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We thank Dr. Çörekçioğlu and colleagues for their interest in our recent publication demonstrating an important association between side-branch predilatation and mortality in patients with coronary bifurcation stenoses [1].

While we fully agree that side-branch predilatation is a questionable matter, we hardly see the value of the application of the V-resolve risk score in our population [2]. This score was based on the visual estimation of only one observer. Furthermore, the score was developed in a retrospective, single-center study, without external validation and different observers. The authors of the study mention themselves that the V-RESOLVE score should not be regarded as a single criterion of strategy selection in bifurcation revascularization.

We acknowledge the comment regarding the assessment of the significance of the side branch. The inclusion criteria of our study include a side branch diameter $\geq 2.0$ mm. Regarding
side branch protection techniques, we adopted a jailed-wire approach in all patients. Although the study by Dou et al. [3] reported that the jailed balloon strategy is superior to the conventional strategy in reducing SB occlusion, analyses have shown that jailed balloon protection does not translate into lower MACE at 1-year follow-up [4] or a reduction in procedural myocardial infarction [5]. Therefore, we have consistently followed the current recommendations regarding provisional stenting and side branch protection [6].

We appreciate the opportunity to discuss in detail the rate of periprocedural myocardial infarction and periprocedural troponin elevation in our study. Despite the probable protective effect on SB closure the rates of troponin rise >5 × UNL (SBPD vs. SBPD — 46% vs. 28%; $P = 0.003$) post-PCI and rate of troponin rise >20% from the baseline level (SBPD[+] vs. SBPD[–] ± 86% vs. 77%; $P = 0.002$), were significantly higher in the SBPD group. Interestingly, the rate of periprocedural myocardial infarction was not significantly different between groups with and without SB closure – 52% vs. 34%; $P = 0.066$. We performed additional survival analysis dividing the patients into groups with a post-procedural troponin rise 5 × UNL and those without such a significant rise in troponin. The results revealed a non-significant difference in all-cause (23% vs. 24%; $P = 0.415$) and cardiovascular mortality (23% vs. 22%; $P = 0.633$) between SBPD(+) and SBPD(–) in the low troponin group (Figure 1A). However, in the group with high post-procedural troponin, there was a numerically higher all-cause (34% vs. 30%; $P = 0.095$) and cardiovascular (26.2% vs. 22%; $P = 0.138$) mortality in the SBPD(+) group (Figure 1B).

In conclusion, we believe that our results support the hypothesis that side-branch predilatation is an important marker of bifurcation lesion severity and is therefore associated with worse clinical outcomes. We hope that by addressing the comments raised, the scope of our study has been increased and that it can serve as a basis for future research in the field of coronary bifurcation interventions.
Figure 1. A. Kaplan–Meier curves showing all-cause death in patients without a troponin increase \(>5 \times \text{ULN}\) — patients with side branch predilatation vs. patients without side branch predilatation; B. Kaplan–Meier curves showing cardiovascular death in patients with a troponin increase \(>5 \times \text{ULN}\) — patients with side branch predilatation vs. patients without side branch predilatation.
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