Abstract

Endothelial dysfunction has been associated with many pathophysiological processes, such as inflammation and oxidative and nitrosative stresses, endothelial cells are precociously exposed to circulating signaling molecules and physical stresses, like in sepsis and septic shock.

Impairment of endothelial function leads to phenotypic and physical changes of the endothelium, with deregulated release of potent vasodilators nitric oxide and prostacyclin, reduction of vascular reactivity to vasoconstrictors, associated with leukocytes and platelets aggregation, and increase in inducible nitric oxide synthase expression that can exert a negative feedback on endothelial nitric oxide synthase expression, with subsequent deregulation of nitric oxide signaling.

Endothelial dysfunction therefore plays a major role in the pathophysiology of septic shock and organ dysfunction, and has been suggested to be a predictor of mortality in sepsis. Thus, early detection of endothelial dysfunction could be of great interest to adapt treatment in initial stage of sepsis. Current therapeutics used in sepsis mostly aim at controlling inflammation, vascular function and coagulation.

Ultrasound measurements of brachial artery reactivity in response to stagnant ischemia provide estimates of micro vascular function and conduit artery endothelial function and would therefore predict sepsis and severe sepsis outcome.

Key words :

endothelial function – Sepsis – Ultrasound .