To further explore this issue, large registries should systematically screen and report updated data regarding ScT.

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Please note: Dr. Latib serves on the advisory board for Medtronic. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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APPENDIX For a supplemental table, please see the online version of this article.

Absorb Bioresorbable Vascular Scaffold Resorption and Imaging Findings

Clarifying Current Misconceptions

The report by Dommasch et al. (1) presents the case of a patient with proximal late focal in-scaffold restenosis 44 months following implantation of 2 Absorb bioresorbable vascular scaffolds (Abbott Vascular, Santa Clara, California). The lesion in the proximal scaffold was successfully treated by implantation of a metallic stent (XIENCE, Abbott Vascular). It is the nomenclature used to describe the imaging findings that requires further clarification.

There have been in recent years several publications describing the in vitro and in vivo behavior of poly-L-lactic acid (PLLA), the structural material of the Absorb bioresorbable vascular scaffold. The degradation of this device follows the typical PLLA behavior curve, which has been well characterized for decades in the published data (2). In the in vivo environment, considering the limited ranges of pH and temperature that are compatible with life, polymer degradation kinetics is controlled by the presence of water alone. Also, PLLA undergoes bulk degradation, meaning that degradation occurs equally throughout the whole material. There are no enzymatic processes and no cellular or inflammatory process involved, given that neither macromolecules nor cells can penetrate the polymer backbone; water alone drives this well-understood hydrolysis reaction. Then, the terms used to describe these imaging findings ("malabsorption and delayed or prolonged resorption") are inaccurate because small-molecular-weight species will continue to form overtime and diffuse away as long as the polymer has access to water with sufficient mobility (i.e., >0°C) to undergo hydrolysis.

The authors observe the presence of transparent boxes by optical coherence tomography at 3.5 years. In vivo degradation studies indicate that by 3 years, PLLA has been fully consumed by hydrolysis, and small-molecular-weight species and monomer have diffused away from the region (3). Furthermore, histological examination of healthy porcine coronary arteries shows that polymer is replaced with provisional matrix with various degrees of cellularity. This was confirmed with polarized light and various histological stains on explanted samples (4). As polymer degrades, extracellular matrix replacement occurs following the architecture of the strut (5). Then, it is not uncommon to identify "strut-like" structures in optical coherence tomography imaging at long term in some of the patients undergoing bioresorbable vascular scaffold implantation. This observation has erroneously led operators to conclude that the polymer has been either "malabsorbed" or it is still present. Thus, the term "malabsorption" (a term borrowed from nutrient transport in the intestine) is an inaccurate description of this phenomenon. The extracellular matrix resulting from polymer resorption is autologous in nature, and thus has a low potential to be thrombogenic or pro-proliferative (5).

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