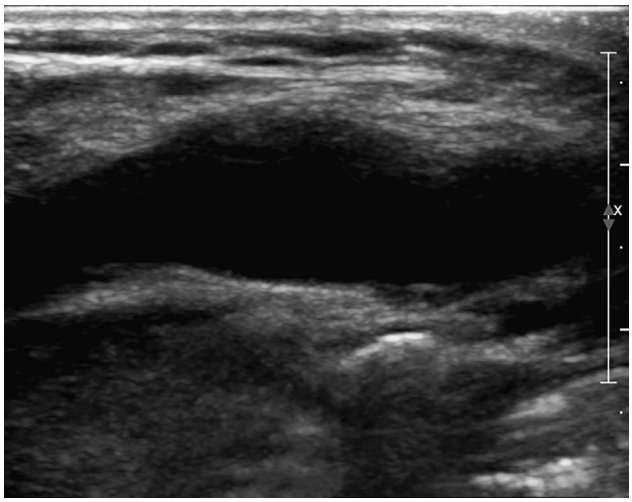

Figure 2 B-mode ultrasound image of the RICA bulb in Case 2. The dilated portion has an area of non-laminar flow with stasis or reverse flow, which may account for the formation of echolucent thrombus in a low-flow continuous state.

diastolic flow. The mean PSV decrease was 35 cm/s in the common carotid artery and 33 cm/s in the internal carotid artery. Similarly, we noted a decrease in the PSV in both of the patients we described. The effect of decreased PSV is difficult to determine because of the continuous flow, which results in a compensatory increase in peak end-diastolic velocity.

In many patients who have cerebral infarction after LVAD implantation, the cause of the infarction is unknown. However, we do know that stasis produced by low or continuous flow can cause thrombus formation, especially in areas of dilation, such as the carotid bulb (Figure 2). Although we routinely perform pre-operative carotid Doppler studies on all patients undergoing LVAD implantation, in the past we have not routinely performed post-operative studies unless we suspected stroke. Even in those cases, not all patients had post-stroke carotid Doppler studies and in the majority of the patients studied, retrospective visualization of the carotid bulb was inadequate to diagnose thrombus formation.

Based on the results in the first 2 patients reported herein, we undertook the prospective study (June 2013) described earlier. Now, all patients who have continuous-flow pumps implanted have specially designed carotid ultrasound exams, which will allow us to better measure flow dynamics before device implant and routinely after implant. In these studies, we are also assessing the status of the carotid bulb and aortic valve opening times and calculating volume–flow rates.

We believe that the role of stasis in the carotid bulb and the potential for thrombus formation should be further investigated. The laminar flow of continuous-flow pumps is accentuated if the aortic valve is not opening, which is generally the case for 1 month or so after implantation, and can result in an area of stasis in the dilation of the carotid bulb. This may be particularly problematic when the aortic valve begins to open. In the cases reported herein, the aortic valve was not open after implant, but in the 2 patients who had strokes, it was open at the time of their stroke. The third patient has been maintained consistently at higher flow rates (9,600 rpm) to keep the aortic valve from opening until the thrombus has resolved. Our concern is that the change in flow characteristics and turbulence induced by the aortic valve opening may be a liability for dislodgement of emboli from a carotid-bulb thrombus. We also believe that the impact of carotid-bulb thrombus on baroreceptor response with the consequent effect on systemic blood pressure should be investigated.

In conclusion, preserving aortic valve opening may be helpful in reducing stasis and thus preventing carotid-bulb thrombus and other complications related to continuous-flow pump support.[4] Until more data are accumulated, physicians treating patients after LVAD implant with a continuous-flow pump should consider routine pre- and post-operative 2D Doppler ultrasound screening of the carotid arteries, and, especially, of the carotid bulb.

Disclosure statement

The authors have no conflicts of interest to disclose.

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Aortic valve opening and thrombotic events with continuous-flow left ventricular assist devices

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Despite a steady increase in utilization of left ventricular assist device (LVAD) therapy, widespread acceptance of this technology has been partially limited by its adverse event profile. In particular, thromboembolic complications, such as device thrombosis and cerebrovascular accident (CVA), can be devastating. According to the most recent report by the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS), 11% of patients on device support at 1 year have a new CVA.[1] Strategies to optimize pump operation to minimize adverse

events have been underexplored. Aortic valve opening (AVO) is a readily available marker of LV unloading and can be modified by LVAD speed.[2] In this analysis, we tested the hypothesis that the frequency of AVO and LVAD speed is associated with thromboembolic complications.

We conducted a retrospective chart review of 80 consecutive subjects undergoing HeartMate II (HMII) LVAD implantation at our institution between June 2006 and March 2012. Those with surgical aortic valve closure were excluded. Subjects were categorized into the "thrombosis" group if they had any thromboembolic event identified by computed tomography scan, angiography or device thrombosis. Device thrombosis was confirmed by direct visualization during device exchange, post-mortem explantation or CT scan.

AVO was recorded from all clinical post-operative echocardiograms and scored as follows: 0 = no opening; 1 = intermittent opening; and 2 = opening every beat. For those in the thrombosis group, AVO and LVAD speed were averaged up until the time of event. For subjects without a thrombotic event (TE), parameters were averaged throughout the duration of support or until March 31, 2012. International normalized ratio (INR) and lactate dehydrogenase (LDH) for the 2 weeks prior to the TE were averaged. As per institutional protocol, all subjects were maintained on aspirin and warfarin with a target INR of 1.5 to 2.5. Retrieval and analysis of patient data were conducted after approval from the institutional review board of Montefiore Medical Center.

Data are displayed as mean ± standard deviation, unless specified otherwise. Baseline demographics, AVO and LVAD speed were compared between subjects with and without TE using Student's *t*-test for continuous variables and by chi-square test for categorical variables. An additional subgroup analysis assessing the association of AVO was performed between subjects with and without device thrombosis. Two-tailed $p < 0.05$ was considered statistically significant. Statistical analyses were conducted using GraphPad Prism, version 5.04 (GraphPad, Inc., La Jolla, CA).

Twelve of 80 subjects (15%) had a TE. The median time to a TE after HMII implantation was 99 days (range 22 to 178 days). Four subjects had a sub-therapeutic INR and were not on bridging heparin. Six subjects had CVAs:

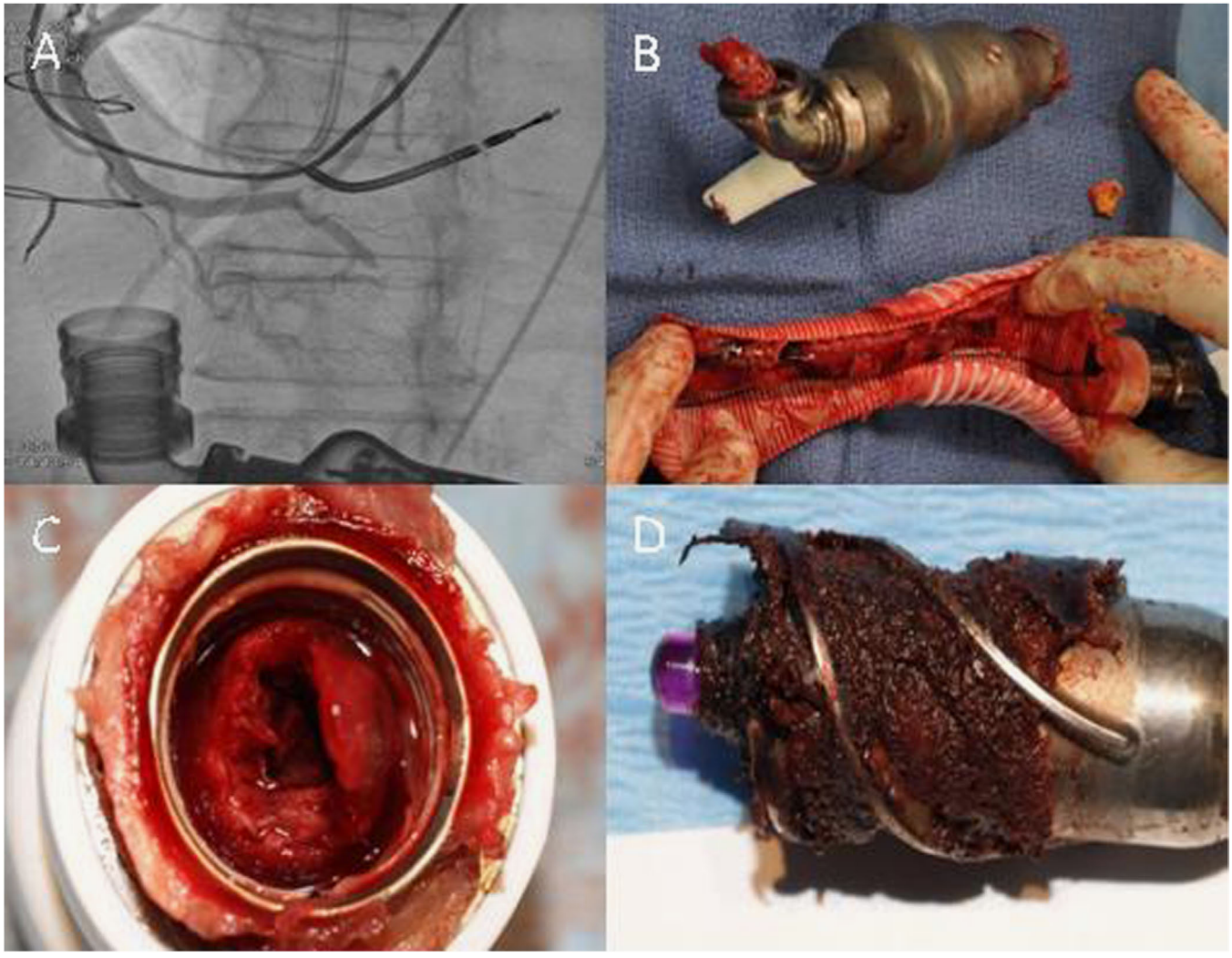

Figure 1 Location of thrombotic events. (A) Coronary angiogram showing a filling defect at the bifurcation of the posterior descending artery. (B) An opened longitudinal section of the outflow cannula showing thrombus adherent to the inner lining. (C) Cross-section of the stator attachment showing internal thrombus. (D) Thrombus on the LVAD rotor.