



## VASCULAR COMPONENT OF PATHOPHYSIOLOGICAL CHANGES IN ACUTE RESPIRATORY DISTRESS SYNDROME

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ARDS is a clinical syndrome of lung injury with hypoxic respiratory failure. Definition ARDS: Ashbaugh (1967), AECC (1994), Berlin (2012), Kigali (2016), Global (2023). There are: 1. Timing:  $\leq 1$  week of risk factor or new/worsening respiratory symptoms. 2. Imaging: Bilateral opacities on X-ray/CT or ultrasound (B-lines, consolidation). 3. Origin of edema: Same as Berlin; pulmonary edema not primarily cardiac. 4. Oxygenation:  $\text{PaO}_2/\text{FiO}_2 \leq 300$  or  $\text{SpO}_2/\text{FiO}_2 \leq 315$  ( $\text{SpO}_2 \leq 97\%$ ), includes non-intubated (HFNO, NIV). 5. Ventilatory support: May include HFNO  $\geq 30$  L/min, NIV, or mechanical ventilation. 6. Setting adaptation: Universal – applicable in all settings. 7. Severity classification: Maintained (mild/moderate/severe) in intubated cases.

ARDS accounts for approximately 1-9 % of admissions to the ICU. Shock, sepsis, and drowning are the most common causes of ARDS. The average mortality rate is 52% (range 28.5%-90%). In patients with ARDS, death is primarily due to sepsis or multiple organ dysfunction. Generally ARDS are the end result of an aggressive inflammatory present scientific thinking suggests that the balance between pro-inflammatory and anti-inflammatory mediators.

ARDS as a syndrome has its own pathophysiological stages, which determine the clinical picture. The clinical picture also has its own staging based on the pathophysiological changes. Over the past two decades, pathophysiological research has focused primarily on pathophysiological changes in the alveoli, neglecting the fact that the acinus is the structural unit of lung tissue. This has become a stereotype, and as a result, less and less attention has been paid to the vascular portion of the acinus.

COVID-19 has brought attention to vascular changes in ARDS, which sometimes become generalized. Vascular changes in ARDS require further study and the search for effective treatment and prevention methods for intensive care programs for patients with ARDS.

This fact requires more effective (and perhaps early and timely) use of existing methods for preventing vascular damage to lung tissue. Critical care specialists must take this into account when developing a comprehensive intensive care program to maximize tissue blood flow, using both pharmacological and instrumental means. It seems relevant to expand the indications for the use of ECMO in critical medicine practice for ARDS.

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