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### **Inflammation and reduced endothelial function in the course of severe acute heart failure.**

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#### **Abstract**

Systemic inflammation and elevated circulating levels of the endogenous nitric oxide inhibitor asymmetrical dimethylarginine (ADMA) have been associated with increased risk in cardiogenic shock (CS). In this prospective study, we assessed, over 4 consecutive days, the changes and possible associations between vascular function, markers of inflammation, and circulating ADMA levels in patients with CS (n = 12) and postcardiotomy heart failure (n = 12, PC-HF). Vasodilator function was measured as a reactive hyperemia index (RH-index) using a finger plethysmograph. Blood samples were analyzed for plasma ADMA, interleukine-6, interleukine-8, intracellular adhesion molecule-1, and vascular adhesion molecule-1. Baseline RH-index was significantly attenuated compared with healthy controls (2.28) for both CS and PC-HF (1.35 and 1.45, respectively, P = 0.001). Although vasodilator function improved in PC-HF patients, it remained attenuated in CS. Inflammatory markers were markedly elevated followed by a significant fall during the observation period in both groups. ADMA levels increased significantly during the observation period for PC-HF, whereas no pattern of change was observed for CS. No association was found between the longitudinal changes in RH-index, markers of inflammation, or ADMA in CS. However, an improved RH-index was associated with decreasing inflammatory markers in PC-HF. ADMA correlated to arterial lactate levels and the degree of organ dysfunction in CS. In conclusion, CS and PC-HF were characterized by a marked inflammatory activation accompanied by an attenuated vasodilator function. ADMA was related to organ dysfunction and degree of hypoperfusion during CS but showed no correlations to inflammation or hampered vasodilator function. The pathogenic significance of these responses needs clarification.

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