The endothelium plays a vital role in vascular homeostasis acting as both a modulator and protective lining of blood vessels. Through the release of a variety of autocrine and paracrine factors including nitric oxide, the healthy endothelium is protective. Vasodilation, inhibition of platelet and leukocyte adhesion retard atherosclerosis progression and maintain plaque stability. It is now well recognized that in response to a variety of cardiovascular and inflammatory risk factors, nitric oxide bioavailability is attenuated. This shifts the balance with vasoconstrictor/mitogenic factors such as endothelin-1 and angiotensin II. The result is endothelial dysfunction, a condition that can be recognized in the laboratory or increasingly in the clinical research arena. A variety of research tools are now available to evaluate endothelial health in patients. Endothelial dysfunction is thought to play a role in a variety of clinical conditions including typical angina, vasospasm and microvascular angina, and atherosclerosis progression. In addition, endothelium-dependent vasodilation is being commonly used in clinical trials of new pharmacological therapy.

The current talk will concentrate on the use of measures of vasodilator endothelial function as surrogate markers of atherosclerosis activity and outcome. Emerging data would suggest that flow-mediated vasodilation, a marker of conduit vessel function might be related to cardiovascular outcomes. Markers of microvascular endothelial function such as hyperemic velocity and shear stress, pulse arterial tonometry and impedance plethysmography may be particularly helpful. The prognostic significance of endothelial function testing will be reviewed.