Dynamic Motion Affects Metallic Stent Fractures

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At present, 80% of percutaneous coronary interventions for atherosclerotic coronary artery disease are performed with a drug-eluting stent (DES). The sirolimus-eluting stent (SES) achieves very low rates of restenosis, around 5%. However, a reported problem for SES is stent fracture that leads to stent restenosis. We have no experience of fracture with Bx Velocity stents, which have the same platform as SES. It is thought that bare metal stents are embedded in thick neointima, so vessel movement does not directly induce serious stress to the body of the implanted stent.

Fractures are mainly reported for SES, but also occur in other types. The risk of SES fracture is high for lesions in saphenous vein grafts or the right coronary artery (RCA), long stenting, overlap stenting, large vessels, and severely angulated lesions. Ino et al. have investigated lesion bending during the systolic and diastolic phases. The difference between the systolic and diastolic angles was defined as the delta angle and it provides a dynamic assessment of the lesion during a cardiac cycle. The stent fracture group showed higher delta angles and a high restenosis rate, although this was unrelated to stent thrombosis.

When considering SES fracture we must understand the structure of a stent. Following the development of the second-generation stent, most designs have been a combination of zigzagged rings along the short-axis with connecting filaments between each ring, creating a flexible stent that can conform to non-simple vessel shapes. When the balloon expands inside the stent, the zigzagged ring scaffold acts as an axial support, while the filaments linking the rings stretch to avoid shortening of the stent. When the SES is deployed in a curved area, the linking filaments remain shortened on the inner curve, while those on the outer curve open and stretch. The difference between the filament lengths at these extremes limits the maximum curve of the SES, given the structural limit of its slotted tube, closed-cell design.

When a SES is deployed for a lesion along a bend, the stent column is twisted during the cardiac cycle. Long-axis bending concentrates the force along just a few filaments, which can result in breakages. Filament stress is also stronger at the edges where stents overlap, as the columnar structure is very stiff at sites of overlap. Tsunoda et al. have reported on coronary stent deformation in an experimental study. They deployed 3 types of stents in an elastic silicon tube from 1 end while the other end was rotated by a motor drive unit. Stent fracture did not occur with the Express2 and Tsunami stents, which have an open-cell design, but did occur with the SES. Stent fracture occurred because of breakage of the connecting filaments at the linked ends adjacent to the zigzagged rings. No defor-

![Figure 1. Schema of sirolimus-eluting stent (SES) structure.](image-url)
formation of the zigzagged rings was observed, and the surface of broken filaments was flat and smooth, so on fluoroscopy there are 2 different axes of columnar stents. According to their results, SES fracture is not related to metal fatigue. A very rare cause of stent fracture, we thought, was a geometric change in an epicardial artery. A patient was treated with a SES in the left anterior descending because of heart failure. The patient’s cardiac function improved in the follow-up period, but there was a stent fracture, which may have been related to a reduction in the size of the left ventricle and increased ejection fraction with hyperkinetic motion.

Another cause of stent fracture was overexpansion (Figure 2B). Overdilatation of the stent shortens the longitudinal length of the zigzagged rings, and the connecting filaments need to elongate to maintain the columnar structure. The link between filaments is unable to withstand high tension and ultimately fractures, which is what can occur with ostial stent placement in the RCA with big balloon dilatation.²

By the very nature of its design there is a physical limit to stent expansion. Because the filament length is fixed the closed-cell design cannot achieve severe bending, though it provides a firm scaffolding for the vessel with hard lesions. During the motion of the cardiac cycle, both ends of the columnar structure move in a hinge motion along the bend of the vessel. It is thought that twisting motion causes the most severe stress to filaments. If one filament breaks, the twist stress increases on the other filaments, thus breaking several filaments and finally causing complete stent fracture. If the number of connecting filaments is reduced, the cell structure of the stent would be more open, with better flexibility to conform, though there would also be less scaffolding and increased plaque prolapse. Future research on stent design should be carried out to accommodate long-axis twisting movements, which may require a construction material different to stainless steel.

References