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New insights in atherosclerosis: Endothelial shear stress as promoter rather than initiator

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Abstract

The etiology of focal distribution of atherosclerotic lesions has received much attention for many years. Current theories focus on mechanical factors such as low endothelial shear stress as an initiating factor for atherosclerosis formation. However, some evidences revealed that it could not be initiator of endothelial damage. We hypothesize that endothelial damage results from the fatigue effect of pulse pressure on endothelial layer. In our model, heart rate, magnitude of pulse pressure, geometry and chemical environment of endothelial layer determine the rate of endothelial damage accumulation, and low endothelial shear stress acts as promoter of atherosclerosis rather than initiator. If this model is correct, it can provide a framework for speculating about the risk of endothelial stress rupture in the population as a whole and in patients undergone arterial grafting procedures, and how this might be reduced.

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