

The 1st Vulnerable Patient Satellite Symposium

Introducing a New Era in Preventive Cardiology

Annual Scientific Sessions of American Heart Association 2003, Orlando, FL



Bullet Points from Dr. Stone's Presentation

- While detection of already-vulnerable plaques is critical, it is equally important to identify areas in the coronary arteries that will become vulnerable (prone to rupture, thrombosis, or progression).
- Although cardiovascular atherosclerotic disease is a systemic abnormality with major metabolic and immune components, it does not occur evenly through the arterial beds. The distribution of atherosclerosis may vary in different arterial beds in the same person and may vary person to person. The dynamic pattern of blood flow and the morphology of the local segment of artery in 3-D space will determine the forces (shear stress) acting on that part of the artery, and these local shear stresses are likely responsible for the at least some of the local manifestation of the disease.
- Changes in shear stress directly affects the activity of endothelial cells. Decreased shear stress below the physiological range impairs endothelial function and may result in expression of certain inflammatory adhesion molecules. It also affects the permeability of endothelial layer and other properties of endothelial surface (i.e. antithrombotic and antiproliferative effects). Also it has been shown that excess free radicals are created at the site of low shear stress which may link the mechanical stress to biochemical oxidative stress and create the ground for development of atherosclerotic plaque.
- Therefore shear stress imaging may provide significant information to identify the trajectory of the natural history of atherosclerosis and to find the areas of the artery prone to development or progression of atherosclerosis. Normal physiological shear stress varies between 10-50 dyne/cm²
- Coronary shear stress can be visualized by combining coronary IVUS and angiography images using 3 D reconstruction of arterial segments and computational flow dynamic technology.
- Dr Stone's group has just completed a pilot study of 10 patients in whom they measured baseline coronary shear stress and coronary artery morphology, and then repeated the measurement at 6 month follow up. They discovered that areas with low shear stress at baseline developed progression of atherosclerosis in followup, with thickening of the coronary artery wall. This progression of local atherosclerosis was associated with outward remodeling of the outer artery wall, so that a luminal obstruction would not develop. Accordingly, local shear stress did not change. These observations of plaque progression and outward remodeling are exactly what was hypothesized by Glagov 15 years ago on the basis of an autopsy study. Areas of high shear stress at baseline, i.e.,

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some degree of existing obstruction and luminal narrowing, also developed outward remodeling in order to lower shear stress in that local area. This lowering of the local shear stress towards a more physiologic level would provide a more protective environment for the endothelial cells and the artery wall in that area.

- Therefore areas of low shear stress with substantial outward remodeling (~40% increase in the ratio of plaque area to outer artery or EEM area) may have a high likelihood of plaque progression and perhaps formation of vulnerable plaque. It may be that these local arterial areas are high risk and are the ones that should be considered for preemptive therapeutic interventions such as stenting.