Multisystem Organ Failure
MSOF

Essay

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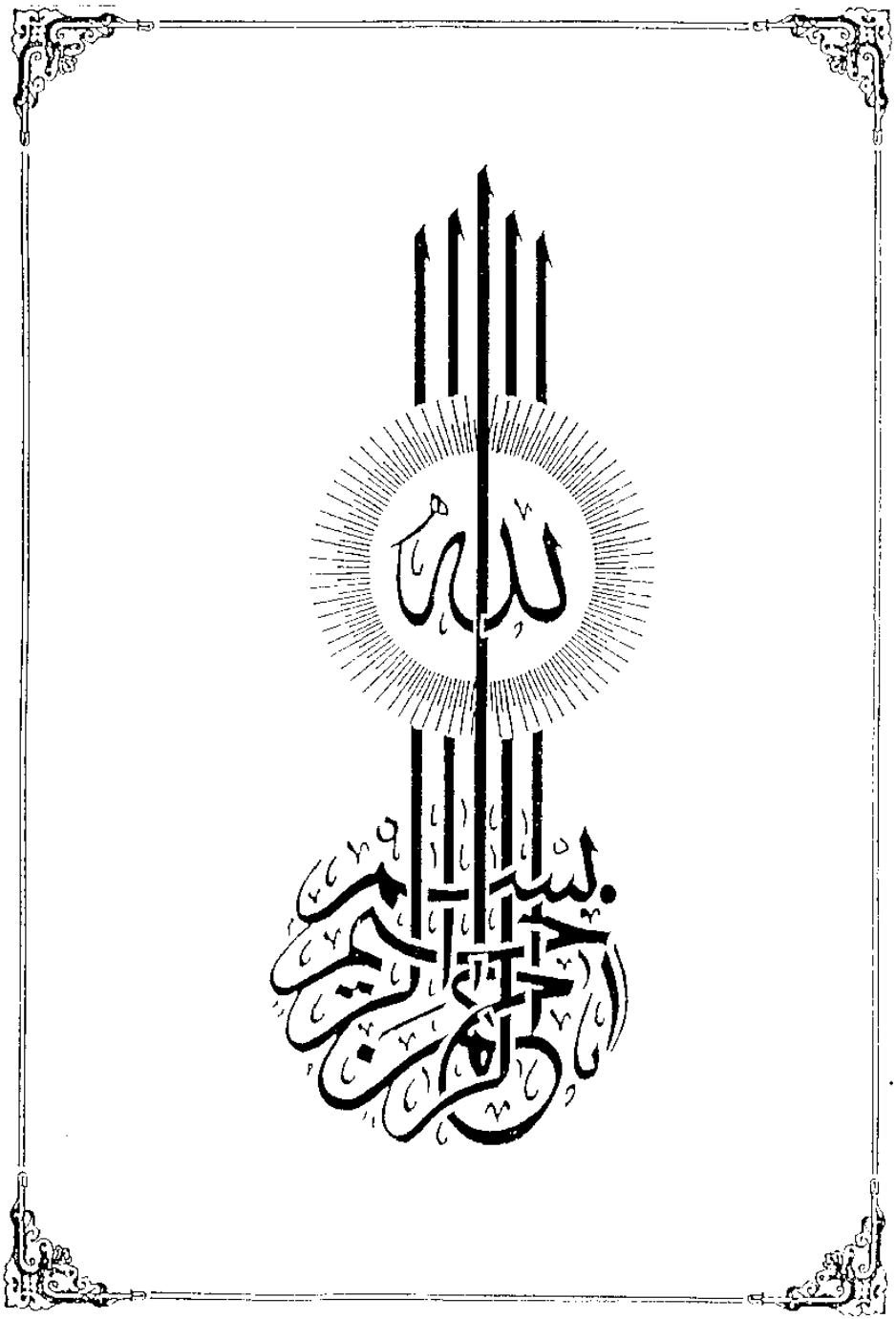

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To

My Parents,

My Wife

&

My Little Ahmed

List of Tables

List of Figures

Contents

Introduction

The past two decades have witnessed the emergence of a new syndrome termed Multi-system Organ Failure (MSOF) which today represents the number 1 cause of death in surgical intensive care units. The basic pathophysiology of this syndrome remains to be fully elucidated ***(Deitch, 1990)***.

Thus, at a time when spectacular advances in the field of organ transplantation have revolutionized the therapy of patients with end-stage single organ failure, our inability to successfully treat patients with acutely failed organs remains the major unsolved problem in the critically ill post-operative or post-injury patient ***(Fry, 1992)***.

In many ways it is not surprising that, as our ability to support organ function and prolong survival in patients with highly lethal conditions has improved, we have uncovered new and unexpected clinical problems such as MSOF, for which no definitive therapeutic answers are initially available. Although there is no substitute for good surgical technique and mature clinical judgment in optimizing survival, until we understand the basic pathophysiology of MSOF, our ability to devise rational and effective therapeutic options will be severely constrained ***(John, 1992)***.

We have to remember that the role of the surgeon in the ICU has the potential to move in two different directions. Inattention or a lack of commitment by surgery which will lead to withdrawal from the critical care setting ***(Walt, 1985)***. This would be a significant loss for surgery as well as for future generations of surgeons. Surgery instead may commit to a greater interest and involvement in ICUs, which would help to continue the surgeons active role in all aspects of medical progress ***(Edmund, 1992)***.

Review of Literature

Historical Background

One characteristic of surgical progress has been the identification, investigation and subsequent conquest of new clinical problems. In world war I, the causal relationship between acute blood loss and irreversible "Wound shock" was not known, and consequently acute shock was a common cause of death. Based on studies performed in the post-world war I period establishing the role of acute blood loss in the development of shock, blood was used liberally during world war II to prevent and treat shock, thereby largely eliminating the previously common syndrome of irreversible wound shock ***(Deitch, 1990)***.

In spite of improved early survival, many of these patients went to die of acute renal failure. During the Korean war, acute renal failure remained the most common cause of delayed death in successfully resuscitated patients and therefore was a focus of intense investigations on the pathophysiology of post-traumatic renal insufficiency which eventually led to the realization that injury induced renal failure could be largely prevented by resuscitating these patients with sufficiently large amounts of crystalloids (to maintain renal perfusion) in addition to blood.

Thus based on this better understanding of the physiology of injury and the need for acute volume resuscitation, acute

renal failure was largely prevented in the Vietnam conflict ***(Shires, 1972)***.

As more severely injured patients survived for longer periods, however, a new syndrome {Adult respiratory distress syndrome(ARDS)} emerged, and in the 1970s, the lungs became the organ system limiting survival ***(Ashbaugh, 1967)***.

Today, although the mortality rate of patients with ARDS remains high, improvements in the management of these patients have resulted in the cause of death shifting from impaired gas exchange to MSOF, and currently more than 75% of the patients dying with ARDS now die of MSOF and systemic hemodynamic instability rather than of hypoxia. Viewed in this light, MSOF clearly represents the next, but surely not the last, obstacle that must be passed to improve survival in the critically ill surgical patient ***(Montgomery, 1985)***.

General Concept

MSOF is a nonspecific expression of critical illness involving progressive failure of two or more organ systems, which is driven by the presence of numerous circulating mediators ***(Carrico, 1986)***.

Many investigators believe the presence and activity of numerous mediators provide the common pathway to the development of MSOF, with or without septicemia and shock ***(Goris, 1985)***.

But from where do these mediators come and why are they released? Following bodily insult, specific primary events occur that trigger the release of mediators into the circulation from various sources as the body seeks to protect itself and recover from the insult. The primary events are:

1) Neuroendocrine activation.
2) Activation of inflammatory / immune response.
3) Endothelial damage ***(Nuytinck, 1988)***.

1. Neuroendocrine activation:

Following an insult, activation of the neuroendocrine system stimulates the release of numerous substances into the circulation, including ACTH from the anterior pituitary,