

RES MEDICA

Journal of the Royal Medical Society



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Abstract

In recent years clinicians concerned with intensive care have become increasingly aware that pulmonary insufficiency may develop shortly after the apparently successful immediate resuscitation of the critically ill. The incidence of this complication is high, a figure between 30% and 50% being generally accepted (1). In its initial stages respiratory tract infection does not appear to play a part though bronchopneumonia may later supervene.

The impact of this adult respiratory distress syndrome (ARDS) on surgeons dealing with extensive trauma, sepsis, burns and haemorrhagic shock has been dramatic and since 1968, when the first conference devoted to the pulmonary effects of nonthoracic trauma was held (2), investigation into the problem has been energetic. Essentially two opposing viewpoints have evolved to account for the occurrence of ARDS. That they are interrelated should become clear in the following discussion.

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ISSN: 2051-7580 (Online) ISSN: 0482-3206 (Print)

Res Medica is published by the Royal Medical Society, 5/5 Bristo Square, Edinburgh, EH8 9AL

Res Medica, Spring 1972, 6(6): 15-17

doi:[10.2218/resmedica.v6i6.884](https://doi.org/10.2218/resmedica.v6i6.884)

PULMONARY INSUFFICIENCY IN THE CRITICALLY ILL

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In recent years clinicians concerned with intensive care have become increasingly aware that pulmonary insufficiency may develop shortly after the apparently successful immediate resuscitation of the critically ill. The incidence of this complication is high, a figure between 30% and 50% being generally accepted⁽¹⁾. In its initial stages respiratory tract infection does not appear to play a part though bronchopneumonia may later supervene.

The impact of this adult respiratory distress syndrome (ARDS) on surgeons dealing with extensive trauma, sepsis, burns and hemorrhagic shock has been dramatic and since 1968, when the first conference devoted to the pulmonary effects of nonthoracic trauma was held⁽²⁾, investigation into the problem has been energetic. Essentially two opposing viewpoints have evolved to account for the occurrence of ARDS. That they are inter-related should become clear in the following discussion.

AN IATROGENIC CAUSE?

Serious trauma, sepsis and burns share at least one thing in common with hemorrhagic shock: they all lead to a decrease in the circulating blood volume with reduction of tissue perfusion. Effective means of fluid replacement are now universally available and proper concern is given to replenishing losses of the formed elements of blood, plasma and electrolytes in the proportions required by the individual case. However the practice of fluid replacement to sustain an adequate circulation is not without its dangers. The exuberant use of crystalloid solutions, while preserving renal function, may so load the pulmonary circuit that there ensues an interstitial edema consequent on a lowered intravascular osmotic pressure. Therefore the clinician must be aware of the delicate balance that exists between a renal demand for adequate perfusion and a pulmonary sensitivity to fluid overload.

The pulmonary microcirculation may also

suffer from infusions of blood banked too long or inadequately matched. In this context it is often not fully realised that the lung is the first vascular bed to meet and react with materials delivered by vein. Thus the sludge from old blood, or the potency of pharmacological agents, exert their maximum effect on the pulmonary vasculature. While the lung appears to perform remarkable feats of detoxification and inactivation this function can be outmatched, especially in the sick patient, and thereafter endothelial and alveolar cell damage will occur. High pressures of oxygen administration will also seriously impair pulmonary function but awareness of this danger has led to a cessation of such therapy.

Although ARDS cannot simply be explained by errors in clinical management, especially since it frequently develops prior to hospital treatment⁽³⁾, it is salutary to remember that the clinician can worsen the respiratory function of a patient. Moreover any gross mishandling of drugs or fluids will have far more serious consequences in the critically ill patient than in his normal counterpart.

ENDOGENOUS FACTORS?

The other explanation of ARDS complicating nonthoracic injury stresses that anoxic, injured and inflamed tissue releases a variety of deleterious agents into the circulation. These products of proteolysis or membrane dissolution may be directly injurious to the lung or may trigger the release of other pathological factors. Systemically, activation of Hageman Factor will result in kinin formation and intravascular clotting. Locally, histamine and serotonin may be released in the lung. Normally the spread of these agents is contained by natural inhibitors. But in prolonged sepsis and severe trauma, especially allied to inadequate nutrition and hypoxia, the protective mechanisms of the body are frequently overwhelmed.

There is ample experimental data to suggest that endogenous agents, probably circulating in

pathological amounts, can seriously alter pulmonary morphology and function⁽⁴⁾. A number of factors have been suggested, with various degrees of clinical correlation, and include endotoxin⁽⁵⁾, fibrinopeptides⁽⁶⁾, microemboli⁽⁷⁾, bradykinin⁽⁸⁾ and fat embolism⁽⁹⁾. In any individual case of ARDS a number of these agents may be implicated, working directly or indirectly, and perhaps in a characteristic sequence yet to be defined.

CHARACTERISTICS OF THE LESION

Clinically the earliest manifestation of ARDS is physiological shunting in the lung. The results from both a ventilation — perfusion imbalance and a change in ventilatory pattern in which hyperventilation and a reduction of tidal volume are apparent. A shunt of over 20% is one of the most sensitive indicators of impending respiratory failure and few survive shunts of 60%⁽³⁾. Evidence of significant shunting on admission to hospital is associated with an extremely high mortality, therapeutic reversal of the damage incurred by the lungs proving well nigh impossible with currently available techniques.

The patient responds to the shunt with hyperventilation but cannot compensate for its effect so that the pO₂ falls progressively, reaching values below 60 mm of mercury. While a respiratory alkalosis is usually evident initially, being superimposed on the attendant metabolic lactacidemia of the severely ill patient, as ventilatory failure and increasing shunt develop the pCO₂ begins to rise. In most instances this is a terminal feature. Radiologically the chest is often normal at the outset, in spite of a developing hypoxemia. Within one or two days, however, a diffuse mottling in both lung fields appears, to be followed by the X-ray features of consolidation and atelectasis. These changes occur either with or without the clinical signs and symptoms of the pneumonic process. Lung compliance also decreases and adds greatly to the work of ventilation. This is dramatically demonstrated by the increased pressures required to ventilate the lungs of the patient artificially. Occasionally pressures in excess of 20 cm of water are necessary, often making pressure cycled respirators ineffective.

Studies with radioactive indicators⁽¹⁰⁾ have shown that the pulmonary capillaries are relatively impermeable to the sodium ion on its first passage through the normal lung. This characteristic is lost in patients with pulmonary insufficiency following major operation or

injury. Pathological specimens of the pneumonic lung clearly demonstrate the result of this deranged function and the following features are regularly seen with microscopy, although in varying degrees.

1. an oedematous thickening of alveolar walls.
2. focal atelectasis and in severe cases the formation of hyaline membranes.
3. erythrocyte and platelet intravascular engorgement, diapedesis and intraalveolar aggregation.
4. leucocyte proliferation, margination and adherence to the pulmonary capillary endothelium.

Compared to normal leucocytes these cells contain irregular electronlucent areas and appear to have undergone partial degranulation⁽¹¹⁾. As observed in the shock state, the emigration of these leucocytes is decreased. Therefore the membranes and cellular elements of the lungs present many of the features of the classical inflammatory response though certain differences are evident.

Accepting that the etiology of ARDS remains controversial and is almost certainly multifactorial, its effective treatment rests solidly on the accurate and early interpretation of clinical signs. A careful assessment must perforce include a watchfulness for tachypnea, frequent chest physical examination with accompanying X-rays, blood gases and electrocardiograms where indicated. Obviously care of the primary lesion is of paramount importance. In sepsis the eradication of any septic focus, whether by drainage, resection of non-viable bowel or amputation, is essential. Gram negative sepsis is so common that it is well to remember the high resistance of these organisms to the penicillins. Systemic kanamycin, gentamycin and polymyxin B are antibiotics of proven efficacy against most gram negative bacteria. In cases of fulminating peritonitis lavage of the peritoneal cavity with kanamycin dissolved in normal saline is also meeting with some success the critical need of the patient to counter bacterial and endotoxin invasion from the gut reservoir.

Careful assessment of the loss of vital fluids and electrolytes allows a reasonably accurate titration to be carried out in terms of acid-base balance, hydration and haemoglobin concentration, which should be maintained above 12.5 grams/100 mls if possible. The central venous pressure should not be allowed to rise precipitously, and while absolute values can be misleading, rapid fluctuations must be carefully monitored. In cases of a high central

may be of benefit and diuretic therapy may be necessary; however the increased pressure may simply reflect the rise in pulmonary artery venous pressure elevation of the head and chest pressure that accompanies pulmonary damage. Respirator support is essential in those patients showing a pO₂ much below 60 mm of mercury. The decrease in lung compliance in ARDS has been attributed to a loss of pulmonary surfactant. Saline lung washes from patients with post traumatic pulmonary insufficiency reveal a decreased concentration of surfactant⁽¹⁾. The question is whether this decrease reflects diminished production of surfactant as a primary problem of ARDS or whether it results from poorer extraction of the surface active agent from lungs made atelectatic by another mechanism. Whatever the cause of the diminished lung compliance may be, the maintenance of continuous positive pressure throughout the ventilatory cycle by a respirator is of great benefit and ensures that the occurrence of further atelectasis is minimised. With proper use of respirators and fastidious tracheal toilet patients with severe pulmonary insufficiency can be ventilated for several days.

A variety of drugs have been used in the treatment of ARDS. As already mentioned, antibiotics and diuretics are of proven efficacy provided that their use is appropriate to the situation. Steroids, perhaps on account of their membrane stabilising property, have been shown, at least experimentally⁽¹²⁾, to be of some benefit, and in an attempt to reduce intravascular aggregates in the pulmonary microcirculation both heparin and low molecular weight dextran have been advocated. If

vasoactive peptides are definitely shown to be circulating then the use of antoproteolytic agents to block their formation at source holds out much hope, as may the use of carboxypeptidases which destroy the kinins.

As a final concept it should be remembered that the lung is an actively metabolising organ and not merely a passive membrane exchanger of gases. Normally it deals efficiently with infective or toxic agents reaching it from the air or blood. However this capacity may be overcome under conditions of hypotension, severe injury and sepsis with resultant inflammatory changes and progressive impairment of pulmonary function. If ARDS is not recognised early a relentless fall in blood oxygen tension will seriously impair the recovery processes of the critically ill patient and at autopsy will be found a heavy, sodden and hemorrhagic pair of lungs with numerous areas of atelectasis and consolidation.

SUMMARY

A progressive and pernicious pulmonary complication that often attends nonthoracic trauma, sepsis, burns and circulatory disturbances is discussed. The reason for the rise in the incidence of this adult respiratory distress syndrome (ARDS) is complex but reflects paradoxically the apparent success in the immediate resuscitation of the critically ill. Although the etiology of ARDS is probably multifactorial certain lines of treatment suggest themselves and should be vigorously pursued. Above all, early recognition of the syndrome may prevent the remorseless deterioration of the lung that so frequently accounts for the death of the patient.

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