Early and late results of intracoronary arterial stenting after coronary angioplasty in dogs


ABSTRACT Intimal dissection with acute closure represents the major complication associated with percutaneous transluminal coronary angioplasty (PTCA). Intracoronary stent devices offer the possibility of treatment for this sequela. We developed a balloon catheter-mounted, flexible coil stent for use in such cases. To determine the utility of this device and its immediate and long-term influence on arterial patency, 39 mongrel dogs had the stent placed after PTCA of the left circumflex or left anterior descending coronary arteries. Thirteen animals were treated before and after the procedure with warfarin. In this group there were three early deaths associated with stent thrombosis. Twenty-six animals were subsequently treated before and after with aspirin and dipyridamole. There were no early thrombotic events associated with stent placement in these animals. Late arteriographic examination revealed patent vessels in all dogs. Diameter stenosis for warfarin-treated dogs was 8 ± 5% (mean ± SD) at 2 months (n = 9), 6 ± 4% at 6 months (n = 5), and 11 ± 7% at 12 months (n = 3). Diameter stenosis for aspirin/dipyridamole-treated dogs was 9 ± 3% at 2 months (n = 8), 8 ± 5% at 6 months (n = 12), and 5% at 12 months (n = 1). Light and scanning electron microscopic analyses of stented arteries demonstrated incorporation of the stent wires into the arterial wall. Early findings included mild thrombosis localized to areas of wire entrenchment followed by rapid regrowth of endothelial and/or pseudoendothelial cells over trenches, exposed wires, and elastica. Late histologic studies revealed that the stented segments had thinning of the media, mild neointimal proliferation over stented wires, and an otherwise normal intimal surface. Flexible intracoronary stents can be placed safely, rapidly, and accurately from a femoral arterial entry site and can maintain long-term patency in the dog. Further testing in other preparations will elucidate the effects of stenting in atherosclerotic vessels.


CURRENTLY AVAILABLE balloons used in percutaneous transluminal coronary angioplasty (PTCA) are capable of producing a cylindrical lumen of predetermined diameter within most arteries dilated. Early ischemic complications usually relate to closure of the vessel lumen immediately after balloon deflation or in the following 24 hr. Lumen reduction results from encroachment by dissecting intima, plaque, and medial structures, elastic recoil of the vessel wall, and occasionally arterial spasm. Once blood flow is reduced, thrombosis and total occlusion may occur.

The occluded artery can be redilated but more often it is necessary to proceed to emergent surgical revascularization.1, 2 The increased mortality of coronary bypass surgery when performed under these conditions is usually associated with ongoing myocardial ischemia.3 Although coronary perfusion catheters are now available, the flow provided by currently available devices is suboptimal.4

Dotter5 first introduced the concept of implantable prosthetic stents to maintain the luminal integrity of dilated arteries and the technique has recently been applied to human coronary arteries after PTCA.6 Problems encountered include difficulties in the precision of percutaneous implantation and immediate and late closure usually related to thrombosis.6–8 We developed an intracoronary stent device that can be mounted on a balloon catheter and expanded to a predetermined diameter to maintain the dilated lumen. The present study was undertaken (1) to determine whether the stent

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could be placed safely and precisely within canine coronary arteries via a “percutaneous” technique and (2) to examine the immediate and long-term arteriographic patency and the histologic response of the arterial wall.

Methods

**Balloon-mounted intracoronary stent.** The stent is made of monofilamentous stainless steel (0.006 inch diameter) formed into a interdigitating coil structure and wrapped tightly around a standard, deflated balloon dilatation catheter (figure 1). The expanded diameter of the stent is determined by the diameter of the polyvinyl balloon catheter used and the inflation pressure employed at the time of stent expansion. Balloon catheters of 3 mm diameter were used in the present study. The deflated balloon catheter is flexible and positioned in the dilated coronary artery segments by a standard over-the-guide wire technique.

The integrity of the metal coil was tested by inflating the device inside a 3 mm diameter polyvinyl tube and subjecting the system to mechanical flexion (30 degrees) at a rate of 120/min for 2 weeks (2,419,200 flexion changes). The system was continuously flushed with twice normal saline. Examination of the coil at 2 weeks revealed no structural defects.

**Animal preparation.** Thirty-nine mongrel dogs weighing 20 to 30 kg and free of heartworm disease were obtained for use in this study. All animal care and handling was performed in accordance with NIH guidelines for the care and use of laboratory animals. An initial group of 13 animals was pretreated with warfarin sodium 5 mg orally per day for 3 days. After problems were experienced in maintaining the dog colony on this oral anticoagulant, the remaining 26 animals were given aspirin 325 mg and dipyridamole 75 mg orally three times daily for 3 days before stent placement.

The animals were anesthetized with pentobarbital sodium (25 mg/kg iv). Supplemental pentobarbital was administered as necessary. Because of the degree of anticoagulation, difficulty in restraining the dog’s hind limbs and difficulty in maintaining effective compression, hematoma formation was avoided by performing a femoral arterial cutdown and arteriotomy. A No. 8F or 9F introducer sheath was then placed in the femoral artery. A No. 8F or 9F angioplasty guiding catheter was introduced and advanced over a 0.038 inch guidewire under fluoroscopic control to engage the ostium of the left main coronary artery. Left coronary arteriography with meglumine diatrizoate (Renografin 76) was performed.

**Instrumentation.** A 3.0 mm diameter, 20 mm length balloon dilatation catheter was advanced over a 0.014 inch steerable guidewire to the site selected for instrumentation (left anterior descending, n = 11; left circumflex, n = 28). To induce endothelial damage, the balloon was inflated twice to a pressure of 8 atm for 20 to 30 sec, deflated, and then withdrawn. A catheter for placement of the stent coil (with a 3.0 × 20 mm balloon) was then introduced and advanced over the guidewire to the site dilated. The balloon was inflated once to 8 to 10 atm for 30 to 45 sec; it was then deflated, and negative pressure was maintained for approximately 30 sec. The catheter was advanced slightly to disengage the stent, then slowly withdrawn, leaving the stent in place. Repeat left coronary arteriography was performed, the guide catheter was removed, the femoral arteriotomy was repaired, and the surgical incision was closed. Heparin (10,000 U iv) was administered during the course of the procedure, and penicillin (500,000 to 1 million U im) was administered after completion of the procedure. The experimental protocol was approved by the Animal Experimentation and Care Committee of Emory University.

**Poststenting treatment and examination.** The medications used for pretreatment were continued for a period of 30 days in each group, with the aspirin and dipyridamole given only once daily. Catheterization for follow-up angiography was performed in the manner described above at approximately 2, 6, and 12 months after instrumentation. Angiography was performed in at least two projections, usually the right anterior oblique and left anterior oblique. Animals were killed for pathologic examination by intracoronal or intravenous injection of a lethal dose of sodium pentobarbital. The entire heart was then excised, washed, and fixed in 10% buffered formalin.

**Arteriographic analysis.** Arteriograms were measured by an experienced angiographer on a Siemens projector using digital electronic calipers (Sandhill, Littleton, CO). Percent stenosis was determined by measurement of the most severe luminal narrowing at the site of stent placement compared with the mean arterial diameter measured proximal and distal to the site of the stent. The percentage stenosis measured from two views was averaged to obtain individual data points. The data are presented as the mean ± SD of the mean.

**Pathologic examination.** Six dogs pretreated with aspirin and dipyridamole were killed immediately after stent placement to examine the early histologic effects of stent placement. One dog was killed at 3 days, one each at 1, 2, 3, and 4 weeks, four at 2 months, nine at 6 months, and two at 12 months. The stented coronary arteries for light microscopic analysis were dissected from the epicardial surface and examined grossly before extracting the embedded stent wire by freeing one end of the stent and uncoiling the device by pulling from an end of the intact arterial segment. The stented segments and adjacent unstented segments

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**FIGURE 1.** A. Stent coil wrapped firmly on standard PTCA balloon catheter prepared for use. B. Stent fully expanded by maximally inflated balloon catheter demonstrated in transparent flexible tubing. C. Stent fully expanded after removal of deflated balloon catheter.
were then prepared in a standard manner for histologic examination at our institution. Six hearts with long-term stents were forwarded to another pathologist for confirmatory analysis.

A sample of specimens was also prepared for scanning electron microscopy. After dissection of the artery from the epicardial surface, the specimen was fixed by perfusion with 2.5% glutaraldehyde buffered with cacodylate at 100 mm Hg pressure. Specimens of normal and stented arteries were then carefully opened longitudinally to display the endothelial and cut surfaces as well as the ostial origin of any side branches originating within the stent. The specimens were critical-point dried and sputter coated with gold/palladium before scanning electron microscopy.

**Results**

**Stent placement and device failure.** In 36 of the 39 placements, the device was placed precisely in the desired coronary arterial segment, expanded, and maintained in position. During our early learning experience, there were three failures related to operator error. In dog 55719, the balloon was withdrawn before full deflation and the expanded stent was withdrawn out of the coronary artery. A second stent was placed in this animal without complication. In two dogs (Nos. 11034 and 11288) the stents were undersized for the arterial segment dilated and after balloon deflation migrated to a more distal segment. Neither animal suffered an untoward event and the stents maintained their final positions through late follow-up studies.

**Immediate arteriographic and histologic results.** Immediate arteriographic patency was demonstrated in all cases. In no case was the stent placement associated with arrhythmias, ST segment changes, or immediate death. In 10 dogs, a stent was placed so that a major side branch was bridged. In no case was there angiographic evidence of compromised side branch blood flow. In no case was there arteriographic evidence of vessel dissection, perforation, rupture, or immediate thrombotic occlusion of the stent.

Early pathologic examination (n = 6) revealed that the stents embedded into the intima without gross thrombotic debris, perforation, or hemorrhage of the arterial wall. Histologic examination showed that the segment was dilated with loss of endothelial cells and fragmentation of the internal elastic lamina consistent with balloon inflation. In some sections, the media was stretched and thinned, with occasional extravasated erythrocytes evident between muscle cells. Some smooth muscle cells in these areas showed morphologic changes consistent with recent injury. Sections of distal coronary arterial branches and myocardium were unremarkable.

**Early results.** Of the initial 13 dogs anticoagulated with warfarin, four died at 5, 6, 10, and 36 days after stent placement (table 1). In two dogs death was associated with stent thrombosis, one died of a mediastinal hemorrhage, and in the fourth the cause of death was not determined. No premature deaths occurred in the latter series of 26 animals pretreated with aspirin and dipyridamole. Scanning electron microscopy of arteries stented for 3 days and 1 and 2 weeks revealed that the stent wires were firmly embedded within folds of the media muscularis, covered by a fibrin material at 1 week and by endothelial cells and/or pseudoendothelial cells at 2 weeks (figure 2, A and B). The neointimal layer was approximately 300 μm in thickness covering stent wires where expansion had embedded them into the arterial wall. Between the stents the intima was moderately thickened. The intima was sparsely covered with microthrombi, adherent platelets, and white blood cell components, including macrophages. A side branch ostium between stent wires remained widely patent.

**Late arteriographic results.** There were no premature deaths or untoward clinical events in any of the remaining animals. Arteriographic results are shown in table 2. There was no significant difference in late arteriographic results between the two treatment groups and no evidence of a significant increase in luminal nar-

<table>
<thead>
<tr>
<th>Dog</th>
<th>Days to death</th>
<th>Warfarin dose</th>
<th>Appearance of artery</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>11290</td>
<td>5</td>
<td>5 mg daily × 3</td>
<td>Nonoccluding thrombus</td>
<td>Cause of death thought to be secondary to hemorrhage from femoral artery</td>
</tr>
<tr>
<td></td>
<td>4 mg daily × 5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11296</td>
<td>6</td>
<td>5 mg daily × 3</td>
<td></td>
<td>Carcass incinerated before autopsy could be performed</td>
</tr>
<tr>
<td></td>
<td>4 mg daily × 6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>55719</td>
<td>10</td>
<td>5 mg daily × 13</td>
<td>Stent site patent</td>
<td>Improper handling in transport; mediastinal hemorrhage</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11275</td>
<td>36</td>
<td>10 mg × 1</td>
<td>Occluding thrombus</td>
<td>Died 6 days after warfarin therapy stopped</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5 mg daily × 32</td>
<td></td>
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</tbody>
</table>
FIGURE 2. Scanning electron micrographs of longitudinally cut sections of stented arteries showing luminal surfaces. A, At 1 week after placement, stent wires (a) are seen embedded in trenches and (b) covered with fibrin clot. B, At 2 weeks after placement, significant regrowth of intimal cells over trenches has occurred. A patent side branch (arrow) is visible between stent wires. C, At 12 months after placement, a layer of mature endothelial and/or pseudointimal cells cover the stent wires. There was no evidence of microthrombi or adherent platelets. Particulate debris (arrow) was found to be fragments of epithelial cells of uncertain origin. (Original magnifications × 26).

Rowing of the stent site over time. Mean diameter stenosis was 9 ± 4% at 2 months (n = 17), 7 ± 5% at 6 months (n = 17), and 10 ± 6% at 12 months (n = 4). All side branches in surviving dogs were patent at repeat arteriographic study (figure 3). In one additional animal in which the stent appeared stable at initial angiography, late study revealed that the stent had migrated 10 mm distally in the artery. The vessel was otherwise unaffected.

Late histologic results. There was no significant difference in late histologic findings in dogs treated with warfarin or aspirin/dipyridamole and no difference between stented arteries examined at 2, 6, and 12 months. In all cases the stented segment remained dilated compared with control segments (figure 4). The stents were uniformly embedded within a neointimal cellular layer containing abundant mature collagen and averaging 270 μm in maximal depth from the internal elastic lamina to the endothelial surface. This layer was thickest adjacent to the stent wires and was often attenuated away from the wires. In most sections, evidence of old hemorrhage (hemosiderin deposition) could be seen near the wires, and some neovascularity in these areas was also noted. Thinning and slight fibrosis was present in the media underneath the wires; all lay within the original internal elastic lamina. Scanning electron microscopy of an artery stented for 12 months revealed a continuous mature endothelium.
should be flexible to accommodate the normal motion of epicardial arteries but should maintain its position after placement. Stent material should be histologically inert and cover as small an area of endothelium as possible to ensure rapid reendothelialization. The stent must not induce acute thrombosis and must demonstrate long-term patency. Finally, the device should be radiopaque.

This study has demonstrated that such a device can be placed safely in coronary arteries by a percutaneous technique and supports similar findings by others.\textsuperscript{5, 6} Canine coronary arteries approximate human counterparts in size and are ideal for studying the technical utility of such devices. In the present study, the stent-mounted balloon catheter was flexible and of sufficiently low profile to allow its placement in any proximal or mid arterial segment that could be traversed by a guidewire. Although not necessary in the model we used, the stent could be placed over an exchange wire. This is an important consideration when coronary arterial occlusion is due to dissection.

<table>
<thead>
<tr>
<th>TABLE 2</th>
<th>Arteriographic results</th>
</tr>
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<tbody>
<tr>
<td>Dogs</td>
<td>Side branch</td>
</tr>
<tr>
<td>Warfarin treated</td>
<td></td>
</tr>
<tr>
<td>11034</td>
<td>No</td>
</tr>
<tr>
<td>55784</td>
<td>No</td>
</tr>
<tr>
<td>55802</td>
<td>Yes</td>
</tr>
<tr>
<td>55918</td>
<td>No</td>
</tr>
<tr>
<td>11288</td>
<td>No</td>
</tr>
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<td>11276</td>
<td>No</td>
</tr>
<tr>
<td>11299</td>
<td>Yes</td>
</tr>
<tr>
<td>11278</td>
<td>No</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>8 ± 5</td>
</tr>
<tr>
<td>Aspirin/dipyridamole treated</td>
<td></td>
</tr>
<tr>
<td>11549</td>
<td>No</td>
</tr>
<tr>
<td>11545</td>
<td>No</td>
</tr>
<tr>
<td>11547</td>
<td>Yes</td>
</tr>
<tr>
<td>11548</td>
<td>Yes</td>
</tr>
<tr>
<td>11550</td>
<td>No</td>
</tr>
<tr>
<td>11584</td>
<td>No</td>
</tr>
<tr>
<td>11634</td>
<td>No</td>
</tr>
<tr>
<td>11629</td>
<td>No</td>
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<tr>
<td>11650</td>
<td>Yes</td>
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<tr>
<td>11897</td>
<td>No</td>
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<td>11910</td>
<td>Yes</td>
</tr>
<tr>
<td>11888</td>
<td>Yes</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>9 ± 3</td>
</tr>
<tr>
<td>Total</td>
<td>26</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>9 ± 4</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Euthanized for histologic analysis.
\textsuperscript{b}All side branches patent at restudy.

and/or pseudoendothelium incorporating stent wires without evidence of thrombosis, microthrombi, or adherent platelets (figure 2, C).

**Discussion**

Intracoronary stent devices have significant potential for increasing the efficacy and safety of PTCA.\textsuperscript{6} To be effective they should fulfill the following criteria. The stent should be able to be placed accurately and safely within the precise coronary artery segment requiring treatment. Expansion of the stent should be controlled so that a given diameter can be achieved accurately. The material should have properties that allow its expansion within the artery and then to withstand the elastic recoil of the arterial wall tissues it is dilating. It

![FIGURE 3. Arteriography of a left circumflex artery in the left anterior oblique projection at 6 months showing a widely patent vessel and side branches originating from within the stent (area between arrows).](http://circ.ahajournals.org/)

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FIGURE 4. A. Representative sample of a specimen taken from a nonstented segment of a left circumflex artery. The apparently smaller lumen is a fixation artifact. B. Stented segment from the same artery at 6 months. The segment remains widely dilated. The space occupied by a stent wire (a) is seen within the media muscularis and covered by neointima. A moderate, nonocclusive layer of neointima covers the entire luminal surface of the artery. Separation of intima from media (b) is artifactual. (Original magnifications × 43.)

Early technical failure was caused by distal migration of the device after placement in two cases and by withdrawal of the stent in one case. Each was due to relative undersizing of stents, and in the latter case to the failure to allow time for adequate deflation of the balloon before withdrawal. One stent noted to have migrated distally at follow-up probably did so soon after placement and initial arteriography. These problems occurred in our early series of animals and were not encountered as experience increased. Self fixation and stability within the treated artery segment are of paramount importance in these devices. Other stents have used the elastic modular properties of the intrinsic coil design or thermally induced coil expansion to ensure fixation. In these devices comparatively complex placement and release mechanisms are required. The coil design used in this study exhibits mild elastic properties only after expansion to a diameter determined by the diameter of the balloon catheter on which it is mounted and the length of the interdigitating coil loops. Correct sizing proved to be critical to stability, particularly in the relatively “smooth, normal” canine coronary arteries used in the present study. Distal migration may be less of a problem in more irregular, atherosclerotic human arteries after PTCA.

When appropriately sized, the stent coils used in this study were superficially but firmly embedded within the arterial wall (figure 2). In addition to providing stability, this embedding may have promoted rapid covering by neoendothelium. Late histologic studies demonstrated fibrocellular tissue, hemosiderin deposition, and neovascularity surrounding the stent wires. These areas resembled old, organized thrombus. It is possible that localized, nonocclusive thrombus formation with subsequent organization and reendothelialization aids incorporation of this device into the arterial wall.

Atherosclerosis is difficult to induce in dogs and the preparation cannot be used to determine how the present device will function in the presence of atheromatous obstruction. Other animals must be used for this purpose.

In the present study, acute, superficial endothelial injury was induced by first inflating standard balloon catheters before stent placement. This acute injury should have enhanced the early thrombotic potential for the preparation and differs from the canine coronary preparation used by other investigators. Acute thrombosis has been reported in both canine and human coronary arteries stented after coronary angioplasty and was a problem in the initial phase of the current study. Warfarin anticoagulation was used in the first 13 animals. Thrombotic occlusion was the cause of death in one dog, which died 36 days after stent placement (6 days after cessation of warfarin therapy), and was probably the cause of death in another animal, which was erroneously removed from the colony soon after death. Difficulties in maintaining the dog colony on warfarin led to use of antiplatelet therapy with aspirin and dipryridamole in the second 26 animals used in the study. There were no thrombotic events in this group.

In the present study, all animals were treated intraoperatively with heparin. Previous attempts at intra-arterial stenting without the use of heparin has been associated with acute thrombosis and, as with PTCA itself, heparin appears to be a prerequisite.

Acute thrombotic occlusion more than 48 hr after balloon angioplasty is rare, probably because protective endothelium rapidly regenerates. The thrombotic potential of an endovascular stent may also be related
to the speed with which it is covered by neoendothelium. The present device covers approximately 12% of
the endothelial surface and placement involves embedding the coil wires within the original endothelial sur-
face. Histologic examination at 3 weeks demonstrated complete reendothelialization of the stent wires, but
additional studies will be required to determine the precise time course for endothelial covering, the nature
of the cell layer (i.e., endothelial or pseudoendothelial), and the incidence of significant thrombosis
during this time.

Early closure of intra-arterial stents may also be caused by inflexibility of stents inducing arterial kink-
ing at transition zones. Overseizing of stents resulting in abrupt luminal reduction at the distal transition site
has also been reported to promote acute occlusion, and spasm has been reported in man. None of these
problems were encountered in the present study.

Late coronary arterial patency with minimum luminal narrowing was been demonstrated in all animals in
the study. Serial pathologic examinations and arterio-
graphic studies in the same animals suggest that after
the initial regenerative process, the endothelium remains stable. Of importance was the finding that both
minor and major side branches originating from within
the stent remained patent (figures 2 and 3). These data
confirm previous results in canine coronary arteries
with different metal endocoronary prostheses. These
data cannot be transferred to expectations in human
coronary arteries but are encouraging because the endo-
thelial response in dogs to endovascular prostheses
closely approximates that observed in man. Preliminary
human studies with stents of a flexible, metal mesh
design also suggest that long-term patency can be
maintained even in atherosclerotic arteries that have
previously restenosed after balloon dilatation. Of
some importance has been the finding by Palmaz et al.
that a mesh stent design maintained patency in
atherosclerotic rabbit aortas by restricting further ath-
erogenesis to layers external to the stent. Studies are in
progress to determine whether the coil design used in
this study will show similar effects.

Another potential, albeit unlikely, problem in the
placement of these prostheses within constantly mov-
ing coronary arteries is that of metal fatigue with frac-
ture of coils and subsequent migration of fragments
within the arterial wall. Given the fine nature of the
material (0.006 inch diameter), its relatively low mass,
and the degree to which the material is firmly incor-
porated into the tissues of the arterial wall, such conse-
quences seem remote. Long-term studies are under
way to investigate this possibility.

This study has demonstrated that a flexible coillike
endovascular stent device can be precisely positioned
within the coronary arteries via a percutaneous tech-
nique. Used with antiplatelet agents, the stent was not
associated with acute thrombotic events, and long-term
patency has been documented. The device has the
potential for maintaining an effective lumen in patients
with early closure after PTCA.

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