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Does my patient really have acute respiratory distress syndrome (ARDS)?

Laurent Brochard

Geneva University Hospital, Geneva University, Department of Intensive Care, 1205 Geneva, Switzerland

Correspondence:
Laurent Brochard, Geneva University Hospital, Geneva University, Department of Intensive Care, 4, Gabrielle-Perret-Gentil Street, 1205 Geneva, Switzerland.
Laurent.brochard@hcuge.ch

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“Does my patient really have ARDS?” Didn’t you hear this phrase quite often? It merely reflects the feeling of many clinicians that the current definition(s) tend to put under the same umbrella (acute lung injury or ALI, and acute respiratory distress syndrome or ARDS) a vast group of very different patients, having different presentations and prognosis [1,2]. The acute respiratory distress syndrome was described in 1967 as a new combination of abrupt symptoms, radiological signs and clinical manifestations characterized by severe defects in oxygenation requiring the use of mechanical ventilation, and the clinical appearance of acute pulmonary edema but with no underlying elevated left atrial pressures that could explain this presentation [3]. It was described to occur in the course of severe conditions, which over the years remained dominated by sepsis of pulmonary or non-pulmonary origin. The other circumstances associated with this syndrome, such as aspiration, trauma, multiple transfusions, extracorporeal circulation and pancreatitis have varied in frequency across the years and the series [4–7]. Last but not least, the syndrome was characterized by a high mortality and was synonymous of the need for intensive care. Experimental research and autopsy recognized that such a syndrome could be associated to a non-specific form of alveolar injury characterized by alterations in the distribution and the type of epithelial cells, concomitant endothelial abnormalities, presence of hyaline membranes, and progressive presence of endoalveolar fibrosis [8,9]. This picture could happen without infection and was included in a vast, non-specific category referred to as acute lung injury. The term diffuse alveolar damage was coined to describe the pathological findings. Can we then say that ARDS is synonymous with (or the clinical manifestation of) diffuse alveolar damage? If yes, should pathological findings be considered the most accurate and specific test for defining ARDS? Obviously, this would result in major limitations for reliably making the diagnosis in patients. In addition, one also needs to think to the meaning and the consequences of making the diagnosis of ARDS, taking the standpoint of the basic scientist, the clinical scientist and the clinician in charge of the patient. The basic scientist would like to understand the molecular pathophysiological pathways which lead to generate this huge pulmonary inflammatory response, the associated risk factors that may aggravate or trigger this cascade of inflammation and the possible role of an underlying individual susceptibility to develop this syndrome from a genetic standpoint. Understanding these mechanisms could lead to preventive measures and to develop targeted therapies at a molecular level. The clinician researcher would obviously like to transfer this information to patients and test biomarkers, genetic markers and appropriate targeted therapies in patients presenting this
syndrome. The clinician researcher also wants to determine the epidemiology and the short- and long-term course and outcome of this syndrome. Importantly, he/she would like to test strategies that may be directly useful to clinicians for the everyday clinical management of these patients.

But for a clinician in 2011, the main consequences of making the diagnosis of ARDS concern the supportive therapy he or she will apply to these patients. Apart from the debated issue of steroids and their possible benefits, clinicians are essentially managing the clinicophysiological consequences of ARDS, and do not act on the underlying mechanisms. The consequences of ARDS result from three major physiological derangements: a major defect in oxygenation; a poor efficiency of the lungs at eliminating CO₂; reduced lung volumes and compliance. For each component, the key issue for the daily management is to evaluate the price to pay to improve the physiological consequences of these problems [10]. It took many years to realize that the price to pay for normalizing arterial blood gas values was most of the time unacceptably high and the problem of ventilator-induced lung injury is now central in our clinical approach [11]. In this regard, it is interesting to ask whether it is acceptable to consider a patient with bilateral pneumonia as having ARDS (a syndrome, not a disease), even if it is unsure that pathology would primarily find diffuse alveolar damage instead of diffuse infectious pneumonia. Even if we forget the feasibility aspect of the histological diagnosis, the answer to this question is not straightforward. First, there is clearly a large overlap between the two classifications, i.e., pure infectious pneumonia versus diffuse alveolar damage. Second, provided that the clinician has some tools to correctly assess the clinical consequences of the three major abnormalities, this differentiation may not matter too much from the standpoint of the supportive therapy. The clinical issue is to correctly evaluate each of the three components: gas exchange (it may require to know the influence of hemodynamics, of intracardiac shunt, etc.), high dead space (it may require to know which level of minute ventilation would be needed for a normal PaCO₂), and low compliance (it may require to estimate or measure the functional residual capacity or the end expiratory lung volume, to have an assessment of recruitability, etc.) in order to individualize the settings of mechanical ventilation and choose the best compromise between the different objectives versus the risks of the therapies. If the clinical discussion was about reaching a molecular target within the lung inflammatory cascade, the reasoning around the definition would be clearly different.

This raises the whole issue of the clinical definition of ARDS [12–14]. This definition should follow a pragmatic approach. On the one hand, having a common definition is absolutely necessary to make reliable clinical research. In this regard, the American–European consensus has been extremely helpful [12] despite its moderate specificity. On the other hand, a 2011 definition should also help the clinician at better defining the type of optimal support for his/her patient. The new Task Force launched by ESICM and ATS should eventually come soon with a new definition following these principles.

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References


