Stent Strut Thickness and Restenosis

To the Editor:

Kastrati et al recently reported1 significantly less restenosis with lesions randomized to deployment of one stent design (ACS RX Multi-Link, [ML]) than with a related design (Multi-Link RX Duet, [DT]) from the same manufacturer (Guidant). They attributed the difference in restenosis to the difference in strut thickness.

Although the two stent designs do differ significantly in strut thickness, there are other differences in design and mechanical properties that might impact on restenosis. The DT has greater radial strength: when tested in vitro in a pressure chamber, the DTs collapsed at a higher pressure (1.81±0.01 atm) than the MLs (0.77±0.03 atm, P<0.05) (unpublished observations). Some experimental data suggest that greater strut hoop strength may cause increased neointimal proliferation.2 The extent of metal coverage of vessel wall is greater for the ML (22.3%) than for the DT (16.5%). The gap size or uncovered area between the metal coverage of vessel wall is greater for the ML (22.3%) than for the DT (16.5%). The gap size or uncovered area between the metal coverage of vessel wall is greater for the ML (22.3%) than for the DT (16.5%).

Besides strut thickness, there are other differences in mechanical and physical properties between the expanded ML and DT stents that are equally plausible explanations for the differences in restenosis rates between the two designs. These differences may have implications for the design of stents with low restenosis rates.

John A. Ormiston, MBChB
Mark W.I. Webster, MBChB
Peter N. Ruygrok, MBChB
Mercy Angiography
Newmarket, Auckland
New Zealand

Response

We appreciate the interest of Ormiston and colleagues in our article, in which, by comparing 2 stent types with similar design, we found that the one with a thinner strut thickness induced less restenosis.1 Without pretending to have been exhaustive, we discussed in that article some of the potential mechanisms by which strut thickness can influence the process of neointima formation. Another possible mechanism, as pointed out by Ormiston and colleagues, may involve differences in radial force. For 2 stents with identical metal composition and similar radial design, such as those used in the ISAR-STEREO trial, a stent with a greater strut thickness is expected to have a greater radial strength. This has also been confirmed by the unpublished observations of the authors of the letter. We agree with Ormiston and colleagues that the relation between the radial strength of the stent and the degree of subsequent neointima formation may provide additional explanation for the differences in restenosis between the thin- and the thick-strut stents; yet, further studies are needed to clarify the exact mechanisms by which the thin-strut stents reduced restenosis in the ISAR-STEREO trial.

We also discussed in our article the potential role of the subtle differences in design between the 2 stent types used.1 The thick-strut model had fewer cross-links compared with the thin-strut model, but this did not seem to affect the expanded longitudinal flexibility as shown in a recent study of Ormiston et al.2 In their letter, Ormiston and colleagues suggest that the larger gaps due to the slightly lower number of cross-links in the thick-strut model may have allowed a greater tissue prolapse after the intervention and, consequently, more restenosis at follow-up. Although a definitive answer about the validity of this hypothesis would have required intravascular ultrasound investigation, relevant tissue prolapse through the struts of the thick struts seems to be a remote possibility considering the acute angiographic results in the ISAR-STEREO trial, which clearly favored the thick-strut model. In summary, although the close similarity in stent design between the 2 stent models selected is important, it would be interesting to know whether the concept of a reduction of restenosis by the use of thin-strut devices also applies to stents with different design. This issue is currently being investigated in the ISAR-STEREO 2 trial.

Adnan Kastrati, MD
Julinda Mehilli, MD
Helmut Schüllern, MD
Albert Schömig, MD
Deutsches Herzcentrum Munich, Germany

Josef Dirschinger, MD
Franz-Josef Neumann, MD
Melchior Seyfarth, MD
1st Medizinische Klinik rechts der Isar Munich, Germany

Franz Dotzer, MD
Martin Fleckenstein, MD
Medizinische Klinik I Garmisch-Partenkirchen, Germany

Conrad Pfafferott, MD
Medizinische Klinik I Ingolstadt, Germany


Stent Strut Thickness and Restenosis
John A. Ormiston, Mark W.I. Webster and Peter N. Ruygrok

Circulation. 2002;105:e12
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2002 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circ.ahajournals.org/content/105/2/e12

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/