Letters

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ICU Follow-up Clinics: A New Frontier in Inter-Professional Collaborative Practice

Ariel M Modrykamien has provided an excellent review of the long-term consequences of treatment in the ICU and the importance of developing ICU clinics to provide comprehensive care to ICU survivors.[1] The long-term outcomes of post-ICU admission include reductions in quality of life, lung function, and nutritional status, and impacts on psychological outcomes and cognition. Patients and their close relatives are affected by the trauma of ICU experiences.

ICU follow-up clinics emerged in the United Kingdom, Norway, and Sweden around 1990. "Intensive care aftercare" was introduced in 2002 as a collaborative effort by a nurse and a physician in the United Kingdom,[2] but, as pointed out, most clinics were led by nurses. Many follow-up clinics were initiated by nurses, who also provided intensive care diaries for the patients, to help them come to terms with their ICU experiences, many of which they had no recall.[3] Patient diaries assist the patient in reconstructing the illness narrative.[4] A randomized controlled trial in 6 European countries demonstrated that diaries reduced new-onset post-traumatic stress disorder following critical illness.[5]

My main concern in the presentation of the follow-up clinic as a new paradigm for intensivists is that physicians are encouraged to "take over" a practice that was pioneered by nurses. Follow-up clinics are indeed an area that calls for inter-professional collaborative practice, including nurses, physicians, respiratory therapists, physiotherapists, psychologists, and others. It is a shame if the concept of ICU follow-up becomes medicalized, and fails to recognize the emotional and existential aspects of post-ICU care.

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The author replies:

I appreciate that Dr Egerod has taken the time to read my review[1] and shared her view and experience on this interesting and growing topic, the ICU follow-up clinic.

The concept of following post-ICU patients after hospital discharge originated in Europe, about 20 years ago.[2] As mentioned in Dr Egerod's letter, these clinics have been led by nurses, and then evolved to include participation by a number of other specialties such as physical, respiratory, and speech therapists, nutritionists, pharmacists, and social workers.[3]

Dr Egerod's concern is that my review may suggest that physicians are encouraged to "take over" a practice that was pioneered by nurses. In my opinion, the review is far from supporting the aforementioned statement. Conversely, it suggests that a multidisciplinary team should follow these patients.

The review mentions that in the U.S.A. the concept of an "ICU follow-up clinic" remains in its infancy. We are still learning from prior ICU clinic experiences, but we also recognize that healthcare systems present several differences among countries. Therefore, adjustments and variations from pioneer models are usually required. In our clinic, we promote participation from many services. Our clinic design is truly oriented toward a constructive and comprehensive "add on" rather than the implications of an isolated and competitive "takeover." This is the philosophy I aimed to communicate in the ICU follow-up clinic review.

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The Role of Transient Epithelial Ion Transport Reduction in Rapidly Reversible Pulmonary Edema

In a recent symposium paper including an explanation of the pathophysiology and histopathology of ARDS, the author stated that the early phase of acute lung injury is characterized by leakage of protein-rich edema fluid into the lung.[1] This is not the case: a recent study showed that in this phase of ARDS the pulmonary leak index, as calculated by measuring extravasation of gallium-labeled transferrin, was not elevated.[2]

A key mechanism in early ARDS is reduced pulmonary fluid clearance, which is a predictor of mortality[3] and caused by a reduction in respiratory epithelial sodium and chloride transport removing water from the alveolar space through osmosis.[4] Transient ion channel dysfunction caused by inflammatory mediator induced reversible nitrosylation or phosphorylation[5] explains the rapid reversibility of pulmonary edema in a majority of patients with sepsis induced pulmonary edema. This rapid reversibility is

against structural damage to the alveolar endothelial barrier. Elevated sweat sodium and chloride levels, which reflect impairment of systemic epithelial ion transport, were found to be associated with sepsis related pulmonary edema and its severity, as expressed by ventilation index and duration of mechanical ventilation,[4] and should be evaluated as a prognostic marker and risk factor for ARDS.

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The author replies:

I thank Michael Eisenhut for his interest and comments about a recent symposium paper published in the Journal.[1] While I could not argue against a reduced pulmonary fluid clearance in the early phases of ARDS, disturbances in the endothelial and epithelial sides of the alveolar barrier, manifested by deterioration of the pulmonary function, usually represent the sum total effect of numerous cellular and molecular processes. The importance of endothelial injury to the formation of pulmonary edema in this disorder has been well established.[2] Simultaneous and/or sequential activation of signaling pathways, caused by different insults and stimuli in the pulmonary endothelium, interstitium, and epithelium, result in increased permeability to plasma solutes and water with flooding of the interstitial and alveolar space.

Since in some patients, pulmonary edema can resolve quickly, it is likely that those patients do not have the same characteristic pathological abnormalities as those who have diffuse alveolar damage, as could be the case in many patients from the series of patients reported by Aman et al.[3] As pointed out by Schuster, almost 20 years ago,[4] those patients should not be classified as ARDS but as some alternative form of non-cardiogenic pulmonary edema.

To document the permeability disorder in early ARDS, we measured simultaneously the total plasma protein concentration and the protein concentration in the bronchial aspirate of 22 ARDS patients within the first 12 hours of ARDS onset.[5] In all patients, the bronchial aspirate/plasma protein ratio was ≥ 0.6 (mean 0.75 ± 0.10, range 0.60-0.98) and there were no significant differences between the mean values in survivors (0.78 ± 0.13, $n = 8$) versus non-survivors (0.73 ± 0.09, $n = 14$). In all cases, pulmonary capillary wedge pressure was lower than 14 mm Hg during the study period (mean 9 ± 2 mm Hg). When the relationship between pulmonary capillary wedge pressure and the bronchial aspirate/plasma protein ratio was evaluated, a statistically significant inverse correlation between both parameters were found ($P = .025$).

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CORRECTION

In the paper "Possible prognostic value of leukotriene B_4 in acute respiratory distress syndrome" by Masclans JR, Sabater J, Sacanell J, Chacon P, Sabin P, Rosa O, Planas M. (Respir Care 2007 52[12]:1695–1700), the *complete* affiliation information for the authors should have stated: Intensive Care Unit, Area General, Hospital Universitari Vall d'Hebron de Barcelona, *Department of Medicine, Universitat Autònoma de Barcelona (UAB)*, Barcelona, Spain.