1. Perioperative mortality and morbidity were significantly less with an endovascular approach.
2. Spinal cord ischemia was significantly less in the endograft cohort.
3. The overall stroke rate was similar in both the endograft and open surgical control cohorts.
4. The reintervention rate and continued presence of complications, such as endoleaks, is higher in the endograft group. The presence of endoleaks can lead to future complications, but their significance is still unclear.
5. There was no survival advantage associated with either strategy after 2 years of follow-up.

As technology continues to improve and we as surgeons progress along the learning curve, the long-term complications of endografting might or might not be mitigated. Therefore continued vigilant surveillance of patients treated with endovascular repair is important.

References


Discussion

Dr Joseph S. Coselli (Houston, Tex), Scott, congratulations on an outstanding presentation and for bringing this information from a multicenter clinical trial to us. I believe that your report will be a seminal investigation into an evolving technology that stands to forever alter the way we therapeutically approach descending thoracic aortic pathology. Although I believe that aortic stenting is here to stay, we must shun the pressure of industry-driven initiatives and pursue good science and good medicine with, of course, industry support. We need to shoulder the responsibility of being the patient’s primary advocate.

You and the coauthors importantly infer the problems associated with a nonrandomized multicenter trial. I continue to have problems with the control group. Most of the control subjects, 53%, were historically and retrospectively acquired. Not all institutions contributed patients to this cohort. Data on aortic characteristics were unavailable in many of the open reconstruction control patients. Proximal and distal aortic diameters and aneurysm length, for example, were reported in less than 35% of this cohort; even aortic aneurysm diameter data were missing in 10%. The data support that the open repair group did not end up with more advanced disease because they had larger aortic diameters and were more likely to be symptomatic.

After endograft repair, 17% of the patients had expansion of their aneurysm of greater than 1/2 cm over 2 years. Considering the need for life-long monitoring after endograft repair, especially in the setting of a research protocol, incomplete 2-year follow-up of 14% is concerning. Do you think that 2 years really is enough?
The extent of aorta replaced and the location (ie, proximal or distal or the entire descending thoracic aorta) are related to morbidity, primarily stroke and paraplegia. Were comparisons made between these 2 groups accounts for these?

Seventy-eight percent of the open repairs had extracorporeal support. Was this need for cardiopulmonary bypass and hypothermic circulatory arrest suggestive of extensive disease and associated with increased morbidity or mortality?

One patient died of an aortoesophageal fistula. What do you believe the nature of that particular fistula was, how did it occur, and what lessons do you think were learned?

The incidence of paraplegia and paraparesis after endovascular repair was 5% in patients with prior AAA repair. What was the incidence of paraplegia or paraparesis after previous AAA repair in the open group?

The incidence of stroke in the treatment group was 4%. With the need to traverse the aortic arch with a stiff wire and, for proximal aneurysms, even advance the deployment device into the arch, do you and the authors see a need for transesophageal echocardiography to evaluate the arch for mobile atherosclerotic disease before implementation of the device?

Using a 30% increase in baseline creatinine level to define “renal dysfunction” as a cutoff point captures patients with clinically insignificant increases. Therefore what was the incidence of postoperative dialysis in the 2 groups?

The study excluded patients with recent myocardial infarction or recent stroke, renal insufficiency, and respiratory insufficiency, and interestingly enough, these are the patients in particular who might benefit the most from endovascular repair.

Once again, congratulations.

Dr Mitchell, Thank you, Dr Coselli. I will try and answer as many of your questions as I can remember.

First, is 2 years enough? Absolutely not. We do not know the exact hazard function, but I think these complications will be ongoing, hopefully decreasing with time, but we do not know that. Therefore these patients will require lifelong follow-up.

The question of the control group has been an energized discussion. It is not the best control group, we admit that, but it is the only one that we had, and I think all of us are aware of the difficulties in trying to get a very aware public to enroll in a randomized trial.

I cannot answer about the incidence of paraplegia in the open and control group as relates to previous abdominal aneurysm repairs.

As relates to cardiopulmonary bypass, that was used primarily as an adjunct for each individual site in their routine repair of descending thoracic aneurysms, and, in theory, circulatory arrest and hypothermia were not supposed to be used for these patient populations because they were supposed to be clampable.

Finally, we do agree that transesophageal echocardiography is an invaluable adjunct for the anesthetic management of these patients to look at the arch. There is no question that any manipulation in the arch does predispose this patient population to stroke, even as little as a stiff guide wire, and certainly, having to put your sheath through the arch increases that risk even more.

Dr Coselli, Scott, one quick follow-up. You and your group at Stanford have the longest and probably the largest experience with this particular technology. Would you just comment on your thoughts regarding connective tissue disease, particularly Marfan’s syndrome, applying this approach?

Dr Mitchell, I think you noticed that patients with Marfan’s syndrome were specifically excluded from this, and I would continue to urge that to be an exclusion with the exception of replacing some remnant aorta between 2 Dacron segments. Therefore if you are connecting Dacron to Dacron, I think that would be okay. Otherwise, I would be very pessimistic that this would be effective.

Dr Michael C. Maxwell (Mesa, Ariz). The Achilles’ heel of endoluminal grafting is the endoleak, and I noticed you had a 15% incidence. I talked to other investigators for this graft in the thoracic position, and endoleak, particularly type I, seems to be more common than it is in the abdominal position. Is that something you have also noticed, and if so, is it something that can be watched, unlike in the abdominal position, or does it have to be taken care of when identified?

Dr Mitchell, No, I think type I endoleaks should be managed on detection. Type II and III endoleaks perhaps can be followed, looking at aneurysmal sac size as a surrogate. But we have been very aggressive about trying to eliminate all type I endoleaks.

There was just one question I forgot to answer for Dr Coselli, that there were some aneurysm enlargements that were unassociated with endoleaks. This is the so-called endotension, which did occur with the old graft because it was thinner. The new revised graft has a stouter polytetrafluoroethylene column, and we do not think that these transmembrane leaks will occur, and hopefully this phenomenon will go away.