were associated with more extensive infarction and more severely impaired LV systolic function. It is therefore plausible that a small reduction in median infarct size explains the lower rate of early heart failure events and death seen in the complete revascularization arm of the CvLPRIT trial (1,3). Interestingly, the CvLPRIT cardiac magnetic resonance substudy showed no significant difference in infarct size between the randomized groups prior to hospital discharge (2). This discrepancy probably reflects differences in the substudy populations, and the likelihood that early imaging overestimated infarct size.

All patients had undergone PCI to the IRA and were receiving contemporary optimal medical therapy. This may explain the limited inducible hypoperfusion seen even in the IRA-only group, and the inability of complete revascularization to reduce it further (4). Therefore residual ischemia is unlikely to be an important driver of further events post-PCI for STEMI, and its suppression alone cannot explain the reduced event rate in the complete revascularization arm of CvLPRIT. Finally, routine ischemia testing in asymptomatic patients following hospital discharge after PCI for STEMI may have a limited yield, even in those with unrevascularized non-IRAs.

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Persistent Atrial Fibrillation

Time to Stop Comparing Apples With Oranges

We read with interest the study by Vogler et al. (1), which showed no benefit of full defragmentation versus pulmonary vein isolation (PVI) in patients who underwent catheter ablation of persistent atrial fibrillation (AF). We believe that there are some aspects of this study that deserve reflection, which may account for the observed lack of benefit of the full defragmentation strategy and may lead to the failure of other strategies under evaluation.

This study clearly illustrates that the population of patients with persistent AF is highly heterogeneous; 52 of 205 patients (25.4%) in the initial cohort reverted to sinus rhythm while they underwent PVI, and thus, they were classified by the investigators as having PV-dependent AF. However, we cannot rule out that even among the remaining patients (who did not revert to sinus rhythm after PVI), some of them may have also presented with PV-dependent AF, because after induction of AF from PV foci, AF may have self-perpetuated in the atria even after PVI (2).

In such patients, direct-current cardioversion after successful PVI would likely be enough to allow long-term persistence of sinus rhythm and, this seemed to be the case in most of the patients in the PVI-only group (75% of those who underwent a single ablation procedure). Logically, there would be no benefit of additional defragmentation in this subset of patients, which may have occurred in some patients in the full defragmentation group. The key is to try and identify such patients prospectively.
In contrast, there was a second subset of patients in whom the triggers and/or drivers of AF were not exclusively located in the PVs. In these patients, full defragmentation can theoretically be of interest. However, based on the positive results observed in the PVI-only group, it seems that patients with non-PV-dependent AF were significantly underrepresented in this CHASE-AF (Randomized Catheter Ablation of Persist End Atrial Fibrillation Study) cohort. Accordingly, as the investigators acknowledged in this population, left atrial size was smaller than that in most persistent AF studies, and “short-duration persistent AF” was the rule, with most episodes lasting for less than a year, and time since diagnosis being less than 5 to 6 years. Atrial dilation and AF duration are known to be associated with a higher relapse rate, and thus, are likely to be associated with a more resistant persistent AF phenotype (3) due to structural and electrical atrial remodeling, which is non-PV-dependent.

Therefore, this study illustrates that persistent AF seems to be composed of 2 completely different patient populations: those with PV-dependent and non-PV-dependent AF. Until we are able to clearly identify them, research on the best approach to ablate persistent AF is clearly compromised, and all results will be very difficult to interpret.

The current classification for AF based on symptom and/or episode duration is clearly subjective and insufficient, and this study proves it is poorly associated with the underlying pathophysiological mechanisms. What is required is a more comprehensive AF classification scheme that takes into account multiple cardiovascular risk factors, including hypertension, diabetes, structural heart disease, lipid profile, body mass index, as well as specific electrophysiological (e.g., surface electrocardiographic parameters, endocardial voltage, and electrographic features) and structural phenotypes (e.g., left atrial pressure and/or compliance [4]), fibrosis found on magnetic resonance imaging [5] if validated across multiple centers, a low voltage area in atrial tissue on mapping, mitral valve disease, and left ventricular function) to facilitate stratification of AF phenotypes and identify key factors to determine therapeutic approaches, analogous to the CHA2DS2-Vasc scoring system. Without such a systematic approach, we will continue to compare “apples with oranges” and fail to assess the true impact of different catheter-based and drug interventions, compromising our efforts to halt the progression of this epidemic. International registries of outcomes that incorporate agreed upon clinical, structural, and electrophysiological data, as well as documented procedural approaches, would be a step in the right direction to develop such a scoring system to predict clinical response to ablation.

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Atrial Fibrillation
Beyond the Pulmonary Veins

We read with great interest the results of CHASE-AF (CatHeter Ablation of perSistEnt Atrial Fibrillation) by Vogler et al. (1), who reported that in patients with persistent atrial fibrillation (AF), a strategy aimed at defragmenting the atria via pulmonary vein isolation (PVI), complex fractionated atrial electrograms ablation, and linear ablation resulted in similar arrhythmia-free outcomes compared to a PVI-only strategy. The authors should be commended for rigorously conducting this important study that attempts to address the unanswered question of what to ablate beyond the pulmonary veins in AF patients.

Particularly remarkable are the 25% of study patients (52 of 205) in whom AF terminated acutely with PVI only, which is higher than previously published rates of 8% to 16% (2). The authors provide no additional information about these