**CORONARY, CEREBRAL, AND ABDOMINAL AORTIC ANEURYSMS: SIMILARITIES IN PATHOGENESIS AND IMPLICATIONS FOR TREATMENT**

**S.D. Gertz1**, L.Y. Gavish1, R. Beeri1, D. Gilon1, C. Rubinstein1, Y. Berlatzky1,

A. Bulut1, M. Harlev1, P. Reissman2, L. Gavish1

1Institute of Medical Research (IMRIC), Faculty of Medicine of The Hebrew University and Hadassah, Jerusalem, 2Faculty of Medicine of The Hebrew University and Hadassah, Shaarei Zedek Hospital, Jerusalem, Israel

Considerable evidence has accumulated identifying arteriosclerotic changes (atherosclerotic and/or arteritic) as the principle underlying pathogenetic process in the progression of acquired, non-iatrogenic coronary artery aneurysms (CAA), intracranial arterial aneurysms (ICA), and abdominal aorta aneurysms (AAA). Variable degrees of inflammatory cell infiltration, modification of matrix metalloproteinases, disruption of the internal elastic lamina and elastic lamellae of the media, and degeneration of smooth muscle cells are prominent features of all three regardless of the specific etiology. The well-documented association of ICA with arterial branch orifices of the circle of Willis has provided strong support for the role of hemodynamic forces in its progression and rupture even in the absence of systemic hypertension. The histopathological characteristics of human CAA and AAA have been determined primarily from specimens with advanced pathology in which the initial structural changes have been obscured. In this study we present the evidence from animal models, including the angiotensin-infused, apolipoprotein E-deficient mouse, as well as humans, that points to similarities between ICA, CAA, and AAA in the structural features of early arterial wall lesions and to arterial branch points and curvatures as sites of predilection. Although additional confirmation is necessary, local alterations in the magnitude or directionality of hemodynamic forces at such sites appears to be a common underlying pathogenetic mechanism in the early development and progression of acquired arterial aneurysms in all three locations. Implications for treatment will be discussed.