Meeting report

The 32nd Annual Congress of the Society of Critical Care Medicine, 28 January – 2 February 2003, San Antonio, USA

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Published online: 6 March 2003 *Critical Care* 2003, **7**:193-195 (DOI 10.1186/cc2173)
This article is online at http://ccforum.com/content/7/2/193

Keywords ARDS, evaluation, hemodynamics, multiple organ failure, proteomics

The 32nd Annual Meeting of the Society of Critical Care Medicine was once again a well attended and very interesting meeting. The distinguished faculty of well recognized clinical and basic scientists from all parts of the world presented multiple sessions highlighting the tremendous advances being made in our understanding and treatment of a wide variety of problems encountered in critically ill patients. In addition, more than 600 free communications were presented. These were too wide to be covered in the present report. Rather, we focus on the seven outstanding plenary sessions in which distinguished speakers shared their views on the present and future of critical care practice and research.

Proteomics, system biology and the future of drug design

Michael B Yaffe (Cambridge, MA, USA)

In his lecture, Michael Yaffe alluded to how fundamental science may help in the development of new therapeutic agents. In the past, the search for new drugs was based on random selection of drugs of natural sources, which were secondary tested in all possible models. When the drug was proven to have efficacy, its chemical structure was isolated and the drug was then synthesized. Using this method, unfortunately, the rate of drug discovery has decreased. A more rational approach would be to take into account our understanding of the mechanism of diseases in order to identify target signalling pathways. Use of genomics to identify gene expression of cells in different conditions is unlikely to be helpful because the responses to several stimuli are often similar. For example, gene expression in macrophages is similar following stimulation by Gram-negative bacteria, Gram-positive cocci and mycobacteria. Proteomics may be more helpful in identifying regulatory pathways. Proteomics covers thousand of proteins acting in a changing environment, and hence it is important to identify how and why several proteins are interrelated and interplay in regulatory and/or pathophysiological processes. This leads to the possibility of identifying critical nodes in models and definition of where drugs are likely to act.

Innate immunity and sepsis

Thierry Calandra (Lausanne, Switzerland)

Thierry Calandra reviewed the mechanisms involved in innate immunity, with a special emphasis on the Toll-like receptor. He also stressed the important role played by the pituitary axis in the modulation of inflammation. In this context, the macrophage inhibitory factor (MIF) appears to be crucial. MIF is high in patients surviving sepsis, and the administration of MIF increases survival in experimental models. Interestingly, MIF is implicated in the hypothalamic–pituitary–adrenal axis, being present in the adrenal cortex, whereas dexamethasone induces MIF secretion.

Multiple organ success

Mervyn Singer (London, England)

In a rather provocative talk, Mervyn Singer defended the concept of 'multiple organ success'. There is no doubt that early haemodynamic resuscitation using the so-called 'early goal-directed therapy' approach is crucial in severe sepsis and septic shock, but what about late optimization? The classic appreciation of the pathogenesis of multiple organ failure is of a succession of events that lead from microcirculatory alterations and systemic inflammation to mitochondrial inhibition and shutdown, resulting in cellular bioenergetic failure and finally multiple organ failure.

ACTH = adrenocorticotrophic hormone; ARDS = adult respiratory distress syndrome; MIF = macrophage inhibitory factor; T_3 = tri-iodothyronine.

Nevertheless, a recent histological autopsy study failed to show dramatic histological lesions in patients dying from septic shock. There was little cell death and no correlation between histological findings and the severity of septic shock. Sepsis-induced mitochondrial inhibition explains low oxygen consumption in the presence of high tissue oxygen tension, as reported in experimental work. This decrease in oxygen consumption could represent an adaptive response to sepsis; this is because it has been described in hypoxic conditions, in which the decrease in oxygen consumption was the result of a decrease or abolition of nonessential cell functions, and preservation of vital functions that protect cell integrity. In this context, pushing metabolism by exogenous interventions may be deleterious.

ARDS physiology: an update

Luciano Gattinoni (Milano, Italy)

Mortality related to adult respiratory distress syndrome (ARDS) ranges from 35% to 65%. Substantial progress has been made in our understanding of how mechanical stress can injure the lung, both by altering barrier properties of the pulmonary endothelium and epithelium and by stimulating proinflammatory responses of macrophages and neutrophils, leading to the release of inflammatory mediators either in the alveolar space or in the systemic circulation. Two major determinants of lung injury associated with mechanical ventilation have been clearly identified, namely high pressure/high volume and the shear forces caused by intratidal collapse and opening of the alveoli. The lung protective strategy aims to reduce the impact of both of these determinants. Many experimental animal studies have addressed these concepts. Luciano Gattinoni discussed more particularly the type of animal models used in these experimental studies, specifically the size of the animals (mice versus sheep) and whether the lungs were 'preinjured' before mechanical ventilation was applied. The clinical relevance of experimental ventilator-induced lung injury recently received a resounding illustration from the ARDS Network Trial, which showed a 22% reduction in mortality in patients with ARDS when lung mechanical stress was lessened by tidal volume reduction during mechanical ventilation. Nevertheless, in that study, the researchers did not apply a 'full' lung protective strategy and did not take shear forces into account. Gattinoni reminded us of the concept of baby lung in ARDS and the potential deleterious effect of positive end-expiratory pressure application, leading to hyperinflation in healthy parts of the lungs. In this context, the use of pressure–volume curve monitoring at the bedside (especially, identifying the presence or absence of a lower inflection point) could provide some information on the morphology of the lung injury. Finally, in response to the recent meta-analysis conducted by Eichacker and coworkers [1], Gattinoni elegantly demonstrated, using the ARDS Network Trial data, that the decrease in mortality in patients ventilated with the protective strategy was due to the reduction in tidal volume rather than any potential effect on airway pressure.

New advances in the resuscitation of surgical patients

Elliot Bennett Guerrero (New York City, USA)

In his lecture, Bennett Guerrero revealed how perioperative haemodynamic optimization in high-risk surgical patients is beneficial in terms of morbidity and mortality, provided that the optimization protocol is well defined and standardized. In this context, the study conducted by Sandham and coworkers [2] was commented on and criticized. The absence of a well standardized protocol and the lack of a significant increase in oxygen transport in the protocol group before surgery, the low mortality rate in both groups, and the low recruitment rate were highlighted. In view of these points, it appears that the only conclusion that we can draw from the study is that, even in this population with a low mortality rate, the insertion of a pulmonary arterial catheter is not associated with an increase in mortality. During the perioperative period, the emphasis was placed on a more proactive (as opposed to reactive) approach by the anaesthesiologist (e.g. in face of the appearance of systemic hypotension). The choice of fluid for resuscitation appears to be of importance. In a study including 200 cardiac surgery patients, the use of hydroxyethyl starch in balanced solution for perioperative haemodynamic optimization was associated with less postoperative pain and nausea, less coagulation disorders, and improvement in renal function as compared with the use of hydroxyethyl starch in saline solution.

Endocrine changes in the critically ill: to treat or not to treat?

Greet Van den Berghe (Leuven, Belgium)

Until now, several experimental studies using a therapeutic approach aimed at normalizing the endocrine response have failed to demonstrate efficacy. However, interest in such interventions was recently renewed by the demonstration of the beneficial effects of tight control of glucose levels by high doses of insulin, and of enhancing adrenal function in relative adrenal insufficiency by administering low doses of hydrocortisone. Greet Van den Berghe addressed this apparent dilemma. The endocrine response to acute illness is biphasic. The acute phase is characterized by a decrease in growth hormone, and a decrease in tri-iodothyronine (T_3) with an increase in reverse T_3, whereas thyroxin-releasing hormone levels are constant; adrenocorticotrophic hormone (ACTH) is increased and cortisol is stable. On the contrary, in protracted illness, growth hormone increases and T_3 decreases markedly, whereas reverse T_3 is stable and thyroxin-releasing hormone is decreased; ACTH and cortisol are both low. Are these alterations adaptive (and hence protective), or do they participate in multiple organ failure? New animal models reproducing these late alterations are currently being developed, and such models will help us to answer the burning question – should we treat endocrine alterations in critically ill patients?

Changing physician behaviour in the intensive care unit

Deborah Cook (Hamilton, ON, Canada)

Changing the behaviour of physicians is a difficult process that involves a number of steps. First, we should understand our environment and our current behaviour. Second, we must be explicit about the target behaviour to be changed. This aspect is essential and can be addressed by successively raising the following questions. What aspect of behaviour should be changed? Who should be encouraged to change their behaviour (individuals have different threshold for different things – there are innovators and laggers)? Where should changes be made (in meetings or teaching at the bedside)? How (driving versus restraining force) should changes be instituted? Finally, why should the specific behaviour be changed? Education is fundamental because changes are made by groups, not by individuals. Improvement initiatives should be implemented, with feedback. Do not hesitate to use own data to sensitize your group to their actual behavior. At the end, an evaluation should again be performed to assess the effectiveness of the changes, preferably using a checklist to evaluate the application of the proposed changes and to establish the reasons why the changes were ultimately not applied.

Competing interests

None declared.

References

1 Eichacker PQ, Gerstenberger EP, Banks SM, Cui X, Natanson C: **Meta-analysis of acute lung injury and acute respiratory distress syndrome trials testing low tidal volumes.** *Am J Respir Crit Care Med* 2002, **166**:1510–1514.

2. Sandham JD, Hull RD, Brant RF, Knox L, Pineo GF, Doig CJ, Laporta DP, Viner S, Passerini L, Devitt H, Kirby A, Jacka M, for the Canadian Critical Care Clinical Trials Group: **A randomized, controlled trial of the use of pulmonary-artery catheters in high-risk surgical patients.** *N Engl J Med* 2003, **348**:5–14.