of Diet and Renal Disease equation (Kidney Intern 1999;55:1878-84). The medically treated patients' risk with high-grade ICA stenosis (>70%) for ipsilateral stroke at 2 years was higher in patients with CKD then in those with preserved renal function (31.6% vs 19.3%; $P = .042$). Carotid endarterectomy reduced this risk by 82% and 51%, respectively. Prevention of one stroke in terms of numbers needed to treat was 10 for patients with preserved renal function. However, the number needed to treat to prevent one stroke was only four in patients with CKD. Patients with CKD had similar rates of perioperative stroke and death but higher rates of perioperative cardiac deaths than patients without CKD.

Comment: Twelve years after its initial publication, the NASCET trial is still spinning off interesting—but very thinly sliced—additional pieces of salami. Patients with CKD can be inappropriately denied interventions because of perceived, but not proven, high risk. This phenomenon has been termed "renalism" (J Am Soc Nephro 2004;15:246.2468). The article is interesting because it not only reports patients with symptomatic high-grade ICA stenosis and CKD appear to drive significant benefit from endarterectomy but also raises the concept that patients with CKD, although at higher risk with certain procedures, may actually, in the long-term, derive increased benefit over those without CKD. Also, as the authors pointed out, large randomized trials should consider enrolling, rather than somewhat arbitrarily excluding, patients with CKD. CKD patients may actually derive unexpected and substantial benefit from selected procedures.

Collected World and Single Center Experience with Endovascular Treatment of Ruptured Abdominal Aortic Aneurysms

Veith FJ, Lachat M, Mayer D, and the RAAA Investigators. Ann Surg 2009;250:818-24.

Conclusion: In some patients, endovascular repair of ruptured abdominal aortic aneurysms (AAAs) has a lower procedural mortality at 30 days.

Summary: Endovascular aneurysm repair (EVAR) for ruptured AAA was first reported in the mid-1990s (Ann Surg 1995;222:449-65 and Lancet 1994;344:1645). Since then, EVAR for ruptured AAAs has been reported with varying results. Some authors have concluded that EVAR results in improved survival in patients with ruptured AAA, but others have reported no better results with EVAR than traditional repair. Also, historic controls of open repair results are often used to compare with modern results of EVAR. All reports are case-series. There are no randomized trials comparing EVAR and open repair in patients with similar anatomy and hemodynamic stability.

This article represents an attempt by the authors to summarize the literature with respect to endovascular treatment of ruptured AAAs. The authors examined a collective experience with use of EVAR to treat ruptured AAAs from 49 centers. Each center provided data in the form of answers to a questionnaire; in addition, a separate analysis was performed from 13 centers committed to EVAR treatment for ruptured AAA whenever possible.

Information was obtained on 1037 patients treated by EVAR and 763 patients treated by open repair. In the 13 centers performing EVAR for ruptured AAA whenever possible, EVAR was actually performed in a mean of 49.1% of patients (range, 28%-79%). The 30-day mortality in 680 patients treated with EVAR for ruptured AAA in these centers was 19.7% (range, 0%-32%). The 30-day mortality of the 763 patients treated with open repair was 36.3% (range, 8%-53%; $P < 0.0001$). Of the 1037 patients treated with EVAR for ruptured AAA, 30-day mortality was 21.2%. In the 13 centers using EVAR whenever possible, supraceliac aortic balloon control was obtained in 19.1% ± 12%. An abdominal compartment syndrome was treated by some form of decompression in 12.2% ± 8.3%.

Comment: One cannot argue with the conclusion EVAR has a lower procedural mortality in "at least some patients" and may be preferable for treating ruptured AAAs "provided that they (patients) have favorable anatomy; and adequate skills, facilities, and protocols are available, and optimal strategies, techniques, and adjuncts are employed." This is a classic "mom and apple pie statement." Whether or not it is correct or incorrect is actually relatively unimportant. For the foreseeable future, individual surgeons will need to make individual decisions for the treatment of ruptured AAA in individual patients. I do not agree with Dr Veith that performing a randomized trial of open vs EVAR for treatment of matched patients with ruptured AAA would be like performing a randomized trial on the use of parachutes. I do agree with Dr Veith that such a trial would be difficult to perform and that the performance of such a trial that provided results convincing to all would be nearly impossible.

Efficacy of Aggressive Lipid Controlling Therapy for Preventing Saphenous Vein Graft Disease

Hata M, Takayama T, Sezai A, et al. Ann Thorac Surg 2009;88:1440-4.

Conclusion: Aggressive lipid-controlling therapy may be effective in preventing saphenous vein graft disease after coronary artery bypass grafting (CABG).

Summary: About 25% of saphenous vein grafts occlude ≤1 year of CABG, and 50% occlude ≤ 10 years (Am Heart J 1990;119:1164-84). Despite their relative unfavorable natural history, saphenous vein grafts are still used in >70% of CABG procedures. It appears that plaque rupture with thrombus formation are a major cause of long-term saphenous vein graft disease after CABG (Circulation 2007;71:286-7). It also appears that lowering low-density lipoprotein cholesterol (LDL-C) <100 mg/dL may be effective in reducing atherosclerosis in saphenous vein grafts. In this study, the authors sought to investigate the efficacy of aggressive statin therapy on angioscopic-determined progression of saphenous vein graft disease after CABG.

There were 21 patients after CABG divided into two groups. Group I comprised 10 patients whose serum LDL-C levels and LDL/high-density lipoprotein (HDL) ratios could be controlled to <80 mg/dL and <1.5, respectively. Group II consisted of 11 patients whose LDL-C levels and LDL/HDL ratios were >100 mg/dL and >2.5, respectively. Twenty-seven saphenous vein grafts were assessed by intravascular ultrasound (IVUS) and angioscopy at 12 to 16 months postoperatively.

Serum LDL-C levels in group I were 64.1 vs 130.2 mg/dL in group II. LDL/HDL ratios in group I were 1.36 vs 2.64 in group II. High-sensitivity C-reactive protein in group I was 0.045 ± 0.1 vs 0.116 ± 0.02 mg/dL in group II. All values were significantly lower in group I. In group II, IVUS detected eccentric plaques in 11 of 14 saphenous vein grafts (78.6%). Yellow plaque was present in all 14 saphenous vein grafts by angioscopy, and 11 of these grafts had thrombi. The 13 saphenous vein grafts in group I had no eccentric or yellow plaques, and no thrombi were visible. The intima was entirely clear white.

Comment: The mechanism of failure of saphenous vein grafts in the coronary circulation may be different than that in the peripheral circulation. However, the idea that driving down LDL-C and C-reactive protein levels may improve vein graft patency is intriguing for the peripheral vascular surgeon as well. Indeed, there is evidence suggesting higher primary assisted and secondary patency in lower extremity vein grafts in patients treated with statins (J Vasc Surg 2004;39:1178-85). Well-designed prospective data are needed to assess the effects of statins on suppression of peripheral vein graft lesions.

Randomized Comparison of Strategies for Type B Aortic Dissection: The INvestigation of STEent Grafts in Aortic Dissection (INSTEAD) Trial

Nienaber CA, Rousseau H, Eggebrecht H, et al. Circulation 2009;120; 2519-28.

Conclusion: In survivors of uncomplicated type B aortic dissection, thoracic aortic stent grafts do not improve 2-year survival or adverse event rates despite favorable aortic remodeling.

Summary: Thoracic endovascular aortic repair (TEVAR) was introduced in 1999. The role of TEVAR in improving outcome in uncomplicated type B aortic dissection is unknown. Stable patients undergoing medical treatment with type B aortic dissection have an annual survival rate of >80%; however, aneurysm expansion and late complications do occur. Continued perfusion of the false lumen is a risk factor for adverse outcomes, and complete thrombosis of the false lumen has been associated with improved outcome (Ann Thorac Surg 2007;83:1059-66 and Eur J Cardiothorac Surg 2004;26:359-66). The authors sought to determine whether placement of thoracic aortic stent grafts might improve the prognosis in patients with stable type B aortic dissection.

There were 140 patients who were clinically stable for at least 2 weeks after an index type B aortic dissection. These patients were randomly assigned to receive either elective stent graft placement in addition to optimum medical therapy (n = 72) or to optical medical therapy alone (n = 68) with surveillance. Arterial pressure was treated according to World Health Organization guidelines (attempting to achieve blood pressure of 120/80 mm Hg). All-cause death at 2 years was the primary end point. Secondary end points were aortic-related deaths, progression (with need for conversion or additional endovascular or open surgery), and aortic remodeling.

There was no difference in all-cause deaths between the two groups. The 2-year cumulative survival was 95.6% ± 2.5% with optimum medical therapy vs 88.9% ± 3.7% with TEVAR ($P = .15$). Aortic-related death rate was not different ($P = .44$), and the risk for the combined end point of aortic-related death (rupture) and progression (conversion or additional endovascular or open surgery) was also similar ($P = .65$). There were three adverse neurologic events in the TEVAR group and one patient with transient paraparesis with medical treatment alone. Aortic remodeling (with true lumen recovery and false-lumen thrombosis) occurred in 91.3% of patients with TEVAR vs 19.4% of those who received medical treatment alone ($P < .004$).

Comment: Uncomplicated type B aortic dissection managed with tight blood pressure control and surveillance results in excellence survival rates. At present, TEVAR can be considered an appropriate crossover strategy if complications occur with a previously stable type B aortic dissection as, at least in this study, crossover patients uniformly survived deferred TEVAR. The study supports, at least in the short-term, a complication-specific approach to type B aortic dissection. Follow-up was relatively short in this study, but even with short follow-up, favorable aortic remodeling

with TEVAR was impressive. With longer follow-up, this may translate into improved survival with fewer long-term complications.

Stent Graft versus Balloon Angioplasty for Failing Dialysis-Access Grafts

Haskal ZJ, Trerotola S, Dolmatch B, et al. N Engl J Med 2010;362:494-503.

Conclusion: Revision of a venous anastomotic stenosis of a prosthetic dialysis access graft with a stent graft provides longer-term patency and freedom from repeat intervention than revision with standard balloon angioplasty.

Summary: Secondary patency of hemodialysis grafts is at best 50% at 3 years. Many interventions are typically required to maintain dialysis-access graft patency. The authors sought to test the hypothesis that revision of venous anastomotic stenosis with stent grafts constructed with the same material as the dialysis-access graft itself would improve long-term patency compared with that provided by revision with balloon angioplasty alone. Theoretically, stent graft revision would prevent elastic recoil associated with balloon angioplasty alone and prevent intimal hyperplasia in-growth at the venous anastomosis, resulting in improved patency of the revised grafts.

This was a prospective multicenter trial. There were 190 patients undergoing hemodialysis with dialysis-access grafts and a venous anastomosis stenosis that were randomly assigned to receive balloon angioplasty alone or balloon angioplasty plus placement of a stent graft at the site of the venous anastomotic lesion. Patency of the treatment area and patency of the entire vascular access graft were the primary end points.

At 6 months, patency of the treatment area was greater in the stent graft group than in the balloon angioplasty group (51% vs 23%, $P < .001$). Six-month patency of the dialysis access circuit was improved in the stent graft group vs the balloon angioplasty group (38% vs. 20%, $P = .008$). Freedom from subsequent intervention at 6 months was also greater in the stent graft group than in the balloon angioplasty group (32% vs 16%, $P = .03$). Restenosis was greater in the balloon angioplasty group than in the stent graft group (78% vs 28%, $P < .001$). Other adverse events at 6 months were equivalent in the two treatment groups.

Comment: There is still a need for dialysis-access grafts. Results of this study suggest stent grafts provide better patency in treating venous anastomotic strictures of dialysis-access grafts than that provided by balloon angioplasty alone. Although the results are statistically significant, there are details to be considered before declaring the results clinically significant. Grafts in this study were treated before actual thrombosis. Many access grafts do not come to revision until they have thrombosed, and it is controversial whether surveillance and treatment of patent, but not thrombosed dialysis grafts, actually results in overall prolongation of usable access. In addition, 6 months after revision with a stent graft, there is primary patency in only half the patients. Stent grafts are more expensive than balloon angioplasty alone. It is therefore unclear whether the increased patency at 6 months will translate into longer-term cost-effectiveness and whether an approach of graft surveillance with treatment with this technology of narrowed venous anastomotic areas discovered with surveillance will result in meaningful overall clinical benefit or cost savings.

White Blood Cell Count Predicts All-cause Mortality in Patients with Suspected Peripheral Arterial Disease

Arain FA, Khaleghi M, Bailey KR, et al. Am J Med 2009;122:874.e1-874.e7.

Conclusion: The white blood cell (WBC) count provides incremental information about risk of death in patients with peripheral arterial disease (PAD).

Summary: Inflammation is felt to play a central role in the development and progression of atherosclerosis. Markers of inflammation associated with higher rates of adverse cardiovascular outcome include C-reactive protein (CRP) and WBC count as well as lipoprotein-associated phospholipase A2. The authors postulated that increased plasma levels of CRP and lipoprotein-associated phospholipase A2, as well as the WBC count, would be associated with increased mortality in patients with suspected PAD referred to a vascular laboratory.

The study population was derived from 242 patients (54% men; mean age, 68 years) referred to a vascular laboratory. Ankle-brachial index (ABI) and inflammatory markers were measured at the start of the study. Fifty-six patients (25%) died in follow-up. Death was associated with increased age, history of cerebrovascular or coronary artery disease, increased creatinine level, lower ABI, increased WBC count, and increased CRP level. Patients in the top tertile of WBC count and CRP level had a relative risk of death of 3.37 (95% confidence interval, 1.56-7.27) and 2.12 (95% confidence interval, 0.97-4.62), respectively. Lipoprotein-associated phospholipase A2 was not associated with all-cause mortality. Only WBC count incrementally contributed to prediction of mortality. Inferences were the same when analysis was limited to patients with ABIs <0.9.

Comment: The data show indicators of systemic inflammation are associated with higher mortality in patients referred to a vascular laboratory. This study does not answer the question of whether WBC count is simply a marker of inflammation or has an independent pathologic role. The top tertile of WBC count in this study was defined $>7.9 \times 10^3/ml^3$; thus, even patients with so-called normal WBC counts and PAD are at increased risk of death. However, only about half of the patients were using antiplatelet or statin medications, and it is unclear in the patients using these medications over what proportion of the follow-up these medications were used. Before we start checking WBC counts in patients with suspected PAD, this study will need to be repeated with patients who are receiving aspirin and statin therapy.