New treatment approaches create new disease processes: A short guide on how to reduce unexpected events to a minimum

Martin Czerny, MD, MBA

From the University Heart Center Freiburg-Bad Krozingen, Bad Krozingen, Germany.

Disclosures: Author has nothing to disclose with regard to commercial support.

Received for publication Oct 6, 2018; accepted for publication Oct 8, 2018.

Address for reprints: Martin Czerny, MD, MBA, Albert Ludwigs University Freiburg-Faculty of Medicine, University Heart Center Freiburg-Bad Krozingen, Südring 15 79189 Germany (E-mail: martin.czerny@universitaets-herzzentrum.de).

J Thorac Cardiovasc Surg 2019;157:107-8

0022-5223/\$36.00

Copyright © 2018 by The American Association for Thoracic Surgery

<https://doi.org/10.1016/j.jtcvs.2018.10.027>

Martin Czerny, MD, MBA

Central Message

Understanding the underlying disease process and anticipating interaction between a stent-graft and the native aortic tissue will reduce the remaining risk of dSINE to a minimum.

See Article page 101.

When thoracic endovascular aortic repair was introduced 2 decades ago, enthusiasm was great and open surgery was already deemed futile. As with each and every new technology, the hype flattened as obvious (endoleaks, continuing dilation of landing zones) and nonobvious limitations of the technique became known.¹

One of the initially nonobvious limitations was that retrograde type A aortic dissection, the knowledge of which had to develop over time, was due to interaction of the still rigid stent-graft and the highly elastic aortic wall, where the effect was even more pronounced the more proximal the deployment was done.² We had to learn that particularly in patients with the underlying diagnosis of type B aortic dissection, despite regular diameters, the tissue texture of the proximal thoracic aorta (root, ascending and arch) is not comparable to that of patients with descending aortic aneurysms or penetrating atherosclerotic ulcerations.³

A comparable phenomenon was observed distally with the same underlying mechanism being an unfavorable interaction between a stent-graft and the aortic wall, or better, the dissection membrane leading to membrane rupture.⁴ This process, called “distal stent-graft–induced new entry (dSINE),” functionally creates a new primary entry tear mostly leading to rapid expansion and therefore needs treatment by endovascular extension or open surgery, usually allowing the stent-graft left in place, provided there is no type 1 or 3 endoleak, where the proximal anastomosis usually can be performed directly to the distal end of the stent-graft.⁵

As knowledge developed, several components leading to the development of dSINE became clear: the underlying pathology per se, the size of the stent-graft(s) used, and the time interval between the acute event of aortic dissection and the time of treatment, where shorter intervals seem to be associated with less risk to develop dSINE provided there is no connective tissue disease and stent-graft sizing

is done correctly. We also learned to calculate the size of the thoracic aorta before acute aortic dissection in both type A and type B scenarios, which helps to anticipate the correct size of the stent-graft.^{6,7} Still, the time point of the occurrence remains largely unpredictable.

Finally, it should be mentioned that dSINE not only is an unexpected natural event but also can be intentionally induced for the creation of a distal landing zone in postdissection aneurysmal formation in highly selected cases. This approach is still experimental, and its efficacy has to be confirmed.

Because the perception of dSINE in the cardiovascular community is still in its infancy, the current report provides a highly useful comprehensive summary of etiology, incidence, and treatment options, but, and this might be the most important component, creates awareness and underlines the need for lifelong follow-up in all patients with thoracic aortic pathologies.⁸

References

1. Dumfarth J, Michel M, Schmidli J, Sodeck G, Ehrlich M, Grimm M, et al. Mechanisms of failure and outcome of secondary surgical interventions after TEVAR. *Ann Thorac Surg*. 2011;91:1141-6.
2. Eggebrecht H, Thompson M, Rousseau H, Czerny M, Lönn L, Mehat RH, et al; on behalf of the European Registry on Endovascular Aortic Repair Complications. Retrograde ascending aortic dissection during or after thoracic aortic stent-graft placement—insights from the European registry on endovascular aortic repair complications (EuREC). *Circulation*. 2009;120:S276-81.

3. Czerny M, Weigang E, Sodeck G, Schmidli J, Antona C, Gelpi G, et al. Targeting landing zone 0 by total arch rerouting and TEVAR – mid-term results of a transcontinental registry. *Ann Thorac Surg.* 2012; 94:84-9.
4. Janosi RA, Tsagakis K, Bettin M, Kahlert P, Horacek M, Al-Rashid F, et al. Thoracic aortic aneurysm expansion due to late distal stent-graft induced new entry. *Catheter Cardiovasc Intervent.* 2015;85:E43-53.
5. Jassar A, Kreibich M, Morlock J, Kondov S, Schrumann J, Kari FA, et al. Thoracoabdominal aortic replacement after thoracic endovascular aortic repair with large stent-grafts: diameter correction with modified use of the Vascutek® Siena™ collared branched graft. *Ann Thorac Surg.* 2018; 105:587-91.
6. Rylski B, Blanke P, Beyersdorf F, Desai ND, Milewski R, Siepe M, et al. How does the ascending aorta geometry change when it dissects? *J Am Coll Cardiol.* 2014; 63:1311-9.
7. Rylski B, Munoz C, Reser D, Schoenhoff F, Carrel T, Eckstein HH, et al. How does descending aorta geometry change when it dissects? *Eur J Cardiothorac Surg.* 2018;53:815-21.
8. Hughes GC. Stent graft–induced new entry tear (SINE): intentional and NOT. *J Thorac Cardiovasc Surg.* 2019;157:101-6.