

Stent Strut Thickness: Have We Reached the Minimum?

The role of thin-strut designs in the current generation of stent technology and the impact on clinical outcomes.

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Low strut thickness is an important device feature put forward by manufacturers to promote the potential superiority of any new drug-eluting stent (DES). However, there is a paucity of data in the literature with respect to the benefits of thin stent struts in the DES era. The so-called second-generation DES simultaneously incorporated significant changes in the three DES components: new limus, new polymers, and thinner struts. Thus, their superiority over first-generation DESs cannot solely be attributed to changes in strut size.

WHEN DID THIN-STRUT STENTS COME INTO THE LIMELIGHT?

The thin-strut stent narrative was initiated in 2001, when Kastrati et al randomized two generations of bare-metal stents that had similar alloys and designs: one with a strut thickness of 50 μm and a strut width of 100 μm , and the other at 140- μm thick with a similar width. This angiographic study, ISAR-STEREO,¹ demonstrated a significant reduction in binary restenosis and reinterventions in the thin-strut group, despite a better acute gain in the thick-strut stent group. This study prompted a debate, given that changes in stent design between the two versions resulted in higher radial force and metal-to-artery ratio and could have biased the results.

Two years later, the ISAR-STEREO-2 study found similar results with BX Velocity (the future metallic platform of the Cypher stent, Cordis, a Cardinal Health company) in the thick-strut group.² These two randomized studies, conducted by the same team, summarized the evidence-based knowledge regarding the impact of strut thickness. The authors suggested that the endothelialization process was impaired when a thick-strut stent was implanted, cit-

ing a preclinical study that set the threshold at 75 μm .³ Of note, none of these trials actually showed a reduction in the myocardial infarction rate, and the rate of stent thrombosis was not reported. Consequently, the potential endothelialization slowdown associated with thick struts had apparently no impact on acute ischemic events in these two randomized trials.

STRUT THICKNESS IN THE DES ERA: IS THIS THE REAL ISSUE?

Nowadays, all polymer-based DESs have a strut thickness of < 100 μm , with some small vessel versions as thin as 60 μm . Considering the effect of limus on intimal hyperplasia reduction, which was the only proven mechanism of the potential benefits seen in the thin-strut stent in the bare-metal stent era, the role of strut thickness in terms of restenosis in the DES era became rather questionable in the absence of any data to support this concept. The debate on strut thickness shifted from the issue of restenosis to that of stent thrombosis. The recent development of bioresorbable scaffolds (BRSs) places strut size at the core of the debate. The increased risk of subacute and late thrombosis, when compared with a metallic counterpart,⁴ was mainly attributed to strut thickness. The development of a thin-strut BRS is now considered the last remaining hope for this innovation to survive. However, whether strut size reduction will also affect malapposition, underdeployment, and intraluminal late scaffold dismantling is still not known.

FROM STRUT THICKNESS TO STRUT-TO-ARTERY RATIO

The lack of strut coverage leading to strut endothelialization is a major factor of stent/scaffold thrombosis.^{5,6}

Because this process is generated by contiguity with nonstented areas of the vessel, multiple factors may play a role: strut thickness is only one of them, as strut width and interstrut distance are also important, as well as drug type, drug concentration, and polymer biocompatibility. The recent experience with Absorb bioresorbable vascular scaffold (Abbott Vascular) suggests a major role for the stent area-to-vessel area ratio, which explains why this BRS is so poorly biocompatible when oversized in small arteries, resulting in a strut-to-artery ratio twice that of similar DESs. Thus, stent width and design may have a significant impact on DES biocompatibility. Moreover, because some DESs have a rectangular, rounded, or elliptical shape, it is more appropriate to describe a stent platform using a strut-to-artery ratio rather than strut thickness and to consider this criterion for future studies. Unfortunately, manufacturers are not eager to promote these data that should be provided for each available diameter. Nevertheless, strut size seems to be impactful in instances of malapposition,⁷ potentializing its thrombogenic effect, which supports the device/doctor synergy concept to improve BRS biocompatibility.⁴

SMALL-STRUT STENTS: IS THERE A LIMIT?

Thinner struts are empirically associated with better conformability, lower stent profile, easier recrossability, and less injury to side branches. All of these reasons explain why manufacturers developed thin-strut DESs—the mechanical properties of the first-generation DES were not appropriate for addressing complex coronary lesions. Conversely, despite developments in stent designs, there is a trade-off with regard to radial force, longitudinal compression (Figure 1), and the risk of stent fracture (Figure 2). Because these side effects are not easy to detect, they are probably underreported despite their influence on the risk of ischemic events. Stent enhancement software is the most convenient tool for detecting them, and its use should be encouraged in large DES registries. Due to lower radial force and resistance to longitudinal compression, an ultra-thin stent platform is not optimal for the treatment of ostial or eccentric calcified lesions. Moreover, when overexpanded in very large vessels, such as the left main trunk, very thin-strut stents partly lose their scaffolding property, with possible tissue prolapse, as well as their resistance to longitudinal compression. The development of two stent designs to cover all diameters from 2 to 5 mm induces high variations in strut-to-artery ratio. Instead of a race for the thinnest-strut stent, future directions should focus on stable strut-to-artery ratio in a large range of diameters to provide inter-



Figure 1. Implantation of a second DES to treat a longitudinal compression during left main angioplasty.

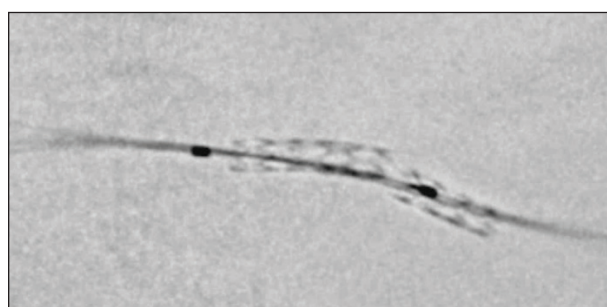


Figure 2. Stent enhancement of a focal in-stent restenosis due to stent fracture.

ventionalists with appropriate tools to treat complex lesions and allow them to choose the right device based on detailed mechanical information not limited to strut thickness. ■

1. Kastrati A, Mehilli J, Dirschinger J, et al. Intracoronary stenting and angiographic results: strut thickness effect on restenosis outcome (ISAR-STERO) trial. *Circulation*. 2001;103:2816-2821.
2. Pache J, Kastrati A, Mehilli J, et al. Intracoronary stenting and angiographic results: strut thickness effect on restenosis outcome (ISAR-STERO-2) trial. *J Am Coll Cardiol*. 2003;41:1283-1288.
3. Simon C, Palmaz JC, Sprague EA. Influence of topography on endothelialization of stents: clues for new designs. *J Long Term Eff Med Implants*. 2000;10:143-151.
4. Stone GW, Abizaid A, Onuma Y, et al. Effect of technique on outcomes following bioresorbable vascular scaffold implantation: analysis from the ABSORB trials. *J Am Coll Cardiol*. 2017;70:2863-2874.
5. Finn AV, Joner M, Nakazawa G, et al. Pathological correlates of late drug-eluting stent thrombosis: strut coverage as a marker of endothelialization. *Circulation*. 2007;115:2435-2441.
6. Nakazawa G, Finn AV, Vorpahl M, et al. Incidence and predictors of drug-eluting stent fracture in human coronary artery a pathologic analysis. *J Am Coll Cardiol*. 2009;54:1924-1931.
7. Lee R, Foin N, Ng J, et al. Early coverage of drug-eluting stents analysed by optical coherence tomography: evidence of the impact of stent apposition and strut characteristics on the neointimal healing process. *EuroIntervention*. 2016;12:e605-614.

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