Intramural Stent Effects on Duplex Velocity Estimates
Christopher S. Koppler, William R. Newton, III, Timothy E. Craven, Rachael J. Ghanami, Shawn H. Fleming, Kimberley J. Hansen, Christopher J. Godshall, Wake Forest University Baptist Medical Center, Winston-Salem, NC

Background: To measure changes in duplex velocity estimates associated with balloon expandable (BES) and self-expandable (SES) endoluminal stents.

Methods: An in vitro vascular circuit model consisting of a pulsatile pump, tubing, and a conduit was created. The pump was programmed to replicate the doppler spectral waveform pattern of the renal and carotid arteries. Conduits consisted of Silygard® (Dow Corning, Midland, MI) tubing of variable compliance with differing wall thicknesses. An intraluminal fluid was created using a cornstarch and water mixture which had an average viscosity of 3.46 cP over the range of fluid velocities observed in this study. Pressure was measured continuously by electromechanical transducer at a site proximal to the conduit. Flow velocities were estimated with duplex ultrasound utilizing a matrix linear array probe (GE Logic E-9, GE Healthcare). Peak systolic velocity (PSV) and end-diastolic velocity (EDV) were estimated at 5 distinct conduit locations. Three replicate velocity measurements were made at each location. Diameter measurements of the conduit at peak systole and end diastole were made, and change in cross-sectional area calculated. After initial velocity estimates, a BES or SES was deployed within the conduit. Velocity estimates were then repeated. Three separate conduits were used. Analysis of PSV and EDV measured under the three arterial conditions (unstented, BES, and SES) was performed using mixed linear models that included a random effect to account for between subject variance. Fixed effects were included for condition, location (five segments marked on each conduit and denoted “A” - “E”), and replicate (1, 2, or 3). All analyses were performed using SAS version 9.2 software.

Results: Compliance was estimated for each conduit under each condition (unstented, BES, and SES). Values ranged from 3.12 × 10^-2 to 8.43 × 10^-2 mmHg. Average PSVs differed by condition. Mean ± SE PSV was 95.8 ± 2.6 cm/s, 97.0 ± 2.7 cm/s, and 101.4 ± 3.7 cm/s for unstented, BES and SES, respectively (P < .0001). Average PSVs differed significantly across location (P < .0001) and replicate (P = .039); however, no interaction between condition and location or replicate were found. EDV values did not differ significantly by condition. Mean ± SE EDV was 36.2 ± 1.0 cm/s, 37.3 ± 1.1 cm/s, and 37.2 ± 1.1 cm/s for unstented, BES and SES, respectively (P = .13). Average EDVs differed significantly across location (P = .001) but not across replicates. The difference in percent decrease in PSV was estimated by comparing paired differences between the unstented and stented conduits calculated at each replicate. Mean ± SE percent change in PSV was −1.0 ± 3.3% for BES minus unstented, and 6.4 ± 3.1% for SES minus unstented (P = .24). Mean ± SE change in EDV was 1.3 ± 5.8% for BES minus unstented, and 3.5 ± 4.4% for SES minus unstented (P = .79). Overall estimates of the effect of stent placement (pooled across BES and SES types) were 3.7% (95% CI: −6.4%, 13.8%) for PSV, and 0.8% (95% CI: −7.1%, 8.3%) for EDV.

Conclusions: The presence of BES and SES were associated with a less than 7% change in estimated PSV. These results suggest that Doppler velocity estimates for renal and carotid arteries are not materially affected by either balloon expandable or self expandable endoluminal stents.

Hormone Replacement Therapy Influences Matrix Metalloproteinase Expression and Intimal Hyperplasia Development after Vascular Injury: A Follow-Up Study
Richard B. Cook, Deirdra J. Mountain, Stacy S. Kirkpatrick, James E. Chalk, David C. Cassada, Scott L. Stevens, Michael B. Freeman, Mitchell H. Goldman, Oscar H. Grandas, University of Tennessee Graduate School of Medicine, Knoxville, Tenn

Background: Postmenopausal women taking hormone replacement therapy (HRT) have increased intimal hyperplasia (IH) following vascular intervention. Matrix metalloproteinases (MMPs) play a major role in IH development, and we have shown that hormone exposure results in unbalanced MMP regulation in vascular smooth muscle cells in vitro. Previously we presented data from a small pilot study suggesting a role for HRT in the development of IH via MMP modulation in vivo, using a postmenopausal rodent model of vascular injury. Here we further investigated the role of HRT as a modulator of MMPs and IH in a larger follow-up study.

Methods: Female rats were aged to 12 months and ovariectomized (OVX). Four weeks later estrogen (E), progesterone (P), estrogen plus progesterone (E/P), or placebo (Plac) were delivered via 90-day slow-release pellets. Following 6 weeks of HRT, each rat underwent balloon angioplasty of the left common carotid artery. At 14 days postinjury, tissue samples were collected and stained for various MMP isoforms.

Results: Following vascular injury I:M was decreased in OVX rats compared to non-OVX controls (Fig 1, n = 5-6). In OVX animals, HRT exposure did not significantly increase the I:M ratios (Fig 1, n = 5-6, P = NS). Injury-induced expression of MMP-2 and –9 was significantly decreased in OVX animals compared to non-OVX controls (Table 1; n = 5-6). MMP-2 and –9 levels were subsequently increased by each type of hormone therapy compared to placebo, with a significant increase in MMP-9 in response to estrogen with and without progesterone (Table; n = 5-6). Conversely, TIMP-2 was significantly decreased by estrogen compared to placebo (Table; n = 5-6). There was no effect on intraluminal MT1-MMP in any group.

Table 1.

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<th>Group</th>
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<tr>
<td>NonOVX</td>
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<tr>
<td>C</td>
<td>OVKX - Plac</td>
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<tr>
<td>MMP-2</td>
<td>2.6 ± 3.7</td>
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<tr>
<td>MMP-3</td>
<td>4.3 ± 0.1</td>
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<tr>
<td>MMP-9</td>
<td>5.0 ± 8.9</td>
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*P < 0.05 vs. NonOVX C

Conclusions: Here we were not able to demonstrate a statistically significant decrease in IH as a result of ovarectomy. Furthermore, HRT at the doses given did not remarkably increase IH. Here we also demonstrate a significant reduction in MMP-2, –9, and TIMP-2 in response to ovarectomy. Subsequent hormone exposure results in the upregulation of MMP-2 and –9 without a counter-regulatory increase in TIMP. We have previously shown elevation in MT1-MMP to occur during the initial phases of IH development; therefore, examination earlier than 14 days postinjury is needed to determine the effect of HRT on this MMP regulatory isoform. Future studies would investigate in vivo manipulation of this unbalanced MMP regulation for prevention of IH in response to HRT exposure.

National Trends in Repair of Intact Abdominal Aortic Aneurysms in the Medicare Population
Mounir J. Haurani, Mark F. Conrad, Virendra I. Patel, Emel A. Ergul, Christopher J. Kwollik, Richard P. Cambria, Massachusetts General Hospital, Harvard Medical School, Boston, Mass

Background: Endovascular abdominal aortic aneurysm repair (EVAR) of intact abdominal aortic aneurysm (AAA) continues to gain favor despite the perceived higher procedural costs when compared to open repair (OPEN). National trends in AAA repair are unknown. This study aims to compare regional utilization of EVAR and its effects on patient mortality and procedural costs.

Methods: All patients in the Medicare database who underwent AAA repair from 2004 to 2007 were identified and stratified into OPEN and EVAR cohorts. Geographic regions were created according to standard US census divisions. Primary outcomes included perioperative mortality, long-term survival and hospital costs.

Results: There were 103,033 patients identified, 68,370 EVAR (66.4% vs 34,663 OPEN (36.6%). Although EVAR is favored nationally, there are significant differences in regional utilization (Table). Between 2004 and 2007, the total number of AAA repairs was not different (25,246 vs 25,850, P = NS), but the percentage of EVARS performed was significantly higher in 2007 (14,001 [55%] vs 19,471 [75%], P < .001). The national 30-day mortality was significantly higher after OPEN (4.9% vs 1.6%, P < .001), however, long-term survival was equal (73.6% OPEN vs 74.0% EVAR P = .04) with no regional differences (Table). Hospital charges for EVAR were significantly less than open (564,380 EVAR vs 568,174 OPEN, P < .0001), as was Medicare reimbursement ($319,000 EVAR vs $224,474 OPEN, P < .001). This is likely due to an increased length-of-stay in the OPEN cohort (3.5 days EVAR vs 9.9 days OPEN, P < .001)