CIRCULATORY SHOCK

Volume 34, 1991

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Editor

605 Third Avenue
New York, NY 10158-0012

WILEY-LISS
A JOHN WILEY & SONS, INC., PUBLICATION
New York • Chichester • Brisbane • Toronto • Singapore

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Volume 34, Number 1 **May 1991**

Second International Conference on Shock
Fifth Meeting of European Shock Society
Fourteenth Annual Meeting of the Shock Society (USA)
Third Vienna Shock Forum
June 2–6, 1991
Vienna, Austria

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CLINICAL RELEVANCE OF MONITORING PLASMA LEVELS OF PROTEINASES, PROTEINASE INHIBITORS, AND CYTOKINES IN THE COURSE OF MULTIPLE ORGAN FAILURE

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Repair and healing or perpetuation of inflammation in response to inflammatory noxae (e.g. multiple trauma, infection) depend on complex interactions of humoral and cellular defence mechanisms. Out of the various inflammatory parameters investigated hitherto, proteolytic enzymes, both of the plasma cascade systems (plasma kallikrein, thrombin, plasmin, complement esterases, etc.) and of lysosomal or granular origin (PMN elastase, macrophage cathepsin B, mast cell tryptase) have been shown to be potent effectors of destructive processes contributing to the occurrence of multiple organ failure (MOF) in severe posttraumatic and postoperative courses. Such proteolysis-induced pathomechanisms are greatly enhanced by the concurrently arising (local) imbalance between proteinases and their inhibitory regulators (e.g. α_1-proteinase inhibitor, α_2-macroglobulin, antithrombin III, PAI-1, C1-inactivator). In this respect, cytokines such as TNF or NAP (IL-8) seem to be relevant candidates for sequestration and activation of PMN granulocytes thereby increasing significantly the proteinase burden at the inflammatory focus. In several clinical studies on patients suffering from multiple trauma and/or septicemia we could demonstrate that measurement of cell-derived proteinases (PMN elastase, cathepsin B) and factors of the blood cascade systems (prothrombin, antithrombin III, protein C, C1- inactivator, PAI-1, etc.) in consecutive plasma samples turned out to be a helpful tool for early diagnosis and prognosis of severe multiple organ failure. In contrast, plasma levels of TNF and NAP monitored similarly showed only minor diagnostic significance. Yet, quantification of these cytokines in local body fluids (e.g. bronchoalveolar lavage fluids) clearly indicate their pathogenetic relevance for the development of organ failure.