To the Editor:

We congratulate Van Mieghem and colleagues for the significant contribution they have made to the understanding of the embolic phenomena associated with transcatheter aortic valve implantation through their article, “Histopathology of Embolic Debris Captured During Transcatheter Aortic Valve Replacement.”

Somewhat surprising is the high occurrence of thrombotic material, which was identified in 21 of 40 or 52% of patients and accounted for 70% of embolic cases. Certainly, a preexisting acquired thrombophilic state has been confirmed among transcatheter aortic valve implantation candidates, with echocardiographic studies demonstrating remarkably high incidence of preprocedural intracardiac thrombi and spontaneous echocardiography contrast. This is potentially intraprocedurally through exposure to the artificial surfaces of the catheters and valve frame, damage to the prosthetic and native valve leaflets, and disruption of the endothelium.

To overcome these prothrombotic factors, the investigators used dual antiplatelet therapy and periprocedural intravenous heparin, targeting an activated clotting time of 250 to 300 seconds. Despite these measures, 13 of 21 or 62% of all thrombi were acute. The authors attributed some of this to suboptimal activated clotting time levels (<250 seconds), as identified in 65% of patients at 30 minutes after the first heparin bolus. These findings of early suboptimal activated clotting time levels are a relevant insight into daily clinical practice. Interestingly, however, the target used in this study is less than the recommendation of consensus guidelines of an activated clotting time >300 seconds.

Perhaps of equal importance to the formation of acute thrombi is the influence of the filter-based embolic protection device. First, we note the procedural duration of 181.40±21.00 minutes. This significantly exceeds times reported across the literature to date and clinical experience for procedures in the absence of filter-based embolic protection devices. Furthermore, the additional instrumentation increases thrombogenic surface exposure. Both of these factors may explain the unexpectedly high occurrence of acute thrombosis.

Additionally, we note the contribution of valve/vascular tissue to the overall embolic load. This confirms the behavior of the friable valve tissue and, because this tissue is not excised (as is the case in traditional surgical aortic valve replacement), raises the issue of ongoing tissue embolization in the postprocedural period. Alternatively, given the predominance of Medtronic CoreValve in this study (86%), it may be that this is a phenomenon unique to this valve type. Maximum embolization has been shown to occur during the implantation phase with the Medtronic CoreValve compared with the positioning phase with Edwards SAPIEN valve. This has been attributed to the slower expansion of the Medtronic CoreValve, resulting in the grating of tissue during the implantation stage.

In light of these comments, it is our opinion that care must be exercised when translating the findings of Van Mieghem et al to the transcatheter aortic valve implantation procedure in general because the origin of emboli identified in this study may be unique to transcatheter aortic valve implantation procedures with the Medtronic CoreValve and with use of an embolic protection device.

Disclosures
Associate Professor Walters is a Consultant to Medtronic and Edwards, a past proctor for Edwards, and an investigator for Edwards, Medtronic, and Boston Scientific clinical studies. The other authors report no conflicts.

References
Letter by Fanning et al Regarding Article, "Histopathology of Embolic Debris Captured During Transcatheter Aortic Valve Replacement"
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