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NEW INSIGHTS INTO CARDIOVASCULAR RISK FROM THE EXERCISE CENTRAL WAVEFORM

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Career Development Lectures

NON-INVASIVE ASSESSMENT OF MECHANICAL PROPERTIES AND COMPOSITION OF VULNERABLE CAROTID PLAQUES

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Background: Plaque rupture mechanisms are complex processes, which are dependent on plaque morphology and composition and mechanical characteristics. We have previously demonstrated that the higher risk of plaque complication reported in patients with diabetes and hypercholesterolemia may be due to a specific pattern of strain gradient between plaque and adjacent common carotid artery (CCA) (inward bending stress). In addition, previous studies have suggested that plaque echogenicity is related to the histological components of carotid plaques and that carotid plaque echolucency (low echogenicity) is associated with the development of neurological events.

Objective: of our study was to determine the difference in plaque composition according to the presence of an outward or an inward bending stress (ArtLab system) using an *in vivo* noninvasive approach with a software for videodensitometry analysis, the MIP system (Medical Image Processing, CNR Pisa), which yields the gray levels distribution of a region of interest (ROI).

Patients: The study included 24 patients with a recent cerebrovascular ischemic event and either a plaque on the far wall of CCA. We divided the patients into two groups according to strain behaviour: pattern A (larger strain at the level of plaque than at the level of CCA, outward bending strain), pattern B (plaque strain smaller than CCA strain, inward bending strain). In all patients digital images of plaques were analyzed offline with MIP.

Results: In 16 patients a pattern A and in 8 patients a pattern B strain behaviour were observed. Demographic and hemodynamic characteristics were similar in the two groups. The prevalence of hypercholesterolemia and diabetes were significantly higher in patients with a pattern B strain behaviour (respectively, 100% vs 56.3% $p=0.03$ and 62.5% vs 12.5%, $p=0.04$). Mean gray levels were significantly lower in patients with an inward bending strain compared to patients with an outward bending strain (65 ± 20 vs 95 ± 30 , $p=0.018$).

Conclusions: These results suggest that the higher risk of plaque complication, reported in patients with diabetes and hypercholesterolemia, may be due to a specific pattern of strain gradient between plaque and adjacent CCA and to a different plaque composition.

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NEW INSIGHTS INTO CARDIOVASCULAR RISK FROM THE EXERCISE CENTRAL WAVEFORM

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Abstract: Numerous studies in healthy individuals with normal office blood pressure (BP) have shown that a hypertensive response to exercise predicts

the future onset of hypertension, as well as cardiovascular morbidity and mortality, independent of office BP. The mechanisms underlying the predictive value of exercise BP are incompletely understood. However, it has been proposed that the additional cardiovascular stress imposed by exercise may unmask the presence of concealed hypertension. A new non-invasive method of exercise arterial pressure waveform analysis (and central BP estimation) may provide additional clinical information, as well as insight into mechanisms, beyond the BP obtained by traditional upper arm cuff methods.

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MacDonald Lecture

WALL SHEAR STRESS REVISITED

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Wall shear stress (WSS), the drag of the flowing blood exerted on the endothelial cells, is an important determinant of endothelial function, structure and gene expression. It also plays a role in atherogenesis. Based upon theory WSS is considered to be constant along the arterial tree and similar in a particular artery across species.

In vivo measurements in humans and animals, however, show that the theoretical assumptions regarding WSS and its calculation are far from valid. In humans mean WSS is higher in the common carotid artery (1.1-1.3 Pa) than in the brachial (0.4- 0.5 Pa) and femoral (0.3-0.5 Pa) arteries; the latter values being substantially lower than the theoretically predicted value of $1.5 \pm 50\%$. The lower mean WSS in these conduit arteries can be explained by the high peripheral resistance in these arteries, reducing mean volume flow and inducing reflections. In the femoral artery, adaptation of the peripheral resistance during vasodilatation results in mean WSS values close to those in the common carotid artery. Mean WSS also varies within the carotid and femoral artery bifurcations. Also in animals mean WSS varies along the arterial tree. Mean WSS was found to vary between 2.0 and 10.0 Pa in mesenteric arterioles and between 2.0 and 3.0 Pa in cremaster arterioles, the values being dependent on the site of measurement along the arteriolar tree. Across species mean WSS in a particular artery decreases linearly with increasing body mass. For example, in the descending aorta from 8.8 Pa in mice, to 7.0 Pa in rats and to 0.5 Pa in humans; the flow velocities in these artery being similar in these species.

The observation that mean WSS is far from constant along the arterial tree indicates that Murray's cube law on flow-diameter relations in bifurcations cannot be applied to the whole arterial system. At the present state of the art, it may be concluded that the exponent of the power law varies along the arterial tree, from 2 in the major branches of the aortic arch to 3 in arterioles. The *in vivo* findings also imply that *in vitro* investigations on endothelial gene expression and cellular adhesion the endothelial cells derived from different vascular areas or from the same artery from different