the only independent factor associated with a higher risk of appropriate shock on VF or polymorphic VT (adjusted hazard ratio: 5.21; 95% confidence interval: 1.63 to 18.80; $p = 0.005$). Patients with NSVT were also more likely to have NSVT episodes detected in the monitoring zone (170 to 220 beats/min) during follow-up (14.9% vs. 6.4%; $p = 0.04$).

Presence of NSVT alone identifies a subgroup (30%) of NICM patients (37% implanted with a CRT device) at very-high risk of rapid and potentially life-threatening ventricular arrhythmia (5.0 events per 100 patient-years vs. 1.7 events per 100 patient-years; $p < 0.0001$) (4). Even if ICD treated fast ventricular arrhythmia is not an equivalent endpoint as all-cause mortality, our findings suggest that NSVT may predict a higher risk population of NICM patients who will actually benefit from implantation of a defibrillator in primary prevention of sudden cardiac death. A new randomized study in that population may be needed (5).

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All authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Clinical Electrophysiology* author instructions page.

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Are Rotors Markers of Substrate or a Mechanism of Perpetuation of Atrial Fibrillation?

Rotor Ablation in AF: Many Unanswered Questions

We read with interest the recent paper published in *JACC: Clinical Electrophysiology* by Zaman et al. (1) titled "Recurrent Post-Ablation Paroxysmal Atrial Fibrillation Shares Substrates With Persistent Atrial Fibrillation."

In that paper, the rotors and focal sources are referred to as markers of electrical substrate, whereas earlier publications described the rotor as a mechanism of atrial fibrillation (AF) perpetuation. This is confusing and warrants further clarification.

Secondly, many studies have shown that pulmonary vein isolation (PVI) alone is enough for first or redo ablation in paroxysmal AF patients. Therefore, substrate ablation in paroxysmal AF patients does not seem to be justified.

Furthermore, in the Discussion section, the authors stated that studies suggesting nonexistence of rotors used empirical rules with technical errors such as cycle lengths of 250 to 500 ms in AF that are less consistent with AF. That is not correct. In the study by Yamabe et al. (2), the range of cycle lengths was 137 to 202 ms; AF was terminated by PVI alone without any additional lesions. Similarly, Benharash et al. (3) did not observe any difference in dominant frequency or Shannon entropy between focal impulse and rotor modulation (FIRM)-identified rotors and distant sites. In that study, the minimum cycle length used for AF induction was 180 ms. Also, electroanatomic mapping showed no rotational activation at FIRM-identified rotor sites in 96% of their patients (3).

Moreover, the authors have cited the OASIS (Outcome of Different Ablation Strategies In Persistent and Long-Standing Persistent Atrial Fibrillation) trial (4) and have stated that the paper has "subsequently been retracted due to non-randomization

issues which may impact any comparisons between groups." We disagree with the authors in this regard. Even if the OASIS study groups were considered as consecutive series, the findings still clearly demonstrated the safety and efficacy of the comparison approaches in nonparoxysmal AF patients.

Furthermore, the authors have compared the outcome of the OASIS trial with that of the LIBERATION (Outcome of Atrial Fibrillation Ablation After Permanent Pulmonary Vein Antrum Isolation With or Without Proven Left Atrial Posterior Wall Isolation) trial (5), where a 20% success rate at 1 year from pulmonary vein antrum isolation alone in persistent-AF patients was reported. We find this comparison inappropriate for the following reasons:

1. The authors have claimed that the substantial improvement in the success rate (52% in the FIRM+PVI group of the OASIS trial vs. 20% with PVI only in the LIBERATION trial) was due to the adjunctive FIRM ablation. This is most unlikely because the FIRM-only arm in the OASIS trial had a meager 14% success rate at follow-up.
2. Because the LIBERATION trial was designed to detect proven isolation of pulmonary veins in persistent-AF patients, stringent selection criteria were used to choose patients for this study. Furthermore, the follow-up duration was 3 years, whereas it was 4 to more than 12 months in the OASIS trial. Also, in the LIBERATION study, the success rate at 1 year was calculated using the outcome data collected at the end of 1 year in every patient. Therefore, it is not appropriate to compare the outcome of the LIBERATION trial with that of the OASIS trial.

Several articles from the founders of the FIRM-mapping technology have reported a very high success rate of rotor ablation, although other independent groups of highly skilled operators have failed to replicate their study findings. Clearly, large randomized trials will be necessary to resolve this controversy.

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REPLY: Are Rotors Markers of Substrate or a Mechanism of Perpetuation of Atrial Fibrillation? Increasing Data for Rotational Drivers of Human AF

We thank Dr. Mohanty and colleagues for their letter concerning our study (1). Their philosophy that it is unnecessary to map paroxysmal atrial fibrillation (AF) highlights our central point that "paroxysmal" or "persistent" encompasses heterogeneous AF populations who are often misclassified when examining continuous ECGs. Moreover, recent trials in paroxysmal AF report ~65% ablation success at 1 year, which are lower on longer follow-up and in persistent AF. Accordingly, there is a growing body of published reports on mapping electrical (drivers, autonomic ganglia) and structural (fibrosis, scar) substrates, as well as triggers at repeat ablation.

Dr. Mohanty and colleagues discuss the OASIS (Outcome of Different Ablation Strategies In Persistent and Long-Standing Persistent Atrial Fibrillation) trial (NCT02533843), retracted for stated issues with randomization and enrollment before trial registration. It appears that Gianni et al. (2) initially reported an 'observational study of consecutive non-paroxysmal AF patients' submitted before the registration date of the OASIS trial on ClinicalTrials.gov, of whom 29 were then included as a randomized cohort in NCT02533843 (OASIS) (3). Even if considered as consecutive patients as now proposed, inconsistencies include procedural components that were shorter for driver ablation plus pulmonary vein (PV) antrum isolation than driver ablation alone, and even shorter