

CIRCULATORY SHOCK

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James P. Filkins
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Contents

Volume 34, Number 1

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Program Committee	1
Acknowledgments	3
Program	5
Abstracts	7
Author-Abstract Index	171
Directory	174
Constitution	176
Membership Directory	181
Announcement	216



Volume 34, Number 2

June 1991

Hypotension- and Endotoxin-Induced Alterations in Calcitonin Gene-Related Peptide: Modulation by Dexamethasone X. Wang, C.D. Han, R.R. Fiscus, M. Qi, and S.B. Jones	217
Role of Bradykinin in Ovine Endotoxemia Roberta Mann, Lee C. Woodson, Lillian D. Traber, David N. Herndon, and Daniel L. Traber	224
In Vitro Phosphatidylcholine Peroxidation Mediated by Activated Human Neutrophils Jerry J. Zimmerman and June R. Lewandoski	231
Effects of RA642, A New Central Pressor Agent, on Renal Sympathetic Nerve Activity and Blood Pressure During Hemorrhagic Shock in Rabbits Mayuki Aibiki, Shozo Koyama, and Takashi Fujita	240
Amrinone During Porcine Intraperitoneal Sepsis James B. Hermiller, John P. Mehegan, Vinay M. Nadkarni, J. Alan Paschall, Joseph J. Nevola, Mark A. Fletcher, and Taffy J. Williams	247
Awake Porcine Model of Intraperitoneal Sepsis and Altered Oxygen Utilization Lynne D. Hoban, J. Alan Paschall, Jon Eckstein, David Reusch, James Hermiller, Byron Rowe, Joseph J. Nevola, and Joseph A. Carcillo	252

Therapeutic Intervention in a Rat Model of Adult Respiratory Distress Syndrome: II. Lipoxygenase Pathway Inhibition	
Claudia R. Turner, Margaret N. Lackey, Melanie F. Quinlan, Donald E. Griswold, Lester W. Schwartz, and Eric B. Wheeldon	263
Therapeutic Intervention in a Rat Model of Adult Respiratory Distress Syndrome: III. Cyclooxygenase Pathway Inhibition	
Claudia R. Turner, Margaret N. Lackey, Melanie F. Quinlan, Lester W. Schwartz, and Eric B. Wheeldon	270
Conjugated Desferoxamine Attenuates Hepatic Microvascular Injury Following Ischemia/Reperfusion	
George T. Drugas, Charles N. Paidas, Alan M. Yahanda, Dayna Ferguson, and Mark G. Clemens	278

Volume 34, Number 3

July 1991

Microvascular Exchange During Burn Injury: IV. Fluid Resuscitation Model	
J.L. Bert, B.D. Bowen, R.K. Reed, and H. Onarheim	285
Characterization of an Endotoxemic Baboon Model of Metabolic and Organ Dysfunction	
D. Craig Lindsey, Thomas E. Emerson, Jr., Thomas E. Thompson, Andrea E. John, Melinda L. Duerr, Cecelia M. Valdez, Harnng S. Kuo, Robert B. Bouffard, Russell G. Irwin, Deogracias Canivel, and Michael A. Fournel	298
Hyperdynamic Sepsis in Baboons: I. Aspects of Hemodynamics	
Günther Schlag, Heinz Redl, Seth Hallström, Keith Radmore, and James Davies	311
Extracellular Muscle Surface pO₂ and pH Heterogeneity During Hypovolemia and After Reperfusion	
F. Sjöberg, U. Gustafsson, and D.H. Lewis	319
Molecular Biology of Circulatory Shock: IV. Translation and Secretion of HEP G2 Cell Proteins Are Independently Attenuated During Heat Shock	
Antonio De Maio and Timothy G. Buchman	329
Resuscitation of Hypovolemic Sheep With Hypertonic Saline/Dextran: The Role of Dextran	
John C. Walsh and George C. Kramer	336
Influence of Pentoxifylline and Related Analogues in Endotoxemic Renal Failure	
Kurt L. Berens, Jeffrey D. Langston, Kishor M. Wasan, and David R. Luke	344

Volume 34, Number 4

August 1991

Plasma Kallikrein Generation in Endotoxemia Is Abolished by Ultra High Doses of Methyl-Prednisolone: In Vivo Studies in a Pig Model	
Frode Naess, Olav Roeise, Johan Pillgram-Larsen, Tom Erik Ruud, Jan O. Stadaas, and Ansgar O. Aasen	349
Intraabdominal Sepsis: Enhanced Autooxidative Effect on Polymorphonuclear Leukocyte Cell Surface Receptor Expression	
H. Hank Simms, Ron D'Amico, and Kenneth W. Burchard	356

Relationship Between the Lung and Systemic Response to Endotoxin: Comparison of Physiologic Change and the Degree of Lipid Peroxidation	
Robert H. Demling, Cheryl LaLonde, Ramona Daryani, Deguang Zhu, James Knox, and Yeo-Kyu Youn	364
Effect of Cerebral Ischemia on Hypotension-Induced Increase in Plasma Vasopressin and Hepatic Glycogen Concentration in the Rat	
Bente R. Karlsson, Bjarne Grøgaard, Urban Höglund, Bengt Gerdin, and Petter A. Steen	371
Modification of Pituitary-Adrenal Axis Responses to Hemorrhage by Handling Techniques in Conscious Swine	
Charles E. Wade and John P. Hannon	379
Effect of LTB₄ Receptor Antagonists in Endotoxic Shock in the Rat	
E.J. Li, J.A. Cook, W.C. Wise, W.T. Jackson, and P.V. Halushka	385
Vagotomy Alters the Hemodynamic Response of Dogs in Hemorrhagic Shock	
Eric R. Schertel, Amy K. Valentine, L. Michael Schmall, David A. Allen, and William W. Muir	393
Absence of the Staub-Traugott Effect in Endotoxicosis	
Hideo Inaba and James P. Filkins	398
TRENDS IN SHOCK RESEARCH	
Oxygen-Free Radicals (OFR) and Circulatory Shock	
Ulf Haglund and Bengt Gerdin	405
Author Index to Volume 34	412
Subject Index to Volume 34	413

201

202

203

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

M. Jochum¹, D. Inthorn³, Th. Joka⁴, W. Machleidt², Ch. Waydhas³, H. Redl⁵, H. Fritz¹ Department Clinical Biochemistry¹, Physiological Chemistry², and Surgical Clinics³ of the University of Munich; Surgical Clinic⁴ of the University Essen, Germany. L. Boltzmann Institute for Experimental and Clinical Traumatology⁵, Vienna, Austria.

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.