Factors Portending Endoleak Formation After Thoracic Aortic Stent-Graft Repair of Complicated Aortic Dissection

Daniel Y. Sze, MD, PhD; Maurice A.A.J. van den Bosch, MD, PhD; Michael D. Dake, MD; D. Craig Miller, MD; Lawrence V. Hofmann, MD; Robin Varghese, MD; S. Chris Malaisrie, MD; Pieter J.A. van der Starre, MD, PhD; Jarrett Rosenberg, PhD; R. Scott Mitchell, MD

**Background**—Endoleaks after stent-graft repair of aortic dissections are poorly understood but seem substantially different from those seen after aneurysm repair. We studied anatomic and clinical factors associated with endoleaks in patients who underwent stent-graft repair of complicated type B aortic dissections.

**Methods and Results**—From 2000 to 2007, 37 patients underwent stent-graft repair of acute (≤14 days; n=23), subacute (15 to 90 days; n=10) or chronic (>90 days; n=4) complicated type B aortic dissections using the Gore Thoracic Excluder (n=17) or TAG stent-grafts (n=20) under an investigator-sponsored protocol. Endoleaks were classified as imperfect proximal seal, flow through fenestrations or branches, or complex (both). Variables studied included coverage of the left subclavian artery, aortic curvature, completeness of proximal apposition, dissection chronicity, and device used. Endoleaks were found during follow-up (mean, 22 months) in 59% of patients, and they were associated with coverage of the left subclavian artery (complex, P<0.001), small radius of curvature (type 1 and complex, P=0.05), and greatest length of unapposed proximal stent graft (complex, P<0.0001). During follow-up, 10 endoleaks resolved spontaneously, 6 required reintervention for false lumen dilatation, and 2 were stable without clinical consequences.

**Conclusions**—Endoleaks are common after stent-graft repair of aortic dissection and may lead to false lumen enlargement necessitating reintervention. Anatomic complexities such as acute aortic curvature and covered side branches were associated with endoleaks, illustrating the need for dissection-specific device development. (Circ Cardiovasc Intervent. 2009;2:105-112.)

**Key Words:** aorta ■ dissection ■ surgery ■ complications

---

Endovascular stent-graft closure of the primary intimal tear (PIT) was introduced more than a decade ago as a treatment option for patients with complicated type B aortic dissection.1,2 Goals of stent-graft placement are to reestablish arterial flow to ischemic beds and to abolish pulsatile antegrade perfusion of the false lumen, which should reduce subsequent false lumen enlargement and rupture.3–8 Continued pressurization and perfusion of the false lumen due to endoleak, however, is frequently seen after stent-graft repair of aortic dissection.9,10 Compared with endoleaks encountered after thoracic endovascular aneurysm repair, endoleak physiology in aortic dissection is complex and incompletely understood.

**Clinical Perspective see p 112**

The first prerequisite for successful sealing of the PIT is a proximal landing zone of adequate geometry.11,12 It has long been observed that severe angulation and curvature in the abdominal aorta can result in endoleaks when treating aneurysms.13 Near the aortic arch, the geometry can be severely curved or angulated, rendering sealing of the PIT a challenge. Second, if the distance between the left subclavian artery (LSCA) and the PIT in the dissected aorta is short (less than 20 mm in length), coverage of the LSCA may be required to achieve an adequate seal.14 Coverage of large side branches by endovascular treatment of abdominal aortic aneurysms is associated with type 2 endoleaks, and a similar pathophysiology likely exists in the thoracic aorta.15,16 Inadequate seal or retrograde flow from branches or fenestrations can result in persistent false lumen flow and pressurization. The risk of subsequent false lumen enlargement and aortic rupture due to endoleak is not yet known. The aim of this study was to assess which anatomic and clinical conditions were associated with endoleaks in patients who underwent endovascular stent-graft repair of complicated type B aortic dissection and to examine the clinical consequences of these endoleaks.

**Patients and Methods**

Between January 2000 and November 2007, 37 patients underwent endovascular stent-graft repair of acute (≤14 days; n=23), subacute...
(15 to 90 days; n=10), or chronic (>90 days; n=4) complicated type B aortic dissections. Type B aortic dissections were judged to be complicated if 1 or more of the following 3 clinical problems was present: (1) symptomatic visceral or lower limb ischemia due to compromise of blood flow into branch vessels, including refractory, new, or worsened hypertension indicative of renal malperfusion; (2) false lumen rupture; or (3) pain with documented rapid aortic enlargement, interpreted to be impending rupture. Patients were excluded if arterial access was inadequate (<7-mm diameter of iliac or femoral arteries). Patients with connective tissue disorders (eg, Marfan disease) were preferentially treated by open surgical methods, and only 1 patient with Marfan disease and severe ischemia of multiple vascular beds was enrolled.

Endovascular repair was the default for all other patients. The Gore Thoracic Excluder with the longitudinal spines (n=17) or the redesigned and renamed “TAG” stent grafts (n=20)\textsuperscript{17} were used under an investigator-sponsored investigational device exemption protocol approved by the Food and Drug Administration and by our institutional review board. No abdominal cuffs or components were used. Informed consent was obtained from each patient or legal representative.

Patient evaluation and selection required consensus between the cardiothoracic surgeons and the interventional radiologists, and all procedures were performed collaboratively as previously described.\textsuperscript{18} All patients underwent helical computed tomographic angiography (CTA) before stent-graft placement to determine the location, length and diameter of the dissection and landing zones, and to evaluate the suitability of the iliac and femoral arteries for vascular access. Arterial access was gained either by inguinal or retroperitoneal exposure of the femoral or iliac artery and introduction of a 20 to 24F sheath. One hundred units per kilogram of heparin were administered in patients without frank rupture.

Transesophageal echocardiography was used intraoperatively to further characterize the anatomic relations between the true and false lumens, the PIT, and the branch vessels.\textsuperscript{1} Measurements from calibrated catheter aortography, transesophageal echocardiography, and CTA were used to select the device size. Early practice of using intravascular ultrasound was phased out as the quality of data from the other modalities improved.

Diameter was chosen according to the measurements of the nondissected aorta at the proximal landing zone, and length was chosen to be the minimum judged necessary to cover the proximal landing zone and extend at least 3 cm beyond the distal extent of the PIT. After the stent graft was deployed to cover the PIT, repeat aortography was performed to confirm the position of the device and to evaluate the size of and flow within the aortic lumens and branch vessels. Postdeployment balloon angioplasty was performed judiciously and only proximally in patients with acute dissections because of the fragility of the intimaf flap. Angioplasty was performed routinely in the patients with chronic dissections.

Adjunctive surgical or medical therapies included antihypertensive or vasopressor medications, resection of infarcted bowel, thoracostomy drainage, hemodialysis, and antibiotics as needed. For long-term management, every patient was placed on oral antihypertensive medications including at least β-blockers. Two patients required long-term anticoagulation for deep venous thrombosis, 1 of whom experienced a pulmonary embolism.

The follow-up protocol included clinical evaluation and multiple phase CTA with 3D reconstructions, performed at 1, 6, and 12 months after stent-graft placement, and annually thereafter. An additional scan was performed at 3 months if any issues were raised at the 1-month follow-up. Although not mandated in the protocol, postprocedural CTAs were also routinely performed within 1 week of surgery before patient discharge. In 3 patients, magnetic resonance angiography with gadolinium contrast enhancement was substituted for the CTA because of azotemia and/or allergy. Pathophysiology of any thoracic false lumen filling was analyzed retrospectively on postprocedural scans using multiplanar reformatting capabilities (AquariusNet, TeraRecon, Redwood City, Calif) to classify endoleak type (Figures 1 and 2). Six patients also underwent catheter angiography, in part to delineate pathophysiology and direction of flow in these endoleaks (Figure 3). In accordance with the classification system that exists for endoleak formation after stent-graft repair of abdominal aortic aneurysms, we classified type 1a leaks as persistent flow around an imperfect proximal seal and through the patent PIT, and type 2 leaks as false lumen perfusion involving retrograde flow through branch vessels such as the LSCA, bronchial, or intercostal arteries\textsuperscript{9} and/or flow through natural fenestrations and reentry tears. Endoleaks were classified as “complex” if both factors were present (Figure 1).

Three-dimensional reconstructions of the aortic arch based on CTA were also used to calculate the radius of the arch curvature in each patient. The aortic arch curvature was quantified by calculating the arithmetic mean of the lesser curve radius of curvature and the greater curve radius of curvature at the level of the PIT. Thus, smaller values of radius represent more severe aortic curvature (Figure 4). From these reconstructions, the adequacy of proximal apposition of the stent graft to the aortic wall was also assessed by measurement of the length of lack of apposition to the lesser curvature, sometimes termed the bird beak.

Primary technical success was defined as angiographic sealing of the PIT, with reperfusion of previously ischemic visceral or lower extremity arteries fed by the aortic true lumen if present. Primary end points were incidence of endoleaks and endoleak type in relation to LSCA coverage, aortic arch curvature, completeness of apposition, dissection chronicity, and device used. Secondary end points were necessity for reintervention, neurologic complications, and periprocedural mortality within 30 days. Clinically relevant baseline characteristics are reported with 95% confidence intervals. Continuous variables are expressed as mean±SD. Comparison of continuous variables before and after stent-graft placement was performed with Student t test. For comparison of dichotomous variables, the χ² test was used. P<0.05 was considered to indicate statistical significance. All statistical analyses were performed with SPSS version 10.0 (SPSS Inc, Chicago, Ill).

![Figure 1. Common endoleak pathophysiology after stent-graft repair of aortic dissection. A, Type 1a endoleak: imperfect seal at the proximal landing zone allowing blood to flow around the stent graft, through the PIT, and into the aortic false lumen. B, Type 2 endoleaks: persistent perfusion of the aortic false lumen through natural fenestrations, reentry tears, or retrograde flow from branch vessels. C, Complex endoleak, combining aspects of both type 1a and type 2. Flow in the LSCA and in the aortic false lumen may be antegrade, retrograde, or more complex.](image-url)
Results

Patient demographics are listed in Table 1. Stent-graft insertion was technically successful in all 37 (100%) patients (Table 2). No open surgical conversions were necessary at the time of initial treatment. Average follow-up was 28.3 months in those who survived beyond 30 days and 22.3 months overall (range, 0 to 92 months).

Intraprocedural type 1a endoleaks were observed in 4 acute patients and in 1 chronic patient. All were successfully treated by immediate proximal stent-graft extension. Postprocedural endoleaks were detected in 11 of 23 (48%) acute patients, 8 of 10 (80%) subacute patients, and 3 of 4 (75%) chronic patients, evenly distributed by type. There was a trend toward greater incidence of endoleaks in subacute and chronic patients, especially for type 2 leaks, that did not reach statistical significance (Table 3). There were no significant differences in endoleak formation between the earlier generation Thoracic Excluder and the redesigned TAG (Table 4).

Coverage of the LSCA was required in 20 of 37 (54%) patients in total, including in 13 of 23 (57%) acute patients, 4 of 10 (40%) subacute patients, and 3 of 4 (75%) of chronic patients. After coverage of the LSCA, the incidence of complex leaks was 35% (n=7), where none were seen without coverage of the LSCA (P=0.0005; Table 5). In addition, summing all patients with at least a component of type 2 leak (including type 2 and complicated endoleaks), coverage of the LSCA resulted in endoleaks in 55% of patients (n=11), compared with 18% (n=3) in those without coverage of the LSCA (P=0.017). Incidence of type 1 or type 2 endoleaks alone as a function of LSCA coverage did not reach statistical significance.

The radius of the aortic arch curvature in patients with type 1a endoleaks was 36±12 mm (mean, ±1 SD) versus 65±51 mm in those without leaks (P=0.05; Table 6). In patients with complex endoleaks, the radius was 37±14 mm, not quite reaching statistical significance (P=0.06). Including all patients with a component of poor proximal landing zone seal (type 1a and complex leaks), the radius of curvature was 36±13 mm (P=0.05). Pure type 2 endoleaks did not seem to be related to degree of aortic curvature.

The length of the bird beak varied from zero to 37 mm. The length was shortest in patients who did not develop endoleaks (6.7±8.8 mm) and longest in patients who formed complex type endoleaks (22.6±5.1 mm, P<0.0001; Table 7). Although patients with type 1a and type 2 endoleaks also had longer bird beaks than patients without endoleaks, these did not reach statistical significance. Length of the bird beak was weakly associated with the degree of curvature as defined as the reciprocal of the radius of curvature, with a Pearson coefficient r=0.53. The mean bird beak length without coverage of the LSCA was 9.3±11.8 mm, compared with 16.2±10.9 mm with coverage of the LSCA (P=0.08). Bird beak length tended to be longer with use of the newer TAG design (15.6±12.7 mm) when compared with the older Thoracic Excluder (9.6±9.6 mm), but this also did not reach statistical significance (P=0.13).

Six endoleaks resulted in false lumen enlargement and necessitated reinterventions. Interventions included embolization of the LSCA in 2 patients, surgical repair in 3 patients,
Emboliizations were performed 12 months, 4 months, and 1 week after stent-graft treatment in 3 patients, and were successful in 2. The third patient had a preexisting retrograde propagation of the dissection into the arch and a natural fenestration at the origin of the left common carotid artery, which did not resolve with embolization of the LSCA and adjacent aortic false lumen. This patient and 3 others underwent elective surgical repair at 8, 8, 6, and 3 months after stent-graft treatment, all of which were successful in removing endoleak-related dilatation.

Ten endoleaks resolved spontaneously during follow-up at 6.5±5.4 months after stent-graft treatment, including in 1 of 2 patients on long-term anticoagulation for deep vein thrombosis. Two endoleaks persisted but have not been subjected to reintervention because initial ischemia was rectified and false lumen diameter remained stable. Two patients with endoleaks died during their initial hospitalizations, both from multiorgan failure stemming from the limb, renal, and mesenteric infarction originally caused by dissection.

Two patients with type 2 endoleaks were patients with chronic dissections (14 months and 8 years from initial event) who presented with frank ruptures of false lumen aneurysms and expanding hemothoraces. Despite sealing of the PITs, angiography showed continued filling of the false lumen through abdominal natural fenestrations and reentry tears, and both patients died from inexorable hemorrhage and disseminated intravascular coagulation. Placement of large bare stents extending from the stent graft to the aortic bifurcation was performed in 1 patient but was not successful in eliminating false lumen perfusion.
Two strokes (5%) were observed, both in acute patients who underwent coverage of the LSCA. One had an intraoperative type 1a endoleak and immediately underwent proximal extension. The extension partially covered the left common carotid artery origin, requiring inflation of a trilobed balloon (Gore) within the stent graft and dragging the device distally. The other patient underwent an unremarkable procedure but a postprocedural CT scan of the brain showed widespread infarction in the anterior and posterior circulation distribution bilaterally, and care was withdrawn.

One patient (3%) developed postprocedural paraparesis of the left lower extremity. MRI of the spine revealed an ischemic spinal cord lesion from T3 to T8. A spinal drain was placed on postoperative day 2 immediately after recognition of the paraparesis. Neurologic deficits had not improved after 9 months.

Of the 20 patients who underwent coverage of the LSCA, only 3 developed left upper extremity ischemic symptoms. Two patients’ symptoms were mild and improved over time without reintervention, and 1 underwent successful carotid to subclavian bypass grafting.

Overall 30-day mortality was 7 of 37 (19%), representing 1 procedural complication (the stroke described earlier) and 6 failures to rescue. The failures included the 2 patients with ruptured chronic false lumen aneurysms described earlier. Two acute and 1 subacute dissection patients died due to visceral, renal, or extremity infarction and multiorgan failure, and 1 acute traumatic dissection patient died of other trau-

<table>
<thead>
<tr>
<th>Presenting symptoms*</th>
<th>Acute</th>
<th>Subacute</th>
<th>Chronic</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mesenteric ischemia</td>
<td>9 (39)</td>
<td>6 (60)</td>
<td>0</td>
<td>15 (41)</td>
</tr>
<tr>
<td>Renal ischemia</td>
<td>14 (61)</td>
<td>5 (50)</td>
<td>1 (25)</td>
<td>19 (51)</td>
</tr>
<tr>
<td>Extremity ischemia</td>
<td>6 (26)</td>
<td>1 (10)</td>
<td>0</td>
<td>7 (19)</td>
</tr>
<tr>
<td>Spinal cord ischemia/paralysis</td>
<td>2 (9)</td>
<td>0</td>
<td>0</td>
<td>2 (5)</td>
</tr>
<tr>
<td>False lumen rupture</td>
<td>6 (26)</td>
<td>3 (30)</td>
<td>2 (50)</td>
<td>9 (24)</td>
</tr>
<tr>
<td>Pain with false lumen enlargement (impending rupture)</td>
<td>9 (39)</td>
<td>6 (60)</td>
<td>2 (50)</td>
<td>17 (46)</td>
</tr>
<tr>
<td>Extension to type A</td>
<td>4 (17)</td>
<td>0</td>
<td>0</td>
<td>4 (11)</td>
</tr>
</tbody>
</table>

Data are presented as mean (range) or n (%).

*Percentages of presenting symptoms sum to more than 100% because of multiple ischemic beds or symptoms in many patients.
motic injuries. During follow-up that averaged 22.3 months in length, no stent-graft migration, fracture, dislocation, propagation from type B to type A, or new false lumen rupture was documented. One case of distal device flares eroding through the dissection flap and causing a localized false lumen ulcer was noted 22 months after the original surgery. This was accompanied by mild hemoptysis and was treated by surgical graft repair through a left thoracotomy, but the patient died postoperatively from complications of an arrhythmic arrest.

**Discussion**

Left untreated, 36% to 72% of thoracic aortic dissection patients die within 48 hours.\(^1\) Most cases of type B dissections are uncomplicated and can be managed by medical therapy, including the use of antihypertensive and negatively inotropic drugs. Dissections complicated by ischemia, rupture, or impending rupture have historically been treated by surgical graft techniques, with mortality rates as high as 87% in patients with multiple comorbidities and ischemic beds.\(^2\) Stent-graft repair has emerged as a minimally invasive alternative treatment with high initial technical success rates ranging from 90% to 100%, 30-day mortality rates between 6% and 16%, and encouraging midterm results.\(^1\) Despite the high rates of endoleak (which may not necessarily represent treatment failure), the morbidity and mortality rates reported herein continue to support stent-graft repair in these high-risk patients.

In addition, outcomes after stent-graft repair compare favorably with those of percutaneous fenestration and stenting,\(^3\) a technique effective at relieving ischemia but permissive of continued false lumen pressurization and dilatation. The technical goal of stent-graft treatment is closure of the PIT, but clinical success is defined according to the original clinical presentation. In patients with malperfusion, restoration of arterial flow to the ischemic beds is the primary clinical objective. In the latter group, and for both groups over the long term, endoleaks are an important issue. Endoleaks after thoracic endovascular aneurysm repair are well described and occur in 5% to 35% of patients,\(^4\) but endoleaks after treatment of dissections are strikingly different.

Endoleaks were detected in 59% of patients, much more than that reported in previous studies, reflecting several important distinctions. First, we used a multiple (3 to 5) phase CTA protocol, including an unenhanced phase, an arterial contrast-enhanced phase, and multiple (up to 3) delayed phases, with 3D reconstructions. Contrast medium was injected at 4 to 6 mL/s to maximize arterial enhancement. This protocol was specifically designed to detect endoleaks and is more aggressive than published techniques.\(^5\) Second, many early endoleaks were detected when imaging studies were performed postoperatively, before the mandated follow-up. Ten of these early endoleaks resolved spontaneously and would have been occult had we adhered to a less aggressive follow-up protocol. Excluding these leaks, our overall endoleak rate was 12 of 37 (32%). Third, the follow-up period was relatively long (up to 92 months), so the time at risk for endoleaks was greater in our population than in previous series. However, all but 1 documented endoleaks were detected within the first year of follow-up.

We found that patients with LSCA coverage had a significantly increased prevalence of type 2 and complex endoleaks, supporting thoracic endovascular aneurysm repair studies that found high rates of endoleak when the arch was involved.\(^6\) Dissection flaps that extend to or into the origin of the LSCA complicate the achievement of a seal using a simple cylindrical device. The physiology of blood flow at the origin of the LSCA can be extremely complex, with communication between aortic and LSCA true and false lumens, and directions of flow varying with different phases of the cardiac cycle. Future use of branched endografts should help to address this limitation.

<table>
<thead>
<tr>
<th>Endoleak Type</th>
<th>Acute Patients (n=23)</th>
<th>Subacute Patients (n=10)</th>
<th>Chronic Patients (n=4)</th>
<th>Combined Subacute/Chronic Patients (n=14)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1a</td>
<td>4 (17)</td>
<td>2 (20)</td>
<td>1 (25)</td>
<td>3 (21)</td>
<td>0.77</td>
</tr>
<tr>
<td>Type 2</td>
<td>3 (13)</td>
<td>3 (30)</td>
<td>2 (50)</td>
<td>5 (36)</td>
<td>0.11</td>
</tr>
<tr>
<td>Complex (both 1a and 2)</td>
<td>4 (17)</td>
<td>3 (30)</td>
<td>0 (0)</td>
<td>3 (21)</td>
<td>0.77</td>
</tr>
<tr>
<td>Total</td>
<td>11 (48)</td>
<td>8 (80)</td>
<td>3 (75)</td>
<td>11 (79)</td>
<td>0.07</td>
</tr>
</tbody>
</table>

Data are presented as n (%).

<table>
<thead>
<tr>
<th>Endoleak Type</th>
<th>LSCA Covered (n=20)</th>
<th>No LSCA Coverage (n=17)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1a</td>
<td>2 (10)</td>
<td>5 (29)</td>
<td>0.16</td>
</tr>
<tr>
<td>Type 2</td>
<td>4 (20)</td>
<td>3 (18)</td>
<td>0.86</td>
</tr>
<tr>
<td>Complex (both 1a and 2)</td>
<td>7 (35)</td>
<td>0 (0)</td>
<td>0.0005</td>
</tr>
<tr>
<td>Total</td>
<td>13 (65)</td>
<td>8 (47)</td>
<td>0.29</td>
</tr>
</tbody>
</table>

Data are presented as n (%).
Table 6. Incidence of Endoleak Formation and Endoleak Type in Relation to Degree of Curvature of the Aorta at the Level of the Primary Intimal Tear

<table>
<thead>
<tr>
<th>Endoleak Type</th>
<th>Radius of Curvature, mm</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No leak</td>
<td>65±51</td>
<td></td>
</tr>
<tr>
<td>Type 1a</td>
<td>36±12</td>
<td>0.05</td>
</tr>
<tr>
<td>Type 2</td>
<td>42±6</td>
<td>0.18</td>
</tr>
<tr>
<td>Complex (both 1a and 2)</td>
<td>37±14</td>
<td>0.06</td>
</tr>
<tr>
<td>Type 1a and complex</td>
<td>36±13</td>
<td>0.05</td>
</tr>
<tr>
<td>Type 2 and complex</td>
<td>40±12</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD.

The clinical impact of covering the LSCA is still controversial. Although upper extremity ischemia and vertebrobasilar insufficiency are uncommon and generally reversible, emerging statistics on stroke and paraplegia are raising concern.14–16 Both patients in our series who experienced strokes underwent coverage of the LSCA, and both strokes seemed embolic on MRI and involved the anterior circulation, suggesting that manipulation in the arch rather than compromise of left vertebral perfusion were to blame. In the EUROSTAR registry, coverage of the LSCA was not associated with stroke, but did increase the risk of paraplegia.34 Mortality was not affected by LSCA coverage. These data resulted in guidelines suggesting that LSCA coverage can be managed expectantly unless anatomic conditions such as diminutive right vertebral artery, or left internal thoracic artery to coronary bypass are identified.14,35 We continue to adhere to these guidelines for emergent cases, but preemptive carotid to LSCA surgical transposition or bypass has become our standard for elective cases.

The other factor contributing to formation of endoleaks is the limited capability of current devices to conform to acutely curved aortic anatomy, resulting in type 1a and complex endoleaks. Apposition of stent grafts to the lesser curvature of the aortic arch was generally incomplete and resulted in a bird beak defect. This shortcoming was independent of the generation of stent graft used, but future devices in development address this issue using more conformable and flexible designs. Limitations of this study includes its nonrandomized nature, limited numbers of subjects, and variety of chronicity and symptoms within the cohort. Even optimized imaging protocols do not always reveal direction of flow, compromising the detection, and classification of endoleaks. Furthermore, the data presented are from a single center, but reflect the performances of 10 different cardiovascular and thoracic surgeons and interventional radiologists of different degrees of experience.

In conclusion, these results confirm that stent-graft treatment of patients with complicated type B dissections can be safely performed with a very high initial rate of technical success. Factors that portend endoleak formation were related to involvement of the distal arch, including coverage of the LSCA, severe aortic curvature, and poor lesser curve apposition. Late false lumen dilatation necessitated reintervention in 16% of patients, all of whom demonstrated endoleaks. Because of flow through distal fenestrations and reentry tears, stent-graft treatment was inadequate in treating ruptured chronic false lumen aneurysms, a specific high risk group for whom substantial technological improvements are needed before stent-graft repair can become an acceptable treatment option.

Disclosures
Dr Sze is a consultant for MediGene Inc, Jennerex Biotherapeutics Inc, and Pain Therapeutics Inc, none of which are pertinent to this manuscript. Dr Dake is a consultant for W.L. Gore Inc. Dr Miller is a consultant for Medronic Heart Valve Division Inc and speaker for the St Jude Medical Residency Symposium. Dr Hofmann is on the Board of Directors of Endovention Inc, on the Scientific Advisory Board for Bacherus Vascular Inc, is a speaker for Cook Inc, and has received research grants from StemCells Inc and Siemens Medical Inc, none of which are pertinent to this manuscript.

References
11. Steingrubler IE, Czernak BV, Chemelli A, Gloyd B, Bonatti J, Jaschke W, Waldenberger P, Rieger M, Neuhauer B. Placement of endovascular...


**CLINICAL PERSPECTIVE**

Endoleaks after stent-graft repair of aortic dissections result in continued perfusion and pulsatility of the false lumen. The pathophysiology of these endoleaks differs from that of endoleaks after repair of aneurysms, reflecting the numerous natural fenestrations and re-entry tears typically present in a dissection outside of the stent-grafted zone. Using an aggressive high-sensitivity imaging protocol, we found evidence of endoleak in 59% of treated patients, but almost half of these resolved spontaneously. Of the remaining leaks, 27% led to delayed false lumen enlargement necessitating reintervention. In patients with dissections complicated by malperfusion, stent-graft repair resulted in a high rate of clinical resolution even in the presence of an endoleak. Thus, the definitions of technical and clinical success need to be amended in this patient population. In contradistinction, persistent perfusion of the false lumen after stent-graft repair in 2 patients with ruptured false lumen aneurysms from chronic dissections proved to be fatal. Occurrence of endoleaks was associated with coverage of the left subclavian artery, small radius of aortic curvature, and poor device apposition to the aortic lesser curve. High rates of endoleak should be expected when treating high-risk dissection patients, and long-term surveillance is particularly crucial in patients with documented endoleaks.
Factors Portending Endoleak Formation After Thoracic Aortic Stent-Graft Repair of Complicated Aortic Dissection

Daniel Y. Sze, Maurice A.A.J. van den Bosch, Michael D. Dake, D. Craig Miller, Lawrence V. Hofmann, Robin Varghese, S. Chris Malaisrie, Pieter J.A. van der Starre, Jarrett Rosenberg and R. Scott Mitchell

_Circ Cardiovasc Interv._ 2009;2:105-112; originally published online February 20, 2009; doi: 10.1161/CIRCINTERVENTIONS.108.819722

_Circulation: Cardiovascular Interventions_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 2009 American Heart Association, Inc. All rights reserved.

Print ISSN: 1941-7640. Online ISSN: 1941-7632

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://circinterventions.ahajournals.org/content/2/2/105

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation: Cardiovascular Interventions_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at: http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation: Cardiovascular Interventions_ is online at: http://circinterventions.ahajournals.org//subscriptions/