patients either in terms of differences in baseline characteristics or outcomes. These patients may represent an interesting population due to the mechanism by which their AF terminated, based on previous reports that coincidental elimination (e.g., via circumferential PVI) of rotors and localized AF-sustaining mechanisms may explain termination (3). Specifically, it would be informative to know whether AF terminated prior to isolation of the pulmonary vein (PV) (suggesting coincidental ablation of a localized source) or at the completion of PV isolation (suggesting PV-dependent AF).

The resounding message of CHASE-AF and similar studies is that more is not better with respect to AF ablation. Despite these elegant studies, electrophysiologists continue to seek guidance over what to target in 2 important AF patient populations: persistent AF and recurrent AF. Because recent published data shows that established ablation strategies including complex fractionated atrial electrograms ablation and linear lesions serve primarily to elevate complication risk without improving outcomes, it is incumbent upon future studies to evaluate in randomized fashion ablation strategies that evaluate alternative lesion sets versus those that target AF-sustaining mechanisms in these challenging patient subgroups.

**REFERENCES**


**REPLY: Persistent Atrial Fibrillation**

**Time to Stop Comparing Apples With Oranges**

**Atrial Fibrillation**

**Beyond the Pulmonary Veins**

We thank Drs. Providencia and Lambiase and Dr. Schricker and colleagues for their important comments on our paper. We agree with them that choosing the optimal ablation strategy based on the currently existing classification of atrial fibrillation (AF) according to the latest European Heart Rhythm Association/European Society of Cardiology definition based on AF “phenotype” only is critical and of limited value. The cutoff value to distinguish between paroxysmal, persistent, long-standing persistent, and permanent AF is arbitrary and does not reflect the pathophysiology of AF, especially not of persistent AF. Selecting patients with persistent AF according to that definition does not result in a homogenous collective of persistent AF patients. Up to a quarter of patients (potentially even more) in a study by Tilz et al. (1) and approximately 60% in the CHASE-AF (CatHeter Ablation of perSistEnt Atrial Fibrillation trial turned out to benefit from pulmonary vein (PV) isolation (PVI) (“PV-dependent persAF”) alone after a follow-up of 12 months and even after a follow-up of up to 5 years. These patients remained in sinus rhythm after direct current cardioversion following PVI. It is even more remarkable — as stated by Dr. Schricker — that AF terminated in 25% of the study patients prior to or at the completion of PVI (which occurred in either case). We agree with Dr. Schricker that these patients are an interesting population and deserve focused research for improvement of patient selection.

We will obviously not be able to demonstrate a benefit of additional ablation strategies like the stepwise approach or newer techniques in persistent AF as long as a significant amount of our so-called persistent AF patients are suffering from a “PV-dependent form of AF” and as long as we do not completely understand the underlying pathophysiology. This might be a reason why we failed to demonstrate a benefit of the stepwise approach, although we tried to overcome that problem by excluding patients with termination of AF during PVI. Patient selection prior to the procedure based on AF duration, the type of persistent AF (primary persistent AF versus secondary persistent AF [2]), left atrial size, function, and fibrosis, risk factors of AF and potentially electrophysiological criteria will be crucial in the future. International registries— as proposed by Dr. Providencia and Dr. Lambiase— as

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well as multicenter randomized trials would offer the chance to develop new definitions of AF and better selection criteria for choosing the optimal ablation approach tailored for the individual patient. However, such a registry requires standardization of risk factors and especially of ablation protocols, but it offers the opportunity of a more rapid evaluation of new ablation techniques. Therefore, as of today, it is sometimes challenging to discern apples from oranges in the first place since our understanding of AF pathophysiology remains incomplete.

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Intraluminal Scaffold Dismantling

The Downside of Positive Remodeling?

We read with interest the publication by Räber et al. (1), in which 4 cases of very late scaffold thrombosis (VLSt) were reported. A novel mechanism of VLSt that was not seen with metallic drug-eluting stents (DES), namely that of intraluminal scaffold dismantling (ILSD), was suggested in 3 cases. These cases highlight the concerns that are surfacing following the increasing use of bioresorbable vascular scaffolds (BVS) in complex coronary disease.

Pre-clinical and imaging-based clinical investigations have shown favorable BVS healing characteristics because of luminal enlargement due to positive vessel remodeling once bioresorbed (2). However, recent data suggest that this process is associated with higher rates of adverse events, including subacute stent thrombosis, compared with contemporary metallic DES (3). Furthermore, delayed bioresorption and variability in healing have been observed following BVS implantation in diseased human coronary arteries. In 1 case presented by Räber et al., VLSt occurred as late as 44 months after implantation, with optical coherence tomography (OCT)-defined “preserved boxes” suggesting an unfulfilled bioresorption process. Thus, the implantation of BVS, like DES, does not eliminate very late adverse events.

Careful lesion preparation, appropriate sizing, optimal scaffold expansion, and image-guided intervention have been heralded as prerequisites to ensure favorable outcomes. We report a case where, despite these measures, there was evidence of acquired late malapposition and extensive ILSD at 24 months following BVS implantation.

An 81-year-old woman underwent stenting of a left main (LM) bifurcation lesion that involved the left anterior descending (LAD) and circumflex (Cx) coronary arteries (Figure 1A1). Following lesion preparation, a 3.5- × 11-mm Axxess biolimus-eluting, self-expanding bifurcation stent (Biosensors International, Morges, Switzerland) was deployed at the LM bifurcation, after which a 3.0- × 12-mm and 2.5- × 18-mm Absorb BVS (Abbott Vascular, Santa Clara, California) were positioned in the proximal LAD and Cx arteries, respectively. Proximal optimization, sequential post-dilation (3.25- and 2.75-mm noncompliant balloons in the LAD and Cx at 20 atm, respectively), and kissing balloon post-dilation (at 10 atm) were performed. The angiographic result was excellent (Figure 1A2), with OCT confirming full bifurcation coverage and excellent strut apposition of the Axxess device and BVS in the LAD and Cx arteries (Figure 1B1 to 6). At 2 years follow-up, the patient remained asymptomatic, and planned control angiography with OCT assessment was performed. Angiographically, the proximal Cx was ectatic with focal haziness (Figure 1A3). OCT (Figure 1C1 to 6) revealed free-floating unapposed and uncovered struts protruding into the lumen (Figure 1C1, 3, and 4, white arrows) in the mid-scaffold region, suggesting ILSD. Struts were absent (Figure 1C4, white dotted line) where intraluminal struts should have been. Uncovered malapposed struts (Figure 1C5, red arrows) and covered malapposed scaffold struts (Figure 1C6) overlapping with the Axxess stent proximally (Figure 1B6 and 6; white asterisk, Axxess marker in proximal Cx) were noted. Many unapposed struts had a preserved box appearance. Acquired malapposition caused by important positive vessel wall remodeling (minimal