Predictors of morbidity and mortality with endovascular and open thoracic aneurysm repair

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Background: Open and endovascular thoracic aneurysm repairs are associated with significant complications including paraplegia, stroke, vascular insufficiency, and death. Predictors of adverse outcomes are not well-defined in this patient population.

Methods: The database of the GORE TAG (W.L. Gore, Flagstaff, Ariz) Pivotal Trial comparing the TAG endograft to open repair was interrogated. Univariate (UVA) and multivariate analyses (MVA) of demographic, clinical, anatomic, and procedural variables were conducted to discover possible predictors of serious adverse events for the whole group and for the TAG and open cohort groups separately. Early adverse outcomes occurred within 30 days or the initial hospitalization. *P* value of ≤ .05 was significant.

Results: A total of 140 TAG and 94 open descending thoracic aneurysm (DTA) patients were analyzed, consisting of 128 men and 106 women. Perioperative deaths were 9/94 for open surgery and 3/140 for TAG patients, with 10/12 (7 open, 3 TAG) deaths occurring in men. Two female deaths were both after open surgery. Multivariate analysis showed predictors of death for all patients were symptomatic aneurysms and male gender. Analysis of a combined morbidity/mortality endpoint (stroke/paralysis/MI/death) showed elevated creatinine predicted these events for the whole group. Open surgery (*P* < .001) and increasing aneurysm diameter (*P* < .001) predicted an increased likelihood of any major adverse event. Open surgery was significantly associated with an increased risk of paraplegia (*P* = .002). Vascular complications were more frequent in the TAG (19%) than in open DTA patients (9%) (*P* = .038). Female gender (*P* = .01) predicted vascular complications within the endovascular group. For all analyses, long procedure times were correlated with adverse events. Women were noted to have longer procedure times for both TAG and open repairs.

Conclusion: Elevated creatinine levels and symptomatic aneurysms predict morbidity and mortality, respectively, regardless of repair type. Male gender predicted death after open surgery, and since most deaths (9 of 12) were in this group, male gender predicted death overall, despite women's more difficult endovascular TAA repairs as evidenced by longer procedure times and higher vascular complication rates. All major adverse events and paraplegia were more common for open surgery patients. (J Vasc Surg 2008;48:1114-20.)

With an aging population and new treatment options available, repairs of descending thoracic aneurysms (DTA) are likely to become more common.[1-4] Both open and endovascular repair of descending thoracic aneurysm have been shown to have multiple and serious complications.[5-7] There are few predictors of these adverse events, for open or thoracic endovascular aneurysm repair (TEVAR), making patient education and selection difficult.

Until recently, the Gore TAG stent graft (W.L. Gore, Flagstaff, Ariz) was the only Food & Drug Administration (FDA)-approved thoracic stent graft for treatment of DTA. We examined patients treated with the TAG device as well as the open surgery cohort for the Phase II TAG clinical trial to determine if there were demographic or intraoperative factors that could predict the primary endpoint of death within 30 days. Other endpoints included the combined outcomes of death, paraplegia, stroke, or myocardial infarction. We also sought to determine predictors of vascular complications and of long-term graft-related events such as migration, endoleak, or conversion. All analyses of immediate and long-term complications and possible predictors of major adverse events (MAEs) are new to this work. Similarly, this is the first work to examine graft-related complications in a large population of open and endovascular patients to try to determine predictors of late interventions so that this information could be used in patient selection.

METHODS

The Gore TAG pivotal trial was a multicenter, prospective, non-randomized Phase II study that recruited surgical candidates with DTA from September 1999 through May 2001. Treatment modality (open or endovascular) was determined by the surgeon based on patient characteristics and availability of endovascular grafts. One hundred forty endovascular patients were enrolled. Ninety-four patients with DTA treated by open surgery were used as a control group. Of the 94 open surgical control patients, 44 were concurrent subjects and 50 were historic controls from the enrolling institutions. Details on the two populations have been previously published.[8,9] Briefly, patients with DTA of at least twice the diameter of the normal thoracic aorta and with 2 cm of non-aneurysmal neck for sealing distal to the left carotid artery and proximal to the celiac artery were

eligible for endovascular treatment. TAG devices ranged from 26 to 40 mm in diameter. All devices in this analysis were the original TAG endograft available prior to the revision which eliminated the longitudinal spine and replaced the fabric with a low-porosity material. Open repair was performed according to local protocols at the participating institutions. The extent of the open repair could not extend more proximally than the left carotid artery or more distally than the celiac axis. There were no mandates regarding use of spinal cord protection or use of left heart bypass. High-risk patients, including those with dissection, rupture, mycotic aneurysm, or trauma were excluded, as were medically high risk patients.[8,9] Follow-up exams, 4-view chest x-rays and spiral CT scans were performed at 1, 6, and 12 months and yearly thereafter. These exams were performed at 3 months if an endoleak was present. Five-year follow-ups were concluded for all available patients in August of 2006.

The 5-year results of the Gore TAG pivotal trial were analyzed for predictors of morbidity and mortality. Preoperative demographic and anatomic characteristics were used as independent variables to predict intraoperative results, postoperative complications and long-term outcomes. Univariate (UVA) and multivariate analyses (MVA) were conducted to discover possible predictors of major adverse events. Major adverse events (MAEs) as defined per Sacks criteria,[10] were reported by the study sites and verified by clinical events coordinators. MAEs were those that resulted in a prolongation of treatment, new hospitalization, major disability, or death. Major and minor adverse events were adjudicated by a clinical events committee. Perioperative deaths were those deaths which occurred in the hospital or within 30 days of the initial procedure. Pre and postoperative DTA measurements and endoleak assessments were performed at the study sites. Significant sac size change was defined as ≥5 mm change of the largest diameter of the aneurysm from the baseline 1-month measurement. Migration was defined as a graft shift of ≥10 mm either cranial or caudal compared to the 1-month baseline. Spine fractures, seen in 20 patients in the original TAG study group prior to the revision of the product, were not counted as material or device failures in this analysis due to the acknowledged problem with the product causing the fractures rather than with patient characteristics as a causative agent. Endoleaks were infrequently seen with spinal fractures[9,11] and, therefore, were counted as late graft-related events.

Due to a low frequency of events, the most serious complications of TEVAR and open DTA repair were combined for an endpoint of death, myocardial infarction (MI), stroke, or paraplegia. These events were also examined separately. Patients were analyzed by treatment group and as an entire cohort for predictors of major adverse events.

All variables with a *P* value of .10 or lower in univariate analysis were included in multivariate analyses. Multivariate analysis modeling was assessed using the Hosmer-Lemeshow test for goodness of fit. Early adverse outcomes occurred within 30 days or during the initial hospitaliza-

Table I. Variables used as predictors of adverse events including death

Variable
Treatment group (TAG vs open surgical controls)
Age (years)
Race (White vs other)
Gender
Height (cm)
Weight (kg)
History of coronary artery disease (yes/no)
History of angina (yes/no)
History of myocardial infarction (yes/no)
History of cardiac arrhythmia (yes/no)
History of congestive heart failure (yes/no)
History of stroke (yes/no)
History of peripheral arterial occlusive disease (yes/no)
History of vascular intervention (yes/no)
History of arterial and/or venous thromboembolic event (yes/no)
Symptomatic aneurysm (yes/no)
Smoking history (yes/no)
ASA Class (I–IV)
Creatinine value (mg/dL)
Preoperative hematocrit value (%)
Significant patent intercostal arteries (yes/no)
Maximum proximal aortic diameter (mm)
Maximum distal aortic diameter (mm)
Aneurysm diameter (mm)
Proximal aortic neck length (cm)
Aneurysm length (cm)
Distal aortic neck length (cm)
Right and left common iliac diameter (mm)
Right and left external iliac diameter (mm)
Procedural blood loss (mL)

tion. Predictors of late device-related events at 5 years were analyzed for the TAG group only. A *P* value of ≤ .05 was considered significant. Table I lists variables used as predictors of adverse events.

RESULTS

A total of 140 TAG patients (60 women, 80 men) and 94 open patients (46 women, 48 men) were analyzed. Details of demographic comparisons between the populations have been previously published.[8] The only significant differences in the preoperative characteristics or presentations of the patients was that there were significantly more symptomatic aneurysms in the open surgical group (36/94 [38%] vs 30/140 [21%], *P* = .007). The designation of "symptomatic" was made by the treating surgeon without standardized criteria for this status. The percentage of patients with a history of coronary artery disease in the TAG group was slightly higher (49% TAG vs 36% open) and this trended toward significant (*P* = .06). With these exceptions, there were no other notable preoperative differences.

Mortality. Perioperative deaths were 3/140 (2.1%) for TAG patients and 9/94 (9.6%) for open procedures (*P* = .01 in univariate analysis). Details on the perioperative deaths have been previously reported.[8,9,11] The three TAG deaths were from stroke, cardiac arrest, and sepsis. The 9 open repair deaths were due to respiratory failure (n = 4),

Table II. Univariate analysis of predictors of death for open and endovascular DTA repair (n = 234)

Variable	n	P value	Odds ratio	Lower CI	Upper CI
Hx symptomatic aneurysm	234	.004	0.177	0.046	0.583
Treatment group	234	.012	4.835	1.398	22.247
Creatinine value	203	.013	6.809	1.546	31.073
Male gender	234	.031	0.227	0.034	0.886
Increasing height	233	.045	1.061	1.001	1.131
Increasing aneurysm diameter	224	.077	1.037	0.996	1.078
Increasing weight	233	.088	1.028	0.996	1.061
Left external iliac diameter	144	.133	1.459	0.891	2.380
Max proximal neck diameter	181	.192	1.075	0.961	1.184
Hx embolic event	234	.231	0.337	0.078	2.325
Hx vascular intervention	234	.256	2.0	0.611	7.668
Hematocrit	204	.259	0.924	0.807	1.061
Hx PAD	234	.282	0.451	0.126	2.118
Right common iliac diameter	155	.297	0.854	0.618	1.101
Left common iliac diameter	154	.328	0.840	0.532	1.084
Right external iliac diameter	144	.361	1.182	0.793	1.587
Max distal aortic diameter	179	.366	1.065	0.928	1.204
Distal neck length	162	.397	0.926	0.728	1.086
Hx arrhythmia	234	.407	1.852	0.471	12.273
Hx CHF	234	.417	0.495	0.119	3.365
ASA Class	234	.425	1.468	0.588	4.051
Hx stroke	234	.450	0.522	0.127	3.544
Age	234	.517	1.020	0.964	1.095
Hx CAD	234	.669	0.776	0.236	2.551
Hx angina	234	.677	1.523	0.279	28.379
Hx MI	234	.699	0.763	0.217	3.541
Aneurysm length	173	.717	1.024	0.888	1.155
Proximal aortic neck length	166	.842	0.980	0.763	1.157

Max, maximum; *Hx,* history; *CI,* confidence interval; *PAD,* peripheral arterial disease; *CHF,* congestive heart failure; *CAD,* coronary artery disease; *MI,* myocardial infarction; *ASA,* American Society of Anesthesiology.
Shaded variables did not meet the $P \leq .1$ threshold for inclusion in the multivariable model.

stroke (n = 3), cardiac causes (n = 1), and aorto-enteric fistula (n = 1). Ten of 12 deaths were in men (7 open, 3 TAG) and two were in women (both open surgery DTA repairs). Patients who were categorized as presenting with a symptomatic aneurysm had a 12% death rate (8/66) vs a 2% death rate (4/168) in non-symptomatic patients ($P = .004$ in univariate analysis). The results of the univariate analysis for predictors of death in the full TAG/open surgery cohort are presented in Table II. Even though procedure time was initially found to be significantly associated with death, this was not included in the multivariate analysis as it was thought to be a marker for difficult surgery rather than a causative agent for morbidity or mortality. Additionally, it was also strongly associated with the type of repair, with open surgery having predictably longer operating times. The multivariate analysis for predictors of death in the entire cohort showed only symptomatic aneurysms ($P = .004$) and male gender ($P = .048$) were significant.

Morbidity. A composite endpoint of death/stroke/MI/paraplegia was constructed to try to predict the factors causing the most serious adverse outcomes. Of 17 patients who suffered spinal cord ischemia, 6 died, all in the open surgery cohort. Table III shows the incidence of these events for the entire cohort and by treatment group. Variables were screened in univariate analysis for both the group as a whole and for the treatment groups separately. The results of the univariate analysis are shown in Table IV. The

Table III. Incidence of death/stroke/MI or paraplegia

	TAG (n = 140)	Open control (n = 94)	Total (n = 234)
Composite	11 (7.9%)	17 (18.1%)	28 (12%)
Death	3 (2%)	9 (10%)	12 (5%)
Stroke	5 (4%)	4 (4%)	9 (4%)
MI	1 (0.7%)	1 (1.1%)	2 (0.9%)
Paraplegia	4 (3%)	13 (14%)	17 (7%)

MI, Myocardial infarction.
Note: Patients may have had more than one complication.

multivariate model was run using the listed variables as well as including a variable for treatment group ($P = .006$ in univariate analysis for entire cohort). On MVA, only elevated creatinine ($P = .002$) and treatment group ($P = .006$) predicted combined morbidity endpoint in the entire cohort of DTA repair patients. The predictive value of elevated creatinine is of questionable validity in the open surgery group as many of the historic controls did not have a preoperative creatinine value available. Mean creatinine in TAG patients with the composite endpoint was 1.54 mg/dL vs 1.06 mg/dL in patients without the composite endpoint. For open surgery patients this difference was less pronounced at 1.18 mg/dL with the composite endpoint and 1.09 mg/dL without. Treatment group appears to be

Table IV. Significant predictors of the composite endpoint of death/stroke/MI/paraplegia by treatment group in univariate analysis, separated by treatment group

	Variable	*n*	*P value*	*Odds ratio*	*Lower CI*	*Upper CI*
TAG	Increasing creatinine value	139	.001	11.38	2.77	60.54
n = 140	History of angina	140	.052	0.25	0.07	1.01
	Proximal aortic neck length	136	.064	0.83	0.63	1.01
Open Surgery	Increasing height	94	.024	1.06	1.01	1.11
n = 94	History of smoking	94	.056	0.19	0.01	1.04
	Increasing age	94	.070	1.05	0.10	1.13

CI, Confidence interval.

the best predictor of the combined adverse events. Analysis of myocardial infarction and stroke as separate events did not yield significant predictors, partially due to the low frequency of these events.

Analysis of paraplegia alone showed that in univariate analysis, treatment group ($P = .002$), preoperative hematocrit value ($P = .060$) history of arrhythmia ($P = .059$), and presence of patent intercostal arteries ($P = .091$) were significant. In multivariate analysis, only treatment group was significant ($P = .002$). Univariate analysis of the individual groups failed to show any significant predictors of paraplegia to the 0.05 level in the surgical control group. In the TAG group, decreasing weight was predictive of paraplegia/paresis ($P = .013$), but with so few patient affected ($n = 4$), this finding should be interpreted with caution.

An analysis of predictors of any MAE was conducted for both TAG and open surgery groups. Perioperatively, 28% of TAG and 70% of open DTA patients had at least one MAE ($P < .001$).[11] At 5 years, there was still a significant difference in the occurrence of MAEs, with 57.9% of TAG and 78.7% of open patients having an MAE at any point (log rank $< .001$).[11] In the open group UVA, increasing aneurysm diameter was found to predict any adverse event ($P = .02$). For TAG patients, a low preoperative hematocrit value predicted MAEs ($P = .03$), with increasing creatinine value ($P = .057$) and increasing aneurysm diameter ($P = .082$) trending toward significant. In MVA, using the entire cohort, open surgery treatment group ($P < .001$) and increasing aneurysm diameter ($P < .001$) predicted a significantly increased incidence of any major adverse event.

When asymptomatic aneurysm patients were analyzed as a separate cohort, univariate analysis in the open surgery group showed a history of smoking predicted any MAE. Multivariate analysis demonstrated a high creatinine value ($P = .005$) and short proximal neck length ($P = .009$) predicted events in the endovascular group. Multivariate analysis for all non-symptomatic DTA patients was significant for treatment group (open surgery predicting any MAE) ($P = .01$). Meaningful analysis of the asymptomatic group for risk factors for death was not performed due to the low incidence of this event.

Vascular complications of intraoperative hemorrhage, thrombosis, or acute ischemia were seen in 19% of TAG patients and 9% of open surgical patients ($P = .038$). Male patients had a 10% risk of perioperative vascular events

Table V. Graft-related events at 5 years

Event	*30 (21%)* **
Branch vessel occlusion*	4 (3%)
Deployment failure*	1 (<1%)
Lumen obstruction*	1 (<1%)
Device complication at treatment*	4 (3%)
Endoleak (total number of patients with endoleak at any time)	15 (10.6%)
Increase in diameter ≥5 mm	8 (19%)
Prosthesis migration	1 (<1%)
Prosthesis material failure	1 (<1%)
Extrusion/erosion	0 (0%)
Aneurysm rupture	0 (0%)
Other complication at follow-up	2 (1.4%)

*Intraoperative event at initial procedure.
**Patients may have had more than 1 event.

(13/128) vs a 20% rate in women (21/106) ($P = .01$). Results of the univariate analysis demonstrated that decreasing weight ($P = .007$), endovascular treatment ($P = .027$), decreasing iliac diameter ($P = .034$), female gender ($P = .037$), increasing age ($P = .038$), and symptomatic aneurysms ($P = .046$) were all predictors of vascular complications. However, when put into a multivariate analysis, missing data from the open control group (iliac diameters) prevented a reliable model from being constructed for the entire cohort. Within the TAG treatment group only, gender ($P = .01$) was found to predict vascular complications. Age, body-mass index (BMI), iliac diameter, and symptomatic aneurysm were not found to have a predictive value. Longer procedure times were again found to be correlated with adverse events. TAG procedure times were significantly longer for women at a mean of 180 minutes (range, 65-580) vs 129 minutes (range, 61-290) for men ($P = .001$).

Graft-related events. Graft-related events at 5 years were examined and analyzed for predictive factors. Table V lists these events from the operative period through long-term follow-up. The operative events have been previously described.[9] The number of patients with an endoleak at any time was 10%, and at 5 years of follow-up 4.3% of patients had an active endoleak. A detailed description of the endoleaks, treatment, and all graft-related events in follow-up has been released.[11] In brief, 3 patients have undergone additional extension procedures for endoleak. Nineteen

percent of patients have been noted to have sac enlargement at 5 years of follow-up, and no interventions have been performed for this. One instance of proximal migration was noted and was most likely due to a poor seal at the original procedure. This patient underwent successful conversion. Two additional complications during follow-up were infections due to aorto-esophageal fistula. Both of these patients died as a result of this condition. Analysis of the TAG cohort failed to demonstrate any predictive variable for these events, or for graft events in general.

DISCUSSION

This is the first attempt to define predictors of morbidity and mortality in a combined open and endovascularly treated population of patients treated for descending thoracic aneurysms. We found that symptomatic aneurysms and male gender predicted death in the entire cohort of DTA repair patients. Although the multivariate analysis did not demonstrate a treatment group effect for mortality, this was thought to be related to a confounding effect of symptomatic aneurysms. The negative predictive effect of symptomatic aneurysms is not difficult to understand, even though none of these patients were thought to have an acute rupture. The lack of patient optimization in urgent surgery coupled with the potential hemodynamic instability and inflammatory cascade involved in an acute presentation is known to translate into worse outcomes.[12,13] The fact that significantly more symptomatic aneurysms were found in the open treatment group does have a confounding effect with the treatment group. In univariate analysis, symptomatic aneurysms ($P = .004$) and open repair ($P = .01$) both predicted death. When constructing the multivariate model, symptomatic aneurysms had more weight due to the lower P value, and this, in part, negated the effect of treatment group. The strong predictive effect of male gender, however, is more surprising. There is ample literature describing worse outcomes after infrarenal abdominal aortic aneurysm (AAA) repairs in women vs men,[14-19] and women were noted in our series to have longer procedure times and a higher incidence of vascular complications. We attribute both the longer procedure times and the higher number of vascular complications to smaller iliac arteries found in women. Although there was an association with longer procedure times and all complications, this did not translate into increased mortality in women.

The predictor of serious morbidity in the entire cohort was found to be an elevated creatinine level, although no patients were in renal failure. There is strong evidence that even subtle elevations in creatinine can translate into adverse perioperative events.[20,21] However, this finding is more meaningful in the TAG group as data for the open surgical patients did not always include creatinine values. We interpret these results to indicate that in patients with even subtle elevations in creatinine renal dysfunction may be a marker for systemic vasculopathy and that these patients should be warned that they may have a higher incidence of complications. Although procedure time was found to have a significant predictive effect for many adverse events, the authors determined that this was a marker for difficult surgery rather than a risk factor in and of itself. Additionally, long procedure times were strongly correlated with open surgery and would have altered comparisons within the entire DTA cohort, falsely biasing analyses in favor of the TAG group.

Open surgery and larger aneurysms were found to predict the occurrence of any adverse event in follow-up, and open surgery was the only reliable predictor of paraplegia in the entire DTA cohort. These associations are not surprising, but do lend weight to the argument in favor of TEVAR. This is especially compelling in that adverse events, including endoleaks, and sac enlargements were tabulated out to 5 years. The finding that even with late graft-related events there are significantly fewer MAEs with endovascular DTA repair illustrates that this is a durable procedure and less morbid than open surgery in appropriate patients. Unfortunately, no reliable predictors could be determined for late graft-related events.

The strengths of this study are that data was prospectively collected, and all complications have been verified by independent practitioners and a clinical events committee. There are a relatively large number of patients involved, although even with 234 patients we were unable to perform many important analyses due to the low number of index events or missing data points. Missing data points were primarily seen in the open surgical patients, especially historic controls. These restrictions lead to the formation of the composite morbidity point in order to be able to predict the most serious perioperative events, as each separate event happened too infrequently.

Unfortunately, this population was not randomized to open vs endovascular repair – a study that will most likely never be completed. We know that there were more symptomatic aneurysm patients in the open repair group, and this fact had a profound effect on the overall conclusions of our analysis due to the high death rate within this population. Although there was marginally more cardiac disease within the TAG group, we did not see significant differences in postoperative cardiac events between the 2 groups.

CONCLUSION

Morbidity and mortality within the TAG and open surgery cohorts were low for DTA repair. Symptomatic aneurysms and male gender were found to predict death overall, and both of these variables were found more often in the open surgery group. There was increased morbidity associated with an elevated creatinine for the entire cohort. Vascular complications were seen more often in women and with endovascular repair. Open surgery predicted any major adverse event and paraplegia.

AUTHOR CONTRIBUTIONS

Conception and design: ED
Analysis and interpretation: ED, MS
Data collection: ED, MS
Writing the article: ED

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DISCUSSION

Dr Jeffrey P. Carpenter (Philadelphia, Pa). Drs Dillavou and Makaroun have enlarged our understanding of thoracic aortic repair by identifying some preoperative risk factors for morbidity and mortality after both open and TEVAR [thoracic endovascular aneurysm repair] approaches using the TAG [W. L. Gore and Associates, Flagstaff, Ariz] database. They have identified a number of risk factors, which unfortunately cannot be modified, that predict bad outcomes: gender, height, renal function, presentation, and age, among those.

I would ask, therefore, what is our take-home message regarding patient selection for open or endovascular repair from these data? How would you reduce these findings to actual practice? Can you tell us a little more about the endoleak rate of 10.6%? Are these mostly type II leaks, or are they attachment or junction leaks? Also, could you comment on that sac expansion rate of 19%. Is that endotension as we saw with the abdominal excluder, or is this a different problem with the TAG? What have your investigators done about that issue, and what is recommended?

Also, I am fascinated by your finding in the manuscript, which you provided to me, that the short proximal neck length predicted perioperative morbidity. Perhaps that is a modifiable risk factor. What are the practical implications of this short neck finding related to morbidity?

And then, finally, in light of the finding of prospective randomized trials of abdominal aneurysm repair vs open or endovascular means, specifically that there is no long-term mortality benefit to EVAR when compared with open aneurysm repair. Payment has been denied for EVAR in parts of Europe. EVAR is under very careful scrutiny by payers in this country as well.

Now, I know the TAG database is not a randomized study of open vs endovascular repair of thoracic aneurysms, but it remains the best available data set we have for comparison between open and endovascular groups. Do you have any glimpse of the long-term comparison of mortality between those groups? Does the mortality benefit in the perioperative period that you noted extend to the longer term?

Dr Dillavou. Thank you, Dr Carpenter. Going through the questions one by one, you ask an excellent question about what is the take-home message from these data. I agree that some factors cannot be changed. However, I believe that we can use this information to educate patients and to enhance patient selection. Some proactive measures can also be taken. For instance, if we know that women have a higher chance of vascular complications after TAG endografting, perhaps planning a conduit is the better choice rather than just deciding to forge through.

You ask about the endoleak rate of 10.6% at 5 years. We found that most of these were attachment site leaks, and they were fixed relatively easily over time. Only three patients have had to undergo conversion, two were for aortoenteric fistulas and one was for proximal migration

and dilation of the proximal neck zone. None of the conversions were for endoleaks. All were able to be treated percutaneously.

The sac expansion rate of 19% at 5 years is something that deserves attention. A few patients had endoleaks, but the vast majority of them did not. We attribute sac expansion to the same forces and porosity that we see in the abdominal group. All of these TAG grafts that were put in were the standard expanded PTFE [polytetrafluoroethylene] grafts.

Comparing these grafts to the low-porosity TAGs showed differences that did not achieve statistical significance; at 2 years in the low-porosity group, we saw a 3% sac expansion vs a 13% sac expansion in the regular group. We hope that the low-porosity graft will decrease sac expansion. Thus far, no action has been taken on these patients if no endoleak is found.

You ask about short proximal neck length related to perioperative morbidity. We have found that with carotid–subclavian bypass, there is a statistically higher incidence of stroke after endografting. This is true whether or not the carotid–subclavian bypass was done preoperatively or immediately postoperatively. We hypothesize that this is due to a more diseased arch and more manipulation. All strokes were perioperative events. This is something to take into consideration when planning the operation and educating the patient.

Finally, you ask about our results and compared them to the EVAR I and II trials done in the United Kingdom. We found that there was a significant aneurysm-related survival difference, with a higher aneurysm-related survival in the thoracic endograft group compared to the open surgical group. However, looking at overall mortality, there was no difference between the groups at 5 years. So there is a difference in aneurysm mortality, and it persists to 5 years.

Dr Keith D. Calligaro (Philadelphia, Pa). Dr Dillavou, very nice presentation. I know this was retrospective and you can't really match up the patients. The question is whether patients who underwent open surgical repair did so because they did not have an adequate neck proximal or distal? If there wasn't an adequate landing zone for an endograft, more of the patients undergoing open repair might require clamping above the subclavian or below the viscerals and that alone would increase the morbidity.

Dr Dillavou. No. The clamp site limits were basically the same for both groups, that the proximal clamp could not have been placed more proximal then the left carotid artery and the distal clamp could not be placed more distally than the celiac trunk. So the clamp sites were the same. Some of these were historic controls that were done prior to the availability of the endovascular graft.

Dr Maciej L. Dryjski (Buffalo, NY). I have a question regarding the mortality. How would you explain the higher mortality in the symptomatic group of patients? Is it perhaps that the symptoms were not related to the aneurysm itself, but rather some other medical condition?

Dr Dillavou. To make sure I understand your question, you are asking if I believe that the increased mortality in the symptomatic aneurysm group was because of hemodynamic instability and presentation?

Dr Dryjski. Were these patients hemodynamically unstable, or were the symptoms related to some other condition, not necessarily an expanding aneurysm?

Dr Dillavou. These were not hemodynamically unstable patients. There was no one with a rupture or a leak or anything like that. These were patients who presented, whom the surgeon felt were having pain or other symptoms due to the aneurysm, and in most cases they were done more urgently than a traditional planned aneurysm repair. It is my hypothesis that part of the reason that these patients have an increased mortality is because that their operations probably were less electively planned. We did not find any difference in operating room blood loss or other intraoperative factors, hematocrit values, things like that.

Procedure times were slightly longer for symptomatic vs non-symptomatic aneurysms, but there again, that just may be related to the more difficult nature of the anatomy. There is no concrete reason why the symptomatic aneurysm group had such a dramatic increase in mortality, but my hypothesis is just that the patients were not optimized prior to their repairs. Also, some of these patients may not have been offered elective surgery due to anatomic or medical factors, but because they presented symptomatically they underwent a repair.

Dr Marat Goldenberg (Reading, Pa). I have a two-part question. You said that in women the operative time was longer. Question number 1, do you think this is due to the smaller access vessels or unique characteristics of the aneurysm? Do you think they have higher level of complication because of the longer time under anesthesia or the degree of difficulty?

Dr Dillavou. That's an excellent question. I believe that the longer procedure times were probably related to smaller access vessels and also to complications that were encountered in the operating room. More than half of the vascular complications that we saw were actually dealt with at the time of the procedure—iliac artery ruptures, femoral artery occlusions, et cetera—and so this obviously adds to procedure time. It is my belief that this, the difficult access, is the cause and the long procedure time is the effect, although it is impossible to know for sure.

That is something that we struggled with statistically in this database, and that is that long procedure times, no matter what we were analyzing, long procedure times predicted bad outcomes. But we also saw predictably longer procedure times with open surgery and in patients with complications, which all sort of goes together. And so it is my hypothesis that the long procedure times were an effect rather than the cause of the morbidities.