Is Vascular Surgery Giving up the Vascular Laboratory?

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Objective(s): In an attempt to identify the concerns of vascular fellows regarding their training in vascular surgery, we conducted an anonymous survey consisting of 22 questions at an annual national meeting held yearly in March from 2004 to 2010.

Methods: The fellows were asked to assess their endovascular, open, and vascular laboratory experience as excellent, satisfactory, or mixed. They were queried about who trained them in their endovascular skills, the quality of their didactics, and amount of small cases that yielded no learning experience. Of the 466 attendees, 376 (80%) completed the survey. Up to 84% of those surveyed were men. Second-year fellows comprised up to 66% of those surveyed.

Results: Most (79%) were satisfied with their endovascular experience during their fellowship, and 81% were satisfied with their experience with open cases. Interventional skills were obtained from a vascular surgeon (84%), an interventional radiologist (9%), a cardiologist (1%), or a mixture (5%). The didactics were felt to be excellent, satisfactory, or required some or much improvement in 20%, 64%, 11%, and 4% respectively. The distribution of nonlearning cases was felt to be excellent, satisfactory, or required some or much improvement in 42%, 45%, 10%, and 3% respectively. However, only 64% thought their vascular laboratory experience was excellent or satisfactory. Only 36% actually performed the vascular duplex examination, and only 47% felt that they would feel comfortable in managing a vascular laboratory. Forty-six percent suggested that finding the type of job they wanted was easy in 46% or moderately difficult in 46%. Most (72%) felt that future demand for manpower in vascular surgery will exceed available manpower. In the 2004 survey, the primary source of training for interventional skill was a vascular surgeon in 77% and this increased to 100% in 2010. No other major significant differences were noted from year to year.

Conclusions: The results of this survey suggest that several significant issues are reflected in the minds of vascular trainees. These data suggest that vascular fellows are not being adequately trained in the vascular laboratory. Because the trainees represent the future of our field, we suggest that the vascular laboratory become a specific area of focus in the fellowship training.

Do Ethnic Differences and TransAtlantic InterSociety Consensus Distribution Affect the Incidence of Lower Extremity Amputation?

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Objective(s): There has been recent interest in ethnic differences with a wide variety of disease entities. Some evidence has suggested that particular ethnic populations may be more susceptible to severe manifestations of peripheral arterial disease. This study was initiated to better determine what if any factors affected a diverse ethnic population afflicted with critical limb ischemia (CLI).

Methods: We reviewed all patients presenting with CLI from January 1, 2007, to December 31, 2007. CLI was defined as ischemic rest pain, nonhealing ulceration, or gangrene (Rutherford class 4 and 5) for which a major amputation was imminently required. All patients underwent conventional arteriography and if possible, an endovascular, open, or hybrid procedure for successful limb salvage. Multivariate logistic regression was used to determine any association between ethnic background and Rutherford class, comorbid conditions, TransAtlantic InterSociety Consensus II (TASC II) classification, runoff score, types of intervention (open, endovascular, or hybrid), prior and repeat procedures, death, and 1-year amputation-free survival (outcome).

Results: During this 1-year period, 148 patients presented with primary, secondary, or tertiary CLI. Of these, 58 patients (40%) were black, 57 (40%) were Latino, 28 (20%) were white, and ethnic background could not be determined in 5. There was no difference in clinical presentation among the three races. All groups had similar rates of TASC II D iliac disease; however, black patients had a higher prevalence of TASC II D femoropopliteal disease. Latino and white patients had a higher prevalence of TASC II D infrapopliteal disease. Primary patency rates among the ethnic groups remained similar at 1 year, however the Latino group displayed poorer patency after 1 year compared the black and white groups. All three ethnic groups had a similar 1-year amputation rate of 6.4%.

Conclusions: There does not appear to be a significant difference among racial groups in clinical presentation. There are disparate lesion distributions among the three ethnic groups, with more severe TASC II D infrapopliteal disease in the Latino group, possibly affecting primary patency. There was no clear ethnic difference amongst the groups with regards to overall amputation rate.

Role of Prostaglandins in Venous Thrombus Resolution in a Murine Model of Thrombosis

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Objective(s): Thrombus resolution is a critical process after deep venous thrombosis and the molecular mechanisms involved remain largely undefined. Prior studies in our laboratory indicate that selective cyclooxygenase 2 (COX-2) inhibitors, which have increased cardiovascular complications, impair thrombus resolution. COX-2 is involved in the generation of several prostaglandin products. The purpose of this study was to define the effects of several individual prostaglandins on thrombus resolution to better understand the basic mechanisms of thrombus resolution as well as the effects of COX-2 inhibitors in vascular biology.

Methods: CD-1 outbred mice underwent caval ligation and administration of a variety of prostanoids via Alzet minipumps, then were harvested at day 4 or 12 and measured for thrombus weight. Six groups were used: (1) normal saline vehicle with trace methyl acetate (used as controls for groups 3-5), (2) phosphate buffer vehicle with sodium hydroxide, pH 10 (used as control for group 6), (3) thromboxane (0.1 mg/kg/day), (4) misoprostol (prostaglandin E analog; .1 mg/kg/day), (5) iloprost (prostaglandin E [PGE] and prostaglandin I [PGI] analog; 0.1 mg/kg/day), and (6) PGI2 (0.1 mg/kg/day).

Results: Thromboxane decreased thrombus resolution, as assessed by thrombus weight on postoperative day 12 (control, 0.221 ± 0.064 mg/g [n = 10]; thromboxane, 0.359 ± 0.172 mg/g [n = 9]; $P = .03$). However, no difference was seen in thrombus resolution with the administration of misoprostol (control, 0.221 ± 0.064 mg/g [n = 10]; misoprostol, 0.249 ± 0.061 mg/g [n = 7]; $P = .368$), iloprost (control, 0.221 ± .064 mg/g [n = 10]; iloprost, 0.176 ± 0.088 mg/g [n = 9]; $P = .22$), or PGI (control, 0.286 ± 0.05 mg/g [n = 8]; PGI, 0.254 ± 0.092 mg/g [n = 9]; $P = .396$). To further investigate if thromboxane had an effect of thrombus formation, we compared thrombus weights at day 4 and found no difference (control, 0.84 ± 0.181 mg/g [n = 7]; thromboxane, 0.822 ± 0.199 mg/g [n = 8]; $P = .85$).

Conclusions: The administration of thromboxane, but not PGE, PGI, or their combination, had a significant effect on thrombus resolution. This effect of thromboxane could account for the difference seen previously between nonsteroidal anti-inflammatory drugs and COX-2 inhibitors, because the latter lacks inhibition of thromboxane. This study also identifies thromboxane as a novel potential inhibitory target to accelerate thrombus resolution.

Dyslipidemia Regulates Thrombospondin-1–Induced Vascular Smooth Muscle Cell Chemotaxis

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Objective(s): Nearly one-third of Americans have dyslipidemia, as assessed by decreased high-density lipoprotein (HDL) and increased low-density lipoprotein (LDL) levels. Dyslipidemia and increased oxidized LDL (oxLDL) levels are risk factors for atherosclerosis and restenosis due to intimal hyperplasia (IH) after balloon angioplasty. A key process in IH development is vascular smooth muscle (VSMC) cell migration. LDL and oxLDL stimulate VSMC migration and potentiate agonist-induced migration. In contrast, HDL inhibits VSMC migration to pro-migratory agonists. LDL may induce migration through its lysophosphatidic acid (LPA) moiety, and HDL may inhibit migration through its sphingosine-1-phosphate (S1P) moiety, which stimulates cyclooxygenase 2 (COX2)-dependent prostacyclin release. The effect of LDL, oxLDL, and HDL on thrombospondin-1 (TSP-1)–induced VSMC migration, however, has not been studied. One of the ways the matricellular glycoprotein, TSP-1, contributes to IH development is by inducing VSMC migration. For this study, the interaction of cholesterol components and TSP-1 on VSMC migration was determined. We hypothesized that HDL, S1P and would prostacyclin inhibit, and LDL, oxLDL, and LPA would augment TSP-1–induced VSMC chemotaxis.

Methods: Migration was assessed using a modified Boyden chemotaxis chamber. Quiescent VSMCs were pretreated for 20 hours with serum-free media (SFM), HDL (75 μg/mL), LDL (150 μg/mL), or oxLDL (10 μg/mL). Chemotaxis to SFM or TSP-1 (20 μg/mL) was determined. Next, the effect of S1P (1μM), LPA (1μM), and iloprost (a stable prostacyclin analog, 100nM) pretreatment for 20 minutes on TSP-1–induced chemotaxis was determined. Assays were performed in triplicate and analyzed by analysis of variance, with $P < .05$ as significant.

Results: TSP-1–induced VSMC chemotaxis was inhibited by HDL (−42%), whereas LDL (72%) and oxLDL (61%) augmented TSP-1–induced VSMC chemotaxis. S1P (−52%) and iloprost (−62%) inhibited, and LPA (39%) augmented, TSP-1–induced VSMC chemotaxis.

Conclusions: Cholesterol regulates TSP-1–induced VSMC chemotaxis. HDL inhibition of chemotaxis is likely due to S1P and its ability to stimulate COX-2–dependent prostacyclin release. LDL and oxLDL in-