The authors also conjecture that the scaffold has "collapsed" in the proximal section. Once resorption progresses substantially, structural support of the vessel provided by the scaffold also decreases. If late disease progression leading toward negative remodeling occurs, the scaffold's architecture is unable to prevent such phenomenon. Therefore, in this case, plaque progression leading to negative remodeling rather than "scaffold collapse" is the most likely scenario for the restenosis case seen in this patient.

The successful development of this field requires an exquisite understanding of the mechanisms involved in the process of device resorption and resulting imaging findings. As such, it is key that potential mechanisms of device failure are precisely and accurately described, taking into consideration all mechanisms involved in the process of device resorption. Future device iterations depend on the accurate interpretation of these findings. A thoughtful interpretation and reporting of these findings is critically needed in this field.

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REPLY: Absorb-BVS Resorption and Imaging Findings: Clarifying Current Misconceptions

We greatly appreciate Dr. Granada's interest in our case report (1) and his expert appraisal regarding the nomenclature for precise description of bioresorbable vascular scaffold (BVS) resorption in accordance with imaging findings.

We do agree that the degradation of the Absorb BVS follows a typical curve that has been reported in numerous preclinical data and describes nearly complete absorption after 3 years (2). In contrast, we presented a case in which a BVS restenosis occurred more than 44 months after implantation, differing substantially from the degradation kinetics described in preclinical studies. Remarkably, on optical coherence tomographic (OCT) imaging, nearly all struts or "strut-like" structures were still discernable over the entire length of the 2 scaffolds, and the strut footprints showed typical characteristics of BVS, a situation that typically has been described at a much earlier point in time of approximately 2 years after implantation. Onuma et al. (2) found in a porcine coronary artery model that only 5.4% of struts per BVS were recognizable as preserved boxes at 3 years after implantation. We observed almost exclusively transparent boxes with partly sharp edges and without high-intensity spots in both BVS after more than 3.5 years, which must be interpreted as prolonged absorption or, in other words, degradation. Moreover, in Figure 1H in our report (1), we clearly demonstrated the presence of uncovered intraluminal struts, further indicating markedly delayed resorption, which we described as malabsorption. However, these are all findings on OCT imaging, and we did not perform histopathologic correlation in our case.

We agree with Dr. Granada that the new field of BVS implantation requires an exquisite understanding of the resorption process and the interpretation of OCT imaging findings. Scaffold resorption in our patient was substantially delayed, which may have had a clinical impact. Several more recent clinical studies indicated a significantly higher risk for very late scaffold thrombosis or target vessel myocardial infarction of BVS compared with drug-eluting metallic stents (3,4). Therefore, OCT imaging is a valuable tool, which may provide insight into the complex process of resorption and help visualize pathologies likely to be associated with adverse clinical outcomes. The kinetics of resorption, particularly in

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diseased human coronary arteries, may not always follow the patterns observed in preclinical studies.

Therefore, clinical OCT data must be carefully studied and interpreted in relation to theoretical knowledge. A classification of these OCT findings is essential to standardize the nomenclature concerning resorption and to potentially identify patients who will benefit from BVS placement or, more important, to identify those who are not optimal candidates for BVS implantation.

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