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**CORRESPONDENCE**

## Letters to the Editor

# Stent Overlap in Patients Undergoing Drug-Eluting Stent Implantation

I read with interest the paper by Räber et al. (1) comparing patients with multiple drug-eluting stents (DES) in a vessel with overlap with patients with multiple DES in a vessel without overlap and patients with 1 DES/vessel. The authors demonstrated that major adverse cardiac events were more common in patients with DES overlap than in the other groups at 3 years.

First, because the original study demonstrated a significant difference between sirolimus- and paclitaxel-eluting stent groups (2), it would be of great help if the authors would provide data stratified by stent type. Second, overlapping stent implantation was performed for dissection in some cases (28 of 138, 20%). Dissection might be a cause of creatine kinase elevation (myocardial infarction) rather than overlapping stent implantation itself. To clarify this point, it would be of great help if the authors would provide data about peri-procedural creatine kinase elevation and its association with dissection. Third, target lesion revascularization seems to be determined on a per-patient basis. Because there were twice as many lesions/patient (2.0, 394 in 199 patients) in patients with multiple DES in a vessel without overlap compared with the other groups (1.0, 138 in 134 patients; 1.1, 778 in 679 patients), per-patient analysis might overestimate target lesion revascularization rate in patients with multiple DES in a vessel without overlap.

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## Reply

We appreciate the interest of Dr. Kaneda in our study (1) reporting on the angiographic and long-term clinical outcome in patients with first-generation drug-eluting stent (DES) overlap and take the opportunity to present clinical outcome data up to 3 years stratified/stent type (Table 1) (2). Crude event rates among patients with DES overlap (A), patients with multiple DES in a vessel without overlap (B), and patients with a single stent in a vessel (C) were similar between stent types. Corresponding crude and adjusted hazard ratios (HRs) varied to some extent between stent types, but confidence interval (CI) overlapped widely, and tests for interaction between HRs and stent type were negative, suggesting the absence of a relevant impact of stent type on the clinical outcome among patients with DES overlap.

Dr. Kaneda appropriately raises the question of whether dissections were the source of peri-procedural myocardial infarction (MI) rather than overlapping stent implantations per se. Indeed, peri-procedural MI, defined as any MI occurring within 48 h of the index procedure were more frequent among patients with DES overlap due to dissection (11.1%) as compared with patients with DES overlap related to other indications (0.9%, relative risk: 13.3, 95% CI: 1.3 to 133.0, p = 0.03). When excluding peri-procedural MIs from the analyses, however, we found HRs of MI comparing patients with DES overlap and patients with multiple DES in a vessel without overlap similar to those reported in our paper (1): crude HR: 1.30 (95% CI: 0.47 to 3.58); adjusted HR: 2.07 (95% CI: 0.56 to 7.75). Accordingly, dissections might have contributed in part to the observed impact of stent overlap but do not explain the adverse effect emerging during longer-term follow-up in terms of ischemic end points (death or MI) and restenosis.

We concur with Dr. Kaneda that patients with multiple target lesions are more likely to experience target lesion revascularizations (TLRs) than patients with single lesions. In our study, the hazard of TLR was 1.88 times higher in patients with 2 lesions (95% CI: 1.20 to 2.96) and 3.05 times higher in patients with 3 lesions (95% CI: 1.50 to 6.22) as compared with patients with single lesions (p for trend <0.01). We therefore adjusted, as reported in Table 5 of our article (1), analyses for the number of lesions in the multivariable model. The HR of TLR comparing patients with DES overlap and patients with multiple DES without overlap was 1.26 in the crude analysis (95% CI: 0.76 to 2.11), 1.83 in an analysis adjusted for the number of target lesions (95% CI: 1.06 to 3.19), and 1.94 in the fully adjusted analysis reported in Table 5 of our article (95% CI: 1.05 to 3.58) (1).

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