**ABSTRACTS**

**FIBEROPTIC ANGIOGRAPHY: A METHOD FOR QUANTIFICATION OF INTRAVASCULAR THROMBOSIS.**

Christopher C. Johnson, M.D., James L. Ritchie, M.D., F.A.C.C., John R. Stratton, M.D., F.A.C.C., Dennis D. Fleichgenbe, M.D., University of Washington and VA Medical Center, Seattle, WA.

Application of thrombolytic therapies necessitates improved methods for detection and quantification of intravascular thrombi. Angiographically determined thrombosis was compared to platelet deposition and quantified by intradial platelet labeling. After platelet labeling and thrombus induction using over-sized PTCA balloons, porcine carotid arteries (n=12) were imaged every 0.3 centimeters (cm) by fiberoptic angiography. Arteries were sectioned post-mortem and well counted. Thrombus deposition by angiography was quantified by digitalized measurement of percent luminal area occluded by thrombus. Overall correlation between angiographic thrombus deposition and platelet cm per artery was excellent (r=0.83, p<0.001). Injured arteries without angiographically defined thrombus had 8.3x10^7/linear cm (Lcm) of artery. By comparison, an uninjured vessel had 1.7x10^8/linear cm (Lcm) (p<0.01) while 100% occlusion by angiography predicted 2.4x10^10/Lcm.

We conclude that angiography can provide a useful, semiquantitative measurement of intravascular thrombosis. Balloon injury without intraluminal thrombus is associated with a five-fold increase in platelet deposition within the vessel wall, not detectable by angiography.

**ALPHA-VASCULAR ACTIN Messenger IS INCREASED IN SPONTANEOUSLY HYPERTENSIVE RATS.**

Roth J. Stoka, M.D., Kelly M. Crawford, B.S., Alvin S. Blaufstein, M.D., James L. Leonard, Ph.D., University of Cincinnati Medical Center, Cincinnati, OH.

Spontaneously hypertensive rats (SHR) constitute a genetic model of hypertension. Since SHR aortas have increased medial thickening, we hypothesized that levels of messenger RNA (mRNA) encoding the α-vascular smooth muscle actin would be increased. We measured systolic BP (tail cuff) in 5 SHR and 5 age-matched control Wistar-Kyoto (WKY) rats. BP in the SHR group was significantly elevated (179±7 vs. 99±4 mmHg; mean±SEM; p<0.005).

Total cellular RNA was isolated from thoracic aortas and hearts for Northern blot analysis. The Northern blots were then probed with a labelled oligonucleotide, derived from rat α-vascular actin DNA, which hybridizes only with mRNA encoding α-vascular actin. To measure total actin mRNA we used a second probe which identifies mRNA containing coding sequences common to all actin isoforms. Autoradiography and computerized densitometry were used to compare SHR and WKY muscle actin mRNA levels.

We found more total muscle actin mRNA in both SHR aortas and hearts compared to WKY. SHR aortas contained 1.8 times more total muscle actin message and 4.4 times more α-vascular mRNA than WKY aortae. The increased α-vascular mRNA presumably accounts for the elevation in total muscle actin message. It remains to be determined whether these higher mRNA levels result in increased α-vascular actin content. Likewise, it is unclear if such changes in SHR aortas produce elevated vascular resistance, or whether they are the result of the increased mechanical stress of hypertension.

**AGING AND VASCULAR RESPONSIVENESS TO ENDOTHELIN (ET) IN SHR AND WKY**

Gian Paolo Rossi, M.D., Gabriella Cazگnelli, M.D., Sergio Bova, M.D., & Achille C. Pesonna, M.D., Ph.D., Departments of Clinical Medicine and Pharmacology, University of Padua, Padua, Italy.

We have verified the hypothesis that arteries of hypertensive rats are more responsive to endothelin by comparing the in vitro contractile response to porcine endothelin (ET) of endothelium-free aortic strips from young and matured WKY rats. In addition, the effect of aging was assessed by studying young (11wks) and old (20mos) animals. Cumulative concentration-response curves to endothelin (10^-10 - 5×10^-6 M) were obtained in the presence of indomethacin. Responses were expressed as percent of the maximal contraction to norepinephrine (10^-5 M).

**RESULTS:** No difference among groups in terms of E max was found as no difference could be observed in maximal responsiveness in young and WKY. By contrast, in old SHR a significantly blunted (p<0.005) response was observed as compared to young SHR and both old and young WKY. This was not paralleled by changes in responsiveness to norepinephrine. Thus, our data are not consistent with the hypothesis that arteries of genetically hypertensive rats respond less effectively to endothelin. The specific decrease of responsiveness to endothelin with age in SHR could be due to chronic hypertension.