Stent graft–induced new entry tear (SINE): Intentional and NOT

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The term stent graft–induced new entry (SINE) was originally defined by Dong and colleagues in 2010 as “a new tear caused by the stent graft itself, excluding those created by natural disease progression or any iatrogenic injury from the endovascular manipulation.” Although the term SINE was originally coined in 2010, the issue of stent graft–induced intimal injury was recognized a decade earlier in a report by Ninomiya and colleagues, who described the development of a new “ulcer-like projection” at the distal end of a thoracic stent graft 14 months after implantation in a patient treated for chronic type B dissection (Figure E1). Since these early reports, a large amount of data has been published regarding the incidence, etiology, and outcomes of SINE after thoracic endovascular aortic repair (TEVAR), which will be reviewed in this manuscript. In addition, more recently, some authors have intentionally created intimal tears to allow the stent graft to approximate to the outer aortic wall with the goal of achieving more extensive aortic remodeling, and the limited data available regarding these “intentional SINE” techniques will be briefly reviewed as well.

SINE INCIDENCE

In their sentinel report in 2010, Dong and colleagues noted that the occurrence of SINE is most common after TEVAR performed for an aortic dissection indication. The authors described both proximal SINE, leading to retrograde type A dissection or proximal pseudoaneurysm formation, and distal SINE, resulting in false lumen pressurization and expansion. The reported incidence of SINE after TEVAR varies but may be as high as 25%, with distal SINE being more common and representing up to 80% or more of SINE in some series. Further, the complication of distal SINE has been increasingly reported, with multiple publications in the literature since 2010. SINE are typically delayed in occurrence, with most developing approximately 12 to 36 months post-TEVAR. They are usually asymptomatic and discovered on routine postoperative surveillance imaging, a point that highlights the need for lifelong imaging surveillance follow-up in this population. Reintervention is usually required due to progressive aortic enlargement, which most commonly entails open surgery in the case of the less frequent proximal SINE (Figure 1) and distal extension TEVAR for distal SINE (Figure E2; Table 1).
SINE RISK FACTORS AND ETIOLOGY

A number of risk factors have been associated with the occurrence of SINE (Table 1). Foremost, distal SINE appears to be more common after TEVAR performed for chronic type B aortic dissection. The morphology of the dissected aorta changes over time, with the intimal flap thickening and becoming less mobile. As such, the dissected intimal flap cannot reapproximate to the outer aortic wall after TEVAR in the chronic setting, leading to persistent radial force exerted by the distal compressed stent graft, which may lead to SINE at the distal end of the device over time (Figure E3). Conversely, after TEVAR for acute dissection, the intimal flap is highly mobile, thereby allowing the distal end of the stent graft to fully expand such that the dissection flap may return to its previous anatomic position adjacent to the outer aortic wall.

The most important risk factor for distal SINE appears to be excessive oversizing of the distal stent graft relative to the smaller true lumen. This is mainly applicable to the chronic setting, where the compressed true lumen assumes a crescent shape that can make sizing for device selection more challenging. Previous work has demonstrated that the diameter of the true lumen in the distal thoracic aorta of patients with chronic dissection is approximately one third of the normal diameter in healthy controls, and therefore when endografts are sized only to the proximal nondissected true lumen in the aortic arch, as has been the standard for acute dissection, this results in an endograft that is markedly oversized (>60%) in comparison with the distal true lumen.

This marked oversizing leads to greater radial forces that can then induce intimomedial membrane disruption, as the distal landing zone is entirely within dissected aorta and the

![Image](image_url)

**FIGURE 1.** A, Preoperative 3-dimensional computed tomography angiography reconstruction image demonstrating the proximal bare springs of a thoracic endograft eroding into the wall of the aortic arch with the development of a proximal SINE/arch pseudoaneurysm (arrow). The device was placed for an acute complicated type B dissection 7 years earlier. Note the “spring back” apparent in the device configuration, similar to that described in Figure 2, with the early development of a distal SINE/pseudoaneurysm as well. B, Intraoperative photograph from the same patient showing the open aortic arch with bare springs embedded into the aortic wall (arrow). C, The arch was resected up to the level of the proximal end of the endograft and the proximal bare springs removed from the device. D, The arch was then replaced with a branched Dacron graft with the distal arch anastomosis incorporating the aortic wall and proximal end of the old endograft.
fragile dissection membrane is more prone to injury. This may be especially problematic in patients with connective tissue disorders in whom the aorta has pathologic fragility even in the absence of previous dissection, and several studies have suggested the incidence of SINE to be greater in patients with Marfan syndrome. For example, in their sentinel report in 2010, Deng and colleagues noted a 10-fold greater incidence of SINE in patients with Marfan as compared with patients without Marfan syndrome after TEVAR for type B dissection and suggested trying to avoid the use of TEVAR in this population. Likewise, in a systematic review of the results of TEVAR for type B dissection in patients with Marfan syndrome, Pacini and colleagues noted a substantial risk of surgical conversion and death at midterm follow-up and suggested caution against the routine use of endovascular stent grafting in patients with Marfan syndrome.

Self-expanding TEVAR grafts have a tendency to “spring back” to their initial straight status if passively bent, such as when placed across the aortic arch (Figure 2). This spring-back strength may be especially prominent in devices with longitudinal connecting bars that prevent twisting and kinking but sacrifice flexibility. This spring-back force on the distal end of the stent graft may then lead to distal SINE. Li and colleagues demonstrated that stent grafts with a length <145 mm were associated with a greater risk of distal SINE, potentially due to the leverage effect of the lesser curve of the arch on the device, as shorter devices have greater spring-back force on their distal end.

Although one might hypothesize that the risk of SINE should vary based on device type, eg, stainless-steel, nitinol, presence or absence of connecting bar, etc, SINEs have been observed with all devices, and there is insufficient evidence in the literature to definitively suggest superiority of one device over another with regard to SINE incidence at the current time. Likewise, the currently available TEVAR devices also vary in the radial forces they exert on the aortic wall, and this characteristic of the individual devices may also contribute to SINE occurrence. However, only very limited data are available on this topic, and additional work examining all commercially available devices and sizes is needed to further elucidate the potential impact of this aspect of device design on SINE occurrence.

**SINE PREVENTION**

SINE prevention is important, as their occurrence may lead to pseudoaneurysm formation or retrograde dissection proximally and false lumen repressurization distally, both of which result in therapeutic failure. As SINE are most common at the distal end of a thoracic endograft implanted in the setting of chronic type B dissection, the following are means to minimize their occurrence. Most importantly, and unlike what has traditionally been done when sizing devices for acute dissection (where the device size is chosen based on the diameter of...
The “distal-first” implantation technique as used by our group for thoracic endovascular aortic repair (TEVAR) for chronic dissection. An 84-year-old patient is shown who presented with a large distal arch aneurysm secondary to chronic residual dissection 8 months after previous supracoronary ascending aorta and hemiarch replacement for acute type A dissection at another institution. He underwent first-stage redo-sternotomy for 3-vessel arch debranching, and the video shows his second-stage completion TEVAR procedure performed several days later during the same hospital stay. The beginning of the video shows an intravascular ultrasound (IVUS) probe being used to interrogate the entire aorta to confirm true lumen wire location throughout (IVUS images not shown). The devices are then deployed from distal to proximal in the true lumen, beginning just above the celiac axis with a 31/26 mm tapered device deployed distally, followed by a 34 mm × 20 cm device more proximally. Following a proximal marker arteriogram to roadmap the origin the arch debranching graft and proximal landing zone, a 40 mm × 20 cm device is deployed. The completion angiogram demonstrates no endoleak, and the 1-month follow-up 3-dimensional reconstructed computed tomography angiography image demonstrates a good result with a thrombosed aneurysm sac and thoracic false lumen. The patient continues to do well now nearly 1 year after his 2-stage type I hybrid arch repair with no endoleak and significant shrinkage of his distal arch aneurysm sac. Video available at: https://www.jtcvs.org/article/S0022-5223(18)32821-6/fulltext.

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The “Knickerbocker technique” consists of proximal TEVAR to cover the primary entry tear with a second oversized endograft deployed more distally. A large compliant balloon is then inflated in the distal endograft to rupture the dissection membrane and allow the endograft to expand outward to reach the outer aortic wall and prevent retrograde false lumen flow back up into the more proximal thoracic false lumen (Figure 3). The so-called stent-assisted balloon-induced intimal disruption and relamination technique was first reported in 2014 and involves proximal TEVAR to cover the primary entry tear with distal extension bare metal stents along the length of the distal flap followed by balloon-induced disruption of the distal intimal flap beginning in the distal third of the stent-graft and extending along the length of the uncovered dissection stents so as to recreate a single flow channel throughout the thoracoabdominal aorta (Figure 3). Finally, some authors have used various techniques to create endovascular longitudinal fenestrations in the intimal flap, so-called “intimal flap septostomy,” again with the goal of creating a distal landing zone in which the stent-graft may be apposed to the outer aortic wall.

In summary, SINE is an increasingly reported complication of TEVAR for chronic type B dissection that may lead to therapeutic failure or need for reintervention. A number of techniques have been proposed to limit its occurrence, including distal first endograft deployment with devices sized to the distal true lumen. However, most of the research to date involves single-center reports with small patient numbers, and more work is needed to understand the long-term implications of SINE and its treatment. Further, even with SINE avoidance, thoracic and abdominal growth may still occur in certain cases despite seal of the proximal entry tear, and several novel techniques have been proposed in an attempt to overcome this limitation of TEVAR for chronic dissection. Additional work is likewise needed, however, to assess these techniques in expanded patient populations with the hope that TEVAR may become effective in treating all patients with aneurysmal dilation secondary to chronic type B dissection.

Webcast
You can watch a Webcast of this AATS meeting presentation by going to: https://aats.blob.core.windows.net/media/18AO/27-br-1345-hughes-v2.mp4.
Conflict of Interest Statement
Author has nothing to disclose with regard to commercial support.

References

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FIGURE E1. Sagittal reconstructed computed tomography angiography image from the sentinel report of stent graft–induced intimal injury published in 2002.1 The image demonstrates a new “ulcer-like projection” (arrow), which developed at the distal end of an early generation thoracic stent graft 14 months after implantation in a patient treated for chronic type B dissection. Reproduced from Ninomiya and colleagues with permission.

FIGURE E2. A. Angiogram and B, 3-dimensional reconstructed computed tomography angiography images demonstrating a distal stent graft–induced new entry (arrows), which was treated with distal extension thoracic endovascular aortic repair (C and D). Modified from reference Jánosi and colleagues with permission.
FIGURE E3. Serial 3-dimensional reconstructed computed tomography angiography reconstruction images demonstrating a distal compressed stent graft in the small true lumen after thoracic endovascular aortic repair for chronic dissection (A and B). C, Over time, the persistent radial force of the compressed endograft leads to distal stent graft–induced new entry (arrow). Modified from Jang and colleagues with permission.

FIGURE E4. Curved planar reformat computed tomography angiography reconstruction measuring the mean diameter of the true lumen in the distal landing zone in the distal descending thoracic aorta. The distal thoracic endovascular aortic repair device is then chosen such that there is minimal oversizing of the proposed distal landing zone so as to prevent distal stent graft–induced new entry occurrence. Reproduced from Hughes and colleagues with permission.
FIGURE E5. Concept of retrograde false lumen pressurization via downstream fenestrations. In most cases of DeBakey type IIIb aortic dissection, there are multiple downstream fenestrations that continue to provide flow to the false lumen despite adequate coverage of the proximal primary tear and even with avoidance of stent graft–induced new entry creation. These downstream re-entry tears are especially common in the visceral segment, where they are located at the prior ostia of branch vessels, which now arise from the false lumen such that a channel connecting the true and false lumens at this level exists. Retrograde flow from the abdominal aorta back up into the false lumen of the thoracic aorta is generally only seen when there are patent intercostal or bronchial arteries arising from the false lumen to allow continued flow. This may also be seen when the left subclavian artery either partially or fully arises from the false lumen to allow continued retrograde flow with false lumen pressurization. Reproduced from Rohlffs and colleagues20 with permission.