Vulnerable Brain and Ventricular Assist Devices

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See related article, p 2702.

In the past 50 years, we have learnt to transition mechanical circulatory support devices, such as durable left ventricular assist systems (LVAS), from crisis intervention life-saving devices to the contemporary era of using such therapy in prolonging meaningful life in patients with advanced heart failure. In some cases septic emboli as a result of infections. The causes of strokes with newer LVADs are unclear and could relate to (1) clots that pass through the device (as with atrial fibrillation in patients with a device or with clots that form within the device or in the proximate ventricle), (2) vascular changes as a result of nonpulsatile flow (we know that reduced pulse pressure with the newer devices increases vascular fragility, so even lower levels of blood pressure can cause vascular loss of integrity), and (3) rheological causes (new devices create an acquired von Willebrand syndrome and can predispose to bleeding). Debilitating stroke is a disastrous situation that diminishes the gains from application of LVAS therapy and occurs with enough frequency and is intertwined with other complications of LVAS, that it is a major concern, especially in older patients who are receiving these devices as lifetime therapy without the option of a transplant. Often, the diagnosis leads us to have to shut off these devices for compassionate reasons because the quality of life is immeasurably reduced.

Because the occurrence of stroke is largely unpredictable in an individual and comorbidities are common, we must focus our efforts on performing a thorough evaluation for the risk of stroke preimplantation of these devices, establish optimal surveillance guidelines in follow-up after implant, and ensure good blood pressure control and use of antiplatelet therapy.
Avoidance of hemorrhagic strokes by tightening control of anticoagulation and evaluating those with thrombophilia should be carefully done to avoid ischemic strokes. In patients who do not open their aortic valves on LVAS therapy and demonstrate low peripheral pulse pressure, we should consider surveillance to pick up clot formation at sites such as the proximal aorta and the carotid bulb. We need to do more studies that help us understand neurovascular blood flow characteristics with LVADs and the impact on vascular integrity and research how best to follow these patients in an effort to decrease this devastating occurrence. Until we learn more of this complication, tackle it more effectively, LVAS application to broader populations of advanced heart failure will remain greatly encumbered.

Disclosures

Dr Mehra reports relevant conflicts as follows: Consultant for Thoratec (now St Jude Medical, Inc), HeartWare, Medtronic, Johnson and Johnson (Janssen), and Teva Pharmaceuticals. He is also Editor-in-Chief of the Journal of Heart and Lung Transplantation. The other authors report no conflicts.

References


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