To the Editor:

We read with great interest the work by Bellani et al (1) published in a recent issue of Critical Care Medicine regarding the increase in metabolic activity in the lungs of patients with acute lung injury (ALI)/acute respiratory distress syndrome (ARDS).

In accordance with other investigators (2, 3), the authors have reported an increase in (18)-F-fluorodeoxyglucose (FDG) uptake in the lung parenchyma of a small and heterogeneous group of ALI/ARDS patients using positron emission tomography/computed tomography scan in different phases of the disease. Additionally, the authors found heterogeneity in FDG uptake when regional metabolic activity was correlated with lung aeration compartments (hyperinflated, normally aerated, poorly aerated, and non-aerated regions) based on computed tomography density.

However, we believe that the heterogeneity in metabolic activity in lung compartments reported by Bellani et al relates, at least in part, to the nonhomogeneous pattern of distribution of perfusion in ALI/ARDS patients.

Some studies have reported a redistribution of pulmonary blood flow from poorly and non-aerated regions toward better-aerated areas in an experimental model of ALI/ARDS, most probably induced by the pulmonary hypoxic vasoconstriction (4). Additionally, the hyperinflated areas can also be less perfused depending on the balance between the regional alveolar and capillary pressure. Accordingly, if the alveolar pressure exceeds the capillary pressure, the pulmonary blood flow from hyperinflated regions might be redistributed to normally aerated, poorly aerated, and non-aerated regions (5). However, if the pulmonary hypoxic vasoconstriction mechanism is active, the blood will be redistributed again from non-aerated areas toward normal ones. As a result, the normally aerated areas will be more perfused than the others, but if the pulmonary hypoxic vasoconstriction is inactive (for instance, during sepsis), the pulmonary blood flow will increase across the entire spectrum of computed tomography attenuations from hyperinflated to non-aerated regions.

As can be observed in Figure 4 in the work by Bellani et al, there are some lung areas corresponding to hyperinflated regions, where FDG uptake is low. This observation seems to follow an anatomical segmentation (superior lingular segment in patient 7, and lateral segment in the medium lobe of patient 4). Additionally, it is possible that the observed heterogeneity in FDG uptake is also influenced by other factors, such as the severity of the disease and the presence of comorbidities. Further studies are needed to fully understand the role of perfusion in the heterogeneity of metabolic activity in ALI/ARDS patients.
It is noteworthy that hyperinflated lung areas, presented in Figure 5, correspond to the areas of lower metabolic activity in all but one patient. In seven of these patients (empty symbols), the FDG uptake increased as computed tomography attenuation increased from hyperinflated to non-aerated regions, possibly representing a pattern in which there was no pulmonary hypoxic vasoconstriction compensation. In the other three patients (filled symbols), the metabolic rate was higher in regions of normal lung attenuation where, most likely, more pulmonary blood flow was presented by the redistribution of blood from hyperinflated, poorly aerated, and non-aerated regions toward normally aerated ones.

Based on these aspects we believed that redistribution of pulmonary blood flow is an important aspect to be considered in the analysis of metabolic rate in ALI/ARDS positron emission tomography/computed tomography studies.

The authors have not disclosed any potential conflicts of interest.

Rosana S. Rodrigues, MD, PhD

Department of Radiology, Federal University of Rio de Janeiro, Rio de Janeiro, Brazil;

Alysson R. Carvalho, PhD

Laboratory of Respiration Physiology, Federal University of Rio de Janeiro, Rio de Janeiro, Brazil;

Kathryn A. Morton, MD

Department of Radiology, University of Utah, Salt Lake City, UT;

Fernando A. Bozza, MD, PhD

ICU, Clinical Research Instituto Evandro Chagas, Fiocruz, Brazil

REFERENCES


