ature on intra-abdominal reversible adverse outcomes and permanent adverse outcomes after DTA and TAAA open repair. This was a retrospective review of patients at the authors' institution who underwent open DTA and TAAA repair between January 2002 and December 2008. The authors included relevant preoperative, intraoperative, and postoperative data and performed a propensity score-matched analysis. Data sufficient to permit analysis were available for 240 of 262 patients, with 90 suitable for the propensity-matched study. Reversible adverse outcomes included analysis for renal failure, temporary hemodialysis, and liver failure. Permanent adverse outcomes identified were permanent hemodialysis, 30-day mortality, and paraplegia. The 30-day mortality was 7.1% (17 of 240). Reversible adverse outcomes developed in 40.8% of patients and permanent adverse outcomes in 10%. Propensity score analysis identified decreased odds of developing reversible adverse outcomes in patients undergoing DHCA (odds ratio, 0.32: 95% confidence interval, 0.12-0.85). Rates of acute renal failure (22% vs 46.4%, $P = .03$) and liver failure (17.8% vs 34.3%, $P = .04$) were lower in patients who underwent DHCA compared with the non-DHCA group.

Comment: Regionalization of complex procedures makes sense, and in vascular surgery, open thoracoabdominal surgery should be regionalized. Patients may not like to travel for care, but they do not want to die or become paralyzed as a result of their care either. Only a small minority of hospitals have the patient volume, technical expertise, and institutional resources to really do open TAAA surgery well. That number will be even smaller for those using DHCA to facilitate TAAA surgery. If DHCA is truly a useful adjunct in TAAA surgery, it is still another reason to argue for regionalization of TAAA surgery.

Combination Oral Antiplatelet Therapy may Increase the Risk of Hemorrhagic Complications in Patients With Acute Ischemic Stroke Caused by Large Artery Disease

Itabashi R, Mori E, Furui E, et al. Thromb Res 2011;128:541-6.

Conclusion: The incidence of hemorrhagic complications is likely increased in patients with acute ischemic stroke secondary to large artery disease who are treated with combination antiplatelet therapy.

Summary: It is recommended oral antiplatelet therapy be administered immediately after acute ischemic stroke to prevent recurrence and progression of stroke as well as to prevent other vascular events. It is known that monotherapy with aspirin does not reduce stroke progression (Markus HS et al, Circulation 2005;111:2233-40). However, it is thought that combination antiplatelet therapy, such as that provided by clopidogrel and aspirin, may have a role in reducing stroke progression in patients with stroke secondary to large artery disease (Wong KS et al, Lancet Neurol 2010;9: 489-97; Diener HC et al, Lancet 2004;64:331-7). There is an increased risk of hemorrhagic complications with long-term secondary preventative therapy with combination antiplatelet agents (Bhatt DL et al, N Engl J Med 2006;354:1706-17; Gasparyan AY et. al, J Am Coll Cardiol 2008;5:1829-43). The risk of combination antiplatelet therapy in the short-term treatment of acute stroke is not well understood. This retrospective study evaluated bleeding complications associated with antiplatelet therapy in patients with ischemic stroke secondary to large artery disease who were felt to be at high risk for stroke recurrence or progression. The authors reviewed 1335 consecutive patients admitted ≤7 days of an ischemic stroke or transient ischemic attack between April 2005 and November 2009. There were 167 patients with >50% stenosis or occlusion of a culprit major vessel treated with antiplatelet agents ≤48 hours of admission. Hemorrhagic complications were classified according to the bleeding severity index. Of the 167 patients studied, 108 were treated with combination antiplatelet agents and 59 with one antiplatelet agent. Three major and 11 minor hemorrhagic complications occurred in 14 patients. All major hemorrhagic complications occurred in patients administered combination antiplatelet therapy. The proportion of patients receiving combination agents was higher in those with significant hemorrhagic complications. Older age and receiving combination antiplatelet agents were independent predictors of a significant in-hospital hemorrhagic complication.

Comment: Despite the retrospective study design and the small number of end points, this study raises questions about the overall safety of combination antiplatelet therapy in the treatment of ischemic stroke caused by large artery disease. It is important to keep in mind that antiplatelet agents work through different mechanisms of action. It is therefore not surprising that the untoward effects of the sum may be greater than that observed with individual agents. This study will help determine appropriate statistical power for larger more definitive clinical trials with respect to end points and safety monitoring.

Defining Perioperative Mortality after Open and Endovascular Aortic Aneurysm Repair in the US Medicare Population

Schermerhorn ML, Giles KA, Sachs T, et al. J Am Coll Surg 2011;212:349-55.

Conclusions: Comparisons of in-hospital mortality overestimate the benefit of endovascular abdominal aortic aneurysm (AAA) repair (EVAR) compared with 30-day or combined 30-day and in-hospital mortality. The duration of highest mortality risk extends longer for open repair, and the total mortality impact of AAA repair is not realized until 3 months after repair.

Summary: Perioperative mortality is an important measure of quality for surgical procedures. Definitions of the "perioperative" period differ. Perioperative mortality may be defined as death during the initial hospitalization, ≤30 days of surgery, or all deaths ≤30 days plus any deaths >30 days that occur before hospital discharge. In addition, ongoing risks due to surgery may persist >30 days and beyond hospital discharge. The authors investigated the affects and implications of various methods of calculating "perioperative mortality" with respect to EVAR vs open AAA repair. They used propensity-scoring models to create matched cohorts of U.S. Medicare beneficiaries undergoing EVAR (n = 22,830) or open repair (n = 22,830) from 2001 to 2004. Perioperative mortality was calculated using in-hospital mortality, 30-day mortality, and combined 30-day and in-hospital mortality. The authors also calculated biweekly interval death rates for 12 months to define the duration of increased risk for death after AAA repair. In-hospital, 30-day, and combined 30-day and in-hospital mortality for open repair and EVAR were 4.6% vs 1.1%, 4.8% vs 1.6%, and 5.3% vs 1.7%, respectively. Absolute differences in mortality were similar at 3.5%, 3.2%, and 3.7%. Relative rates of death (95% confidence interval) were 4.2 (3.6-4.8), 3.1 (2.7-3.4), and 3.2 (2.8-3.5). Biweekly interval death rates were highest during the first month after EVAR (0.6%) and during the first 2.5 months (0.5% to 2.1%) after open repair. After 2.5 months, rates were similar for both repairs (<0.5%) and were stabilized after 3 months. The 90-day mortality rates were 7.0% for open repair and 3.2% for EVAR.

Comment: The main point here is not that AAA patients undergoing EVAR or open repair continue to experience procedure-related morbidity beyond their hospital stay or the first 30 postoperative days. We all have seen late procedure-related complications in our patients. However, it seems we, as surgeons, by focusing on 30-day and/or in-hospital morbidity and mortality, underestimate in the surgical literature the effect of our procedures on our patients. Perhaps, for procedures of greater magnitude, such as AAA repair, journal editors should encourage reporting of periprocedural morbidity and mortality data out to 3 months.

Microembolization During Carotid Artery Stenting in Patients With High-Risk, Lipid-Rich Plaque: A Randomized Trial of Proximal Vs Distal Cerebral Protection

Montorsi P, Caputi L, Galli S, et al. J Am Coll Cardiol 2011;58:1656-63.

Conclusion: In patients undergoing carotid artery stenting (CAS) who have a lipid-rich plaque, the use of a proximal cerebral protection system during CAS results in lower numbers of microemboli compared with distal cerebral protection with a filter wire during CAS.

Summary: It is well recognized distal cerebral protection during CAS does not fully prevent embolic complications. The reasons for this are unclear but may include unprotected crossing of the lesion, incomplete apposition of the device to the arterial wall, emboli smaller than the filter pore size, and loss of debris with filter recapture. Proximal endovascular occlusion using balloons to occlude the external carotid artery and the common carotid artery, resulting in blood flow arrest in the internal carotid artery during CAS, may be a better technique for providing cerebral protection. Although the proximal occlusion technique has several potential drawbacks, including patient intolerance to occlusion, potential dissection of the external carotid artery, and need for a larger sheath (8F/9F), the potential to decrease microembolic events during CAS is intriguing. Increased numbers of microembolic signals (MES) detected with transcranial Doppler (TCD) have been associated with a greater prevalence of silent ischemic cerebral lesions detected on post-CAS magnetic resonance diffusion-weighted imaging (DWI). The significance of silent DWI-detected ischemic lesions after carotid intervention is unknown, but there is some suggestion they could be associated with late cognitive decline. Patients with lipid-rich plaque appear also to be at higher risk because they have an increased prevalence of DWI-detected lesions after CAS. The authors studied 53 consecutive patients with lipid-rich plaque undergoing CAS who were randomized to a proximal protection device (n = 26) or distal protection (n = 27) with a filter wire. MES were assessed with TCD during crossing of the lesion, before dilation, stent crossing, stent deployment, stent dilation, and during device retrieval/deflation. DWI was performed before CAS and after CAS at 48 hours and 30 days. Patients treated with the proximal cerebral protection device had a higher rate of ulcerated plaque than those whose cerebral protection was provided with a filter wire (35% vs 7.4%; $P = .019$). Compared with the filter wire, the proximal cerebral protection device reduced the mean number of TCD-detected MES during crossing of the lesion (18 vs 2, $P < .001$), stent crossing (23 vs 0), stent deployment (30 vs 0), stent dilation (16 vs 0), and the total number of MES detected (93 vs 16). Multivariable analysis showed the type of cerebral protection was the only independent predictor of total MES. There was no significant difference in the number of patients with new post-CAS embolic lesions in the