Review of the classics

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ACUTE RESPIRATORY DISTRESS IN ADULTS

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Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnœa, hypoxæmia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxæmia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

This article is the original description of the Acute Respiratory Distress Syndrome (ARDS). Thirty eight years later, and over 10,000 articles published on the subject, a great deal of progress has been made in our understanding of this disorder, yet despite this, many questions remain unanswered. It is of interest to note that in the original description many of the observations made were quite accurate.

The article outlined the clinical, radiological, biochemical and pathological features of a pattern of acute respiratory distress in 12 patients. *"The clinical pattern … includes severe dyspnoea, tachypnoea, cyanosis that is refractory to oxygen therapy, loss of lung compliance, and a diffuse alveolar infiltrate seen on chest X-ray"*. It was noted that none of the patients had chronic lung disease and the precipitant of the disorder was varied with severe trauma preceding onset in seven patients, viral infection in four patients and pancreatitis in one patient.

Although all patients were hypoxaemic, two patients were on room air alone and the remainder on supplemental oxygen with seven on 'respirators'. The $PaCO_2$ ranged from 22 to 63mmHg with pH values mostly normal. Compliance values ranged from 9 to 190 mL per cm water, with a consequent wide variation in measured tidal volumes. Chest X-ray appearances consisted of *"patchy, bilateral alveolar infiltrates"*, the severity of which paralleled the clinical condition. *"At necropsy in seven patients, gross inspection showed heavy and deep reddish-purple lungs… the appearance resembled liver tissue"*. Microscopic appearances were consistent with current descriptions, including the hyaline membrane which prompted comparison with neonatal respiratory distress syndrome.

Therapeutic interventions for this new condition were classified by the authors into those of 'doubtful value' and those of 'apparent value'. The former include digitalis, antibiotics, tolazoline, corticosteroids and intermittent positive pressure ventilation. Changing from pressure to volume control modes resulted in *"improvement in ventilation but little change in oxygenation"*. Two patients improved with steroid therapy. The therapeutic intervention of 'apparent value' was positive end-expiratory pressure (PEEP). Five patients received PEEP levels of 5 - 10 cmH_20. All five demonstrated an improvement in oxygenation. Three recovered rapidly and survived. One died from bleeding complications and one died from *"overwhelming sepsis"*. Only two of seven patients, who did not receive PEEP, survived.

In discussion the authors made several astute observations. *"In view of the similar response of the lung to a variety of stimuli, a common mechanism of injury is postulated. The loss of lung compliance, refractory cyanosis, and microscopic atelectasis point to alveolar instability as a likely source of trouble"*. They postulated that surfactant dysfunction, the knowledge of which was very rudimentary at the time, was a significant contributor to alveolar collapse and acknowledged that PEEP may be beneficial in preventing atelectasis. However, the authors commented that *"The use of positive end-expiratory pressure merely buys time: unless the underlying process can be successfully treated or reversed the prognosis is grave"*.

They also noted that although corticosteroids were apparently useful in two patients, they are of questionable value in the treatment of patients who develop ARDS after trauma.

In short, the authors elegantly elucidated the key pathophysiologic features of ARDS and recommended the use of PEEP in all patients and possibly steroids in a select few. It took many more years to discover the potential harmful effects of ventilation at higher volumes, but otherwise it seems not much has changed.

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