an Achilles heel of PTCA, occurring in about 30-40% of patients. The incidence seems to be accentuated in patients with certain clinical, anatomic and procedure-related risk factors eg males[8-10], diabetics[10,11], patients with low HDL levels[12], recent onset/unstable angina (restenosis rate of up to 58% in patients with angina refractory to medical therapy)[8,10,13,14], severe pre-PTCA high grade stenosis[8,15,16] tubular, diffuse, chronic totally occluded or calcified lesions[16-21], proximal LAD, ostial lesion[16,22-27], multilesional and multivessel dilatation[21,28-31], use of undersized balloons[21,30,32], presence of post-PTCA major dissection[21,30], significant residual stenosis (>30 or >45% diameter stenosis)[21,30,33].

The time-frame for restenosis has been established. It occurs within the first 5 to 6 months following a successful PTCA after which it is quite uncommon[8,21,28,34]. It peaks at about 3 months post-PTCA[28,34]. Hence unless symptoms dictate otherwise, the optimal time for re-study angiography to determine restenosis is between the third and sixth month. Usually when stenosis recurs, it seems to resume its pre-dilated severity [15,21].

The pathomechanism of restenosis is gradually being deciphered. The central role of smooth muscle cell proliferation is no longer in doubt. In contrast, the precise contributory role of thrombus, elastic recoil, vasospasm and wall shear stress is less clear. Early evidence of the pivotal link between smooth muscle cell and restenosis came from post-mortem studies which demonstrated both smooth muscle cell migration from

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Table I - Current Stents under clinical evaluation

| Features<br>Design | Wallstent<br>Self-expanding mesh | Palmaz-Schatz<br>b-m slotted tube | Gianturco-Roubin<br>b-m wire coil | Wiktor<br>b-m wire coil |
|---|---|---|---|---|
| Material | stainless steel | stainless steel | stainless steel | tantalum |
| Metal bulk | +++ | ++ | + | + |
| Strut thickness | 0.06-0.08 mm | 0.1 mm | 0.15 mm | 0.15 mm |
| Flexibility | +++ | + | ++ | + |
| Expansion shortening | +++ | + | ± | ± |
| Radioopacity | + | + | + | +++ |

b-m = balloon-mounted
+++=high; ++=moderate; +=fair
± = none/minimal

the media into the intima, its proliferation and associated extracellular matrix volume expansion[35-37]. Additionally, Nobuyoshi et al[37] also confirmed the direct relationship between the severity of vascular injury and the extent and intensity of intimal hyperplasia which hitherto has only been shown in animal studies. Recently, with the advent of percutaneous coronary atherectomy, antemortem tissues from restenotic lesions were readily made available. The latter have consistently identified intimal hyperplasia as the underlying mechanism of restenosis, concurring with postmortem and animal studies[38-43]. Interestingly, tissues from restenotic plaques following other interventional procedures such as atherectomy, laser and stenting have also revealed intimal hyperplasia to be the cause of restenosis[42, 43]. This is probably not unexpected as smooth muscle cell proliferation is a normal intrinsic repair response to vessel wall injury, a common denominator in all the procedures mentioned above.

Stenting could theoretically prevent restenosis by the following ways:

1 It could tack back intimal flaps and seal off subintimal and medial splits, reducing exposure of deep tissues to blood components and ensuring forward laminar flow by providing a smooth and wide lumen[44]. By doing so, it could facilitate a thin layer of controlled thrombus formation (rather than an uncontrolled excessive thombosis) which is essential for rapid neoendothelialisation.
2 Stenting results in medial atrophy by reducing pulsatile radial wall stress[45,46] rather like that of heavily calcified coronary arteries[47]. This could form a fibrotic barrier to further fibrocellular ingrowth and an attenuation of smooth muscle cellular proliferation.
3 Stenting could compress and compromise the vasa vasorum underlying atheromatous plaques, rendering them ischaemic and retarding their progression[48].

## CONTRAINDICATIONS TO STENTING

### A. Anatomical contraindications

1 Lesion less than 10 mm from the left main coronary artery. Deploying a stent too close to the LMCA always poses the danger of the stent encroaching on the left main.
2 Vessel less than 3-3.5 mm in diameter or funnel-shaped. Stents in small and tapered vessel are prone to thrombotic occlusion[49,50].
3 Vessels with poor distal run-off. Stenting in this situation might promote thrombus formation[50].
4 Lesion at sharp bend or involving major side-branches where stents might increase shear force and side-branch occlusion respectively.
5 Diseased vessel segment with a high tendency to vasospasm. This will only exacerbate stent-induced spasm.

### B. Drug-related contraindication

Problems with the use of anti-platelet and anticoagulant therapy. All stents to date require rigorous anti-platelet medication and anticoagulation. Failure to adhere to this regime will lead to an unacceptable rate of acute stent occlusion due to thrombosis.

### C. Clinical contraindication

Presence of haemodynamic collapse. This is associated with poor antegrade flow which will lead to stent thrombosis.

## STENT DESIGNS IN CLINICAL USE

There are currently 4 common types of stents being tested clinically, one of which is a self-expanding stent while the other 3 are balloon-mounted types (Table I).

### 1. Self-expanding Wallstent

The Wallstent (Medinvent), the first stent design to be implanted in human coronary arteries (and peripheral arteries) is a self-expanding stainless steel mesh stent with a geometrically stable configuration when fully expanded[50]. It is longitudinally very flexible and pliable. For coronary utilisation, the stent varies from 10 to 30 mm in length and 2.5 to 6 mm in diameter when fully expanded. It is mounted on a small catheter (1.57 mm in outer diameter) and delivered on an over-the-wire co-axial system introduced percutaneously. A doubled-over membrane envelops the stent and serves to constrain and elongate the stent into a low profile unit, reduces friction, protects the stent from snagging during tracking and enhances radioopacity when filled with contrast thus facilitating precise stent placement. The optimal fully expanded diameter of the stent should be about 15 to 20% larger than the diameter of the arterial segment to be stented and its length should totally cover the barotraumatised segment. The stent is usually deployed following conventional balloon angioplasty via an exchange wire and a final repeat balloon inflation performed with the stent ("swiss kiss") to firmly embed the stent and smooth out any irregularities. There is a learning curve to it but in experienced hands, the success rate of implantation of the Wallstent is very high indeed whether in an emergent or elective situation.

### 2. Palmas-Schatz stent

The early prototype (Palmaz stent) was a rigid, slotted stainless steel tubular stent 15 mm in length with 12 rows of rectangles which when fully expanded transformed into diamond shaped configurations. It was tedious to employ as it required preloading and crimping onto a balloon and was very inflexible. In fact, the delivery success rate was only 80% [51].

Subsequently, the design was improved upon by Schatz who bisected it into 2 short 7 mm segments bridged by a 1 mm strut. This new design improved longitudinal flexibility, allowed deployment through tortuous arteries and increased the success rate to 95%. However, there was still problem with stent snagging and embolisation. This seems to have been

rectified with the introduction of a new 5F (1.67 mm diameter) superselective delivery sheath[52].

### 3. Gianturco-Roubin stent

It is a balloon expandable coil with a "bookbinder" design, made of stainless steel and is moderately flexible. It is delivered through the standard PTCA set-up like all other current stents and is relatively simple to deploy as it does not shorten appreciably with expansion unlike the Wallstent and Palmaz-Schatz stent.

### 4. Wiktor Stent

The Wiktor stent, unlike the preceding stents, is not made of stainless steel but tantalum, a metal with the added advantage of excellent radioopacity and also a potentially low thrombogenecity[53,54]. It is a wire coil balloon expandable stent.

## CLINICAL EXPERIENCE

### 1. Wallstent

The Wallstent has consistently been shown to produce a much better result in terms of angiographic[55-57] and haemodynamic parameters[58] compared to that of PTCA alone in both native coronary arteries and vein grafts[59]. There is a more regular luminal surface with a smooth transition between the stented segment and adjacent non-stented segment; a further increase in minimum luminal diameter/cross-sectional area and a further decline in translesional gradient compared to PTCA. This is a result of the scaffolding and radial expansion effect of the stent.

In 1987, Sigwart et al[50] first reported on the use of stenting as a bailout procedure following acute post-PTCA closure in 4 patients with exciting results. There was immediate abolition of ischaemia and none of the patients sustained infarction nor required CABG. Subsequent studies have substantiated these early encouraging findings[60,61]. Furthermore, emergent stent implantation was not more technically demanding than elective stenting and early stent thrombosis was observed to be low in all the series (Table II). In our own cohort of 35 patients with emergent stenting, only 3 patients developed early thombotic stent occlusion, 2 were transient and 1 was permanent (unpublished observation). Acute stenting was also associated with a low restenosis rate[60, 62].

**Table II - Stenting in Acute Bailout**

| | Wallstent | Palmaz-Schatz | Gianturco-Roubin | Wiktor |
|---|---|---|---|---|
| Success | >95% | >95% | >95% | High |
| Early occlusion | 6-17% * | 5% | 6% | NA |
| Restenosis | 11-18% | 11% | 33-44% | NA |

NA = no data available
*mostly transient

The other proposed role of stenting was as a secondary prevention option in post-PTCA restenosis and this was first tested clinically in 17 patients with promising immediate and short-term results[50]. A subsequent larger study by the same investigators confirmed their initial results, namely a high success rate with a low incidence of major complications[63]. The thrombotic stent occlusion rate was 6% despite a stringent anti-platelet and anticoagulation regime. The restenosis rate was 8% which was much lower than the restenosis rate for de novo native coronary balloon angioplasty (Table III). A recent study from the European Multicentre Wallstent Registry[57] which enrolled 105 patients has revealed a comparable restenosis rate (13%) over a 6 month follow-up period. Subgroup retrospective analysis have identified certain major predictors of stent thrombosis. Sigwart et al[64] found that unconstrained stent diameter and length and operator experience were key predictors of stent closure. Similarly, there are also risk factors for stent restenosis. The restenosis rate was high (41%) if the delay between previous PTCA and stenting was more than 3 months (as compared to 6% if the delay was longer than 3 months)[65]. In our experience, suboptimal stent placement was also an important risk factor (unpublished results).

**Table III - Stenting in Native Coronary Arteries**

| | Wallstent | Palmaz-Schatz | Gianturco-Roubin | Tantalum |
|---|---|---|---|---|
| Success | >95% | >95%* | High | >95% |
| Early occlusion | 3-16% | 3% | NA | 0-23% |
| Restenosis | 8-13% | 21-35% | NA | NA |

NA = no data available
* with the use of a special 5F protecting sheath

Primary stenting as an adjunct to PTCA to prevent restenosis using the Wallstent has been performed but to date, we are not aware of any published report although from our experience the number of previous angioplasties is not a significant predictor of restenosis. An even bigger problem than native coronary arterial stenosis is stenosis of vein grafts. Repeat bypass surgery is often difficult and, in fact, sometimes impossible. More importantly, it is associated with an increased morbidity and mortality. PTCA in this situation has a high success rate similar to that of native coronary angioplasty but the former is unfortunately faced with a high restenosis rate especially for lesions in the proximal segment and body of grafts[66]. There is also the added danger of distal embolisation of friable atheromatous material during PTCA of old, diffusely diseased grafts[67-69]. Stent implantation in grafts to prevent restenosis and debris embolisation is thus a very appropriate proposition. Furthermore, the fact that stenting of grafts is technically simpler than native coronary arterial stenting due to the absence of troublesome side-branches and vasospasm and the larger caliber of grafts, makes the former an even more attractive option. The first major published experience[70] on saphenous vein graft stenting involving 13 patients with 14 graft stenoses mostly in the mid-segment, noted a high success rate of stent implantation (95%) with no major complication and an angiographic recurrence rate of 20% which was significantly lower than the 30-50% restenosis rate observed with PTCA of body of grafts[66]. Subsequent larger reports have confirmed the high success rate and the attenuated restenosis rate[71, 72]. The incidence of stent thrombosis is around 7%, not dissimilar to that of stents within native coronary arteries[72] (Table IV).

**Table IV - Elective Stenting in Saphenous Vein Grafts**

| | Wallstent | Palmaz-Schatz | Gianturco-Roubin | Tantalum |
|---|---|---|---|---|
| Success | >95% | >95% | NA | NA |
| Early occlusion | 7% | NA | NA | NA |
| Restenosis | 15-30% | 17% | NA | NA |

### 2. Palmaz-Schatz stent

Reports on emergent implantation of this stent model for post-PTCA acute closure or suboptimal results are still scarce. Haude et al[73] and the Arizona investigators[74] attained a high stent implantation success rate with a remarkably low thrombotic occlusion rate (5%). Both groups arrived at the same conclusion with respect to the restenosis rate (Table II). The latter was only about 11-15% for single stent and extremely high for multiple stents (67-100%).

Most data on the Palmaz-Schatz stent are derived from elective implantation in native coronary arteries to prevent restenosis (Table III). A recent multicentre experience[51] re-

ported a high success rate of 95% with the modified articulated stent design in contrast to only 80% with the rigid prototype. The success rate was further improved when the 5F superselective sheath was introduced to reduce stent snagging and embolisation[52]. Other factors which affected the success of stenting were also identified. They included the site of implantation (proximal has higher success than distal), presence of dissection (success better without than with dissection), and operator experience/patient selection (success of 89% in the first 50 patients and 98% in the last 50). The overall major complication rate was low (4%). Interestingly, the investigators did not administer anticoagulant in their first 39 patients and was confronted with an 18% early thrombotic occlusion rate. The latter was dramatically reduced to less than 1% with full warfarin anticoagulation in subsequent patients[51]. More recent studies have similarly found a low occlusion rate (3-4%)[75,76]. Stent occlusion seems to be aggravated by undersized stents and stent placement in severely curved segments or in areas with pre-existing thrombus[75]. Some preliminary data on restenosis are now available[74,77,78]. It is between 20-35% depending on the number and size of the stents[79]. Single stent implantation seems to be associated with a very favourable outcome. The restenosis rate is only 20% when implanted in dilated chronic total obstruction which is usually associated with a 50% recurrence rate following plain old balloon angioplasty (POBA). Occurrence of restenosis after 6 months was uncommon, a situation analogous to PTCA restenosis which implied that stenting did not delay the temporal course of restenosis[78]. This is not unexpected as the underlying pathomechanism of restenosis following both procedures (stenting and PTCA) as alluded to earlier, is the same, namely that of intimal hyperplasia.

Only one preliminary report on the use of this stent in SVG has been published so far[80] (Table IV). Ninety patients with stenoses situated in the body of the grafts were stented. The implantation success rate was 98% and a 6 month follow-up angiographic re-study involving only 31% of patients revealed a restenosis rate of 17%.

**3. Gianturco-Roubin stent**

Although the Gianturco-Roubin stent is easy to implant as it shortens only minimally with expansion unlike the Wallstent and to a lesser extent, the Palmaz-Schatz stent, it does not however produce as smooth a lumen as the other 2 models.

Acute stenting with this endoprosthesis was tested in the first of 3 phase protocols. In Phase I[81], by design all patients were sent for CABG following stent placement. Six patients were stented but wide patency was established in 5. There were no death or infarct post-operatively. In Phase II, definitive stenting was performed in acute and threatened closures. As of June 1990, 150 patients had been stented in this protocol[82]. The stent was successfully placed in 96% of patients with 6% each ending up with thrombosis or in-hospital CABG. The restenosis rate was between 33 to 44% (Table II). In the currently ongoing Phase III of the trial, the issue of restenosis prevention by primary stenting will be addressed. Early results indicate a low thrombosis and emergent CABG rate of 2% each[82]. Restenosis rate is pending.

**4. Wiktor stent**

Data on this stent model are even less and very preliminary. So far results in terms of improvement in rheologic and morphologic parameters and implantation success rate in both acute and elective stenting have been favourable[84,85]. In contradistinction to experimental evidence, tantalum stent in clincial practice is not very much different from stainless steel stents. It is not without any thrombogenicity especially so when used as a bailout device[84] and when patients are not anticoagulated[86]. The restenosis rate of the Wiktor stent is pending.

## STENT-RELATED COMPLICATIONS

**1. Thrombotic occlusion**

This complication which usually occurs in the first 2 weeks following stent implantation is a major stumbling block to a more widespread utilisation of stents. Fortunately, most of them are resolvable with the use of thrombolytic therapy and PTCA. To date, all metallic stents are thrombogenic to varying extent at least until the bare metal is covered by a layer of neoendothelium. The occlusion rate varies from 1 to 40% depending on a large number of factors which include the type of stent, the size and length of the stent, operator experience, vessel anatomic factors (small caliber vessels, poor distal runoff, collaterals, intracoronary thrombus), clinical milieu (hypotension, shock, unstable ischaemic syndromes) and of crucial importance, the stringency of anti-platelet and anticoagulation regime as various investigators have found out the hard way. All stents in current use without exception, require full anticoagulation and anit-platelet agents. The Wallstent and Gianturco-Roubin stent have an average 6% occlusion rate[63,82] which seems to be slightly higher than the 1-4% observed with the Palmaz-Schatz stent[51,75,76].

**2. Vasospasm**

Vasospasm has been reported to occur in up to 11% of patients implanted with the Wallstent[87]. It usually occurs at the ends of the stents soon after stenting and is readily relieved or prevented by intracoronary nifidepine.

**3. Within-stent restenosis**

The incidence of within-stent restenosis varies widely as it seems to be influenced by multiple factors eg the stent model, the number of stents[79], accuracy of stent placement, and whether the stent was used for primary or secondary prevention of restenosis[88]. It is between 8 to 13% for native coronary arteries[57,63] and about 24% for vein grafts[72] with the Wallstent. The restenosis rate is not much different when only one Palmaz-Schatz stent is used (about 11%)[79] but shoots up to a forbiddable 70-100% incidence when multiple overlapping stents are deployed[73,74,79]. In contrast, the use of multiple Wallstents is not a predictor of restenosis. The restenosis rate for the Gianturco-Roubin stent seems to be uniquely high. The current treatment for stent restenosis is by either repeat PTCA or atherectomy[60]. If all fails and should it be necessary, CABG is still a viable option as it is not jeopardised by stenting.

**4. Deployment failure**

Deployment failure due to stent inflexibility and snagging used to be a significant problem with the Palmaz-Schatz stent before the advent of the articulated design and the special low profile covering sheath. The membrane protecting the Wallstent during deployment mitigates against this complication.

**5. Stent migration, perforation, erosion, and infection**

Stent migration and embolisation following failed deployment has been reported in the Palmaz-Schatz stent[51]. This is now very unusual with the use of the new protective sheath. Stent perforation, erosion and infection have not been noted in any of the current stent designs. No antibiotic prophylaxis is presently advocated for uncomplicated Wallstent and Gianturco-Roubin stent implantation to prevent infective endocarditis of the stented site. Some investigators have recommended routine prophylaxis for 3 months following Palmaz-Schatz stenting[51]. The risk of endocarditis must be very small.

**6. Side-branch occlusion**

Animal studies have consistently demonstrated preservation of

side-branches bridged by stents[49,89-92]. However in clinical practice, many investigators have avoided stenting coronary arteries with major side-branches[51,93]. This is probably justified if the pore size of the stents is small, eg Wallstent as the latter will prevent access to these side-branches. Side-branch occlusion has been documented to occur although not commonly so[63]. This was recently confirmed by Fischman et al[94] who described 46 major side-branches bridged by stents, including about one-third with stenosis at their ostium. Only 1 was occluded during the procedure but reopened subsequently.

### 7. Bleeding

This complication is mainly restricted to the vascular access site and is more frequent than with PTCA because the sheaths are removed with the patient fully anticoagulated to minimise stent thrombosis. About 5-10% develop vascular complication requiring transfusion/surgery[51,60,76]. New pneumatic groin compression devices and collagen plugging of access site may reduce this complication substantially.

## CURRENT RECOMMENDATIONS ON THE USE OF STENTS

Bailout stenting for acute post-PTCA closure has been shown in various observational studies to be a feasible, safe and highly effective method of re-establishing antegrade coronary blood flow and maintaining luminal patency. It dramatically relieves ischaemia, salvages myocardium and may permanently mitigate against the need for CABG or may at least attenuate the urgency of surgery, thus minimising the morbidity and mortality associated with emergent CABG and allowing the use of internal mammary graft instead of the less durable vein grafts. The stent thrombosis risk of about 6% certainly seems an acceptable proposition in this situation when weighed against the disadvantages of emergent CABG. Hence, stenting as a bailout procedure is a recommended therapeutic option. The Wallstent, Palmaz-Schatz and Gianturco-Roubin stents are suitable choices although the Gianturco-Roubin stent has a higher propensity to restenose in this clinical setting. It is too premature to comment on the tantalum stent at this stage. Elective secondary stenting of restenotic native coronary lesions has been tested and proven to be a valuable treatment modality. It is safe, has a highly successful procedural rate with minimal major complication. The restenosis rate for the Wallstent appears to be lower than that of the Palmaz-Schatz stent. There is as yet insufficient information on the other 2 types of stent. Stenting stenotic vein grafts is extremely attractive. It is safe, technically easier to perform than native artery stenting, produces superior haemodynamic and anatomic results and has a high implantation success rate. From uncontrolled studies using the Wallstent, the restenosis rate is generally significantly lower than following "stand-alone" PTCA and there is a lesser risk of embolisation of friable atheromatous material. It is probably indicated in the treatment of old lesions (more than 3-5 years) situated in the proximal segment/body of graft where restenosis following PTCA alone is exceedingly high. As for simple distal anastomotic lesions, the indication for stenting is nebulous at best. The Wallstent is probably the stent of choice at present as there is either insufficient or no information on the other 3 stent models at the time of writing this article to make any recommendation on their use in graft stenting.

Another controversial issue in stenting is its place as a primary tool in preventing restenosis of de novo lesions following PTCA. This is especially debatable for low risk lesions with excellent immediate post-PTCA results. The main arguments against stenting in this situation are the problems of having to deal with the small but not insignificant 3-10% risk of thrombotic occlusion of the stents and the potential bleeding sequalae of long term anticoagulation following stent placement. A prospective randomised trial comparing PTCA alone and PTCA with adjunctive prophylactic stenting in this situation will definitely go a long way in solving this dilemma.

## THE FUTURE

The pioneering work on stents in animals and human by Dotter and Sigwart et al has ushered in a new facet in interventional cardiology. The ability to stent coronary arteries and grafts is no longer a dream but a reality with an immense potential clinical utility in circumventing the daunting problems of post-PTCA acute closure and restenosis. Although the perfect stent (Table V) is still out of reach, rapid progress is being made. The future stent should incorporate a more refined delivery system, and more radioopaque and non-thrombogenic biologically inert stent material. Avant garde technologies in their experimental stages are looking into the use of metallic stents coated with heparin, polymer, hirudin, t-PA genes or endothelial cells[95-99] targetted at reducing thrombosis and restenosis. Some of these coated stents have encountered less thrombosis than uncoated ones in the animal model. Polymeric tubular stents are being tested in the laboratory[100]. Biodegradable polymeric endoluminal paving and sealing (with the possibility of coating) are also being looked into[101]. The unresolved problems with the latter are the potential thermal damage to the vessel wall during the molding process, the long-term effects of the polymer on the vessel, the question of side-branch occlusion and the effects of physically separating the intima from blood. Other possibilities include impregnation of stents with platelet inhibitors or antimitogenic agents. The stent of the future which might overcome some of the major shortcomings of current stents will probably the either a biodegradable stent with drug elution capabilities or a coated metallic stent with drug impregnation.

**Table V - Characteristics of Ideal Stents**

| | |
|---|---|
| 1 | Structural integrity with sufficient radial force |
| 2 | Flexible and low profile |
| 3 | Stable once expanded |
| 4 | Simple and safe to use |
| 5 | Nontoxic, biologically inert or biodegradable |
| 6 | Nonthrombogenic |
| 7 | Highly radioopaque |
| 8 | Reliable expandability with large expansion ratio |

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