Abstract: **P5370**

The effect of endovascular baroreflex amplification on sympathetic nerve activity in patients with resistant hypertension: a proof-of-mechanism study

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Background/Introduction: Endovascular baroreflex amplification (EBA) is a novel device-based therapy that has been shown to lower blood pressure (BP) in patients with resistant hypertension (RHTN) in an uncontrolled first-in-human study. It is postulated that EBA lowers BP, at least in part, by inhibiting sympathetic nerve activity (SNA). However, this has never been studied.

Purpose: The aim of this study was to determine the effect of EBA on SNA and sympathetic cardiovascular reactivity.

Methods: In a single-center sub-study of the CALM-DIEM study (Controlling And Lowering blood pressure with the MobiusHD – Defining Efficacy Markers), the endovascular MobiusHD device was unilaterally implanted into the carotid sinus of patients with RHTN (24-hour mean ambulatory systolic BP >130 mmHg on ≥3 antihypertensive medications, including a diuretic) without atherosclerotic carotid disease. Microneurography at the right peroneal nerve and non-invasive continuous BP measurements were performed at baseline and 3 months, after a 2-week washout of antihypertensive medications affecting sympathetic regulation. Changes in resting muscle SNA and cardiovascular responses to cold pressor testing (CPT) were determined.

Results: 10 patients, mean age 52 (±8 years), 6 males, have been included in the study thus far. Of these, 8 patients had a paired measurement at baseline and 3 months. One patient has not yet reached the third month endpoint and one patient was unable to visit our clinic for measurements at 3 months. Two of the 8 paired measurements were excluded from analysis because of poor quality of muscle SNA recording and one was excluded because of frequent premature ventricular complexes. 24-hour mean ambulatory BP (n=8) decreased from 156/98 (±22/17) mmHg to 145/88 (±19/17) mmHg (p=0.028 for systolic and p=0.048 diastolic BP). Analysis of the first 5 patients to complete the study with paired data, suggests a decrease in resting muscle SNA from 38.6 (range 15.7–70.6) to 30.3 (range 5.0–65.9) bursts/min, and from 48.3 (range 21.2–81.2) to 37.9 (range 7.3–75.2) bursts/100 heartbeats, 3 months after EBA. In contrast, cardiovascular reactivity appears unaffected because MAP and HR responses to CPT did not change.

Conclusion: In patients with RHTN we observed a decrease in BP and muscle SNA 3 months after EBA. The finding is consistent with attenuated central sympathetic drive and may suggest that EBA, indeed, improves sympathetic baroreflex restraint. Furthermore, the MAP and HR responses to CPT did not change, indicating that BP reactivity remains intact after EBA.