Defining Acute Respiratory Distress Syndrome (Again): A Plea for Honesty

To the Editor:

I compliment Dr. Ranieri and colleagues on their candor about quandaries within definitions of acute respiratory distress syndrome (ARDS) (1), but I wish they had gone further in explicating the breadth and depth of the quagmire.

Each of the three pillars of the Berlin definition is flimsy. Radiographic criteria of infiltrates have an abysmal interrater agreement, with a kappa score of 0.296 (2). Pa_{O_2}/Fi_{O_2} ratio is confounded by the curvilinear relationship between Pa_{O_2} and Fi_{O_2}, which varies with the degree of ventilation–perfusion inequality and shunt (3, 4), making it totally unsuited as a foundational criterion. The 7-day interval between the inciting insult and onset of symptoms is whimsical.

The original definition by Ashbaugh and colleagues is sufficient to make a diagnosis of ARDS at the bedside (5). For bedside care, none of the subsequent redefinitions represent an advance for clinicians.

The redefinitions of ARDS are all unsatisfactory because the panels have operated at a superficial level. Panels did not address epistemological concerns (epistemology being the very basis on which knowledge rests) and, more constitutively, did not wrestle with ontological elements (the branch of metaphysics concerned with what really exists in the world). Instead, panels focused on shallow epiphenomena.

As I have previously pointed out (6), it is instructive to contrast ARDS with measles. Measles is caused by a nonredundant etiologic agent (virus), with uniform pathogenesis and a rash so characteristic that diagnosis is self-evident. Nosologically, measles constitutes a "natural kind" on etiologic, pathogenetic, and clinical levels. ARDS does not represent a natural kind on any level.

A natural kind corresponds to something real, a cluster of similarities held together by a law of nature rather than being lumped together arbitrarily by people. The taxonomic antithesis of a natural kind is a sociological construct. Redefinitions of ARDS are akin to the boundaries of the Chicago metropolitan area, which has changed enormously over the past 150 years; it is not a geographical entity with real natural borders but a mere sociological construct.

Obsession with definitions of ARDS is a conversation among trialists speaking with one another about how best to homogenize the recruitment of patients into clinical trials. As new definitions are unveiled, the begetters invariably profess that the new formulation will foster better treatment of patients. Not true. The only management step shown to decrease mortality from ARDS is avoiding a tidal volume of 12 ml/kg. Given that a tidal volume of 12 ml/kg is not employed in any patient, making a diagnosis of ARDS has no impact on bedside decisions.

Coronavirus disease (COVID-19) shone a Klieg light on ARDS, and only the most nondiscerning of observers can feel confident that a fetish fixation on the Berlin definition did not contribute to patient mortality at the height of the pandemic (6).

Ranieri and colleagues intimate that a renovated definition of ARDS is envisaged. This time, promulgators need to be honest with clinicians and inform them that the operation is merely a housekeeping chore for trialists and is irrelevant to (indeed, a distraction from) the care of patients. Bedside doctors are better advised to focus attention on delineating unique physiological problems specific to each individual patient, attempting to elaborate bespoke solutions (7).

The advantage of Ashbaugh's original characterization of ARDS is its submission to Ludwig Wittgenstein's admonition to stay silent on matters whereof one cannot speak (sagaciously). I look forward to the authors' responses to the above perplexities before another definition is unfurled. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

Martin J. Tobin, M.D.*
Hines Veterans Affairs Hospital
Hines, Illinois

and

Loyola University of Chicago Stritch School of Medicine
Hines, Illinois

*Corresponding author (e-mail: mtobin2@lumc.edu).

References

1. Ranieri VM, Rubenfeld G, Slutsky AS. Rethinking ARDS after COVID-19. If a "better" definition is the answer, what is the question? *Am J Respir Crit Care Med* 2023;207:255–260.
2. Goddard SL, Rubenfeld GD, Manoharan V, Dev SP, Laffey J, Bellani G, *et al.* The randomized educational acute respiratory distress syndrome diagnosis study: a trial to improve the radiographic diagnosis of acute respiratory distress syndrome. *Crit Care Med* 2018;46:743–748.
3. West JB. State of the art: ventilation-perfusion relationships. *Am Rev Respir Dis* 1977;116:919–943.
4. Tobin MJ, Jubran A, Laghi F. Pa_{O_2}/Fi_{O_2} ratio: the mismeasure of oxygenation in COVID-19. *Eur Respir J* 2021;57:2100274.
5. Ashbaugh DG, Bigelow DB, Petty TL, Levine BE. Acute respiratory distress in adults. *Lancet* 1967;2:319–323.
6. Tobin MJ. Does making a diagnosis of ARDS in patients with coronavirus disease 2019 matter? *Chest* 2020;158:2275–2277.
7. Tobin MJ. Basing respiratory management of COVID-19 on physiological principles. *Am J Respir Crit Care Med* 2020;201:1319–1320.

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