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RESPIRATORY DISTRESS SYNDROME OF SHOCK AND TRAUMA

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Volume XXI in the Series

MAJOR PROBLEMS IN CLINICAL SURGERY

RESPIRATORY DISTRESS SYNDROME OF SHOCK AND TRAUMA: Post-Traumatic Respiratory Failure

by

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Preface

In the ten years that have elapsed since the respiratory distress syndrome following shock and trauma first became generally recognized, much has been learned about this entity and a certain unanimity regarding its treatment has evolved. Although some authorities still question the presence of a specific syndrome, those who deal extensively with major trauma have no doubt of its existence, for it is in fact the old "fat embolism" syndrome. In simple terms, the syndrome can be characterized as diffuse systemic capillary leaking which results in loss of intravascular volume into all the tissues of the body. The syndrome has its most profound effect on the lung—that organ most susceptible to interstitial edema. The syndrome requires the presence of two basic conditions for its development: One is shock and the other is major soft tissue damage. The more prolonged the former and the more extensive the latter, the greater are the chances that respiratory failure will develop.

As a direct result of our increased understanding of the mechanism of injury and of the pathophysiology of the respiratory distress syndrome, some basic techniques for its prevention have become apparent. At the present time, careful monitoring and the early application of preventive measures such as positive pressure ventilation have delayed the onset of the syndrome until secondary complications—most often sepsis—have appeared. In fact, the presence of sepsis in combination with shock seems to be the common denominator precipitating the syndrome when it appears in the later period following injury.

Varying degrees of respiratory difficulty may have a number of different causes, and the respiratory distress syndrome occurs only rarely in a pure form. For this reason, all the most common causes of respiratory difficulty are herein discussed at some length. The respiratory distress syndrome is then defined in terms of its clinical, radiological, laboratory and pathological characteristics.

A thorough understanding of its prevention and treatment requires an equally clear understanding of the syndrome's etiology. This, however, remains the most controversial aspect of the problem. A de-

tailed review of a number of postulated etiologic mechanisms as well as our own conclusions as to its cause are included. Prevention of the syndrome becomes possible as these etiologic factors are identified.

The available modalities for treatment are presented, including respiratory, cardiovascular and nutritional support methods, as well as control of blood coagulation. Finally, the end results of treatment are reviewed and the reversibility of the lung injury is discussed.

Our book is oriented primarily toward the clinical specialist who has occasion to treat trauma and other critical surgical illnesses. It is our hope that the book will prove beneficial to general and thoracic surgeons, orthopedists, urologists, anesthesiologists and maxillofacial specialists, as well as to other skilled personnel involved in the management of the critically injured patient.

F. WILLIAM BLAISDELL, M.D.
FRANK R. LEWIS, M.D.

Dedication

Both authors wish to dedicate this book to their parents: Dr. and Mrs. Frank E. Blaisdell, Jr. and Dr. and Mrs. Frank R. Lewis, Sr. An invaluable heritage that parents can convey to their children is the desire for educational achievement and the moral or financial support to attain it. Both of us received these in abundance during our education and medical training, and any success we may have we owe greatly to them.

Foreword

Pulmonary complications following injury, operation, and almost any form of resuscitative treatment are of extreme importance to the practicing surgeon. Expertise in resuscitation methods has increased the initial salvage rate of patients suffering devastating injuries, and has improved survival. The subsequent problems which occur from the injury or the treatments are frequently manifested in the lung. It is recognized that there are many aspects of this complication, and that it is a constantly changing field. This monograph approaches the problem of respiratory distress from the aspects of prevention, management, and various etiologies. We hope that it will offer a reasonable approach to the entity, and inform the reader of the most up-to-date methods and knowledge.

Paul A. Ebert, M.D.

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Chapter One

RECOGNITION OF THE RESPIRATORY DISTRESS SYNDROME OF SHOCK AND TRAUMA

Historical Aspects

Much was written about the nature of refractory shock during World War I. The sole pulmonary syndrome recognized as a complication of the management of the critically injured patient was massive pulmonary collapse.

World War II introduced the use of blood banking and for the first time permitted salvage of patients with massive blood loss. The major lesion responsible for refractory shock and late mortality in World War II was renal failure. Bywaters' classic article described the syndrome of renal failure following crush injuries in the London bombings.[7] At the end of the war, several articles by Burford and Burbank called attention to the appearance of "wet lung" following thoracic injuries.[6] While these articles mentioned that the "wet lung" syndrome was seen in abdominal injuries as well as head injuries, the specific cases cited in these articles were limited to penetrating injuries of the chest.

Identification of the role of respiratory failure as a cause of mortality following shock and trauma developed slowly in the period between World War II and the Viet Nam conflict.

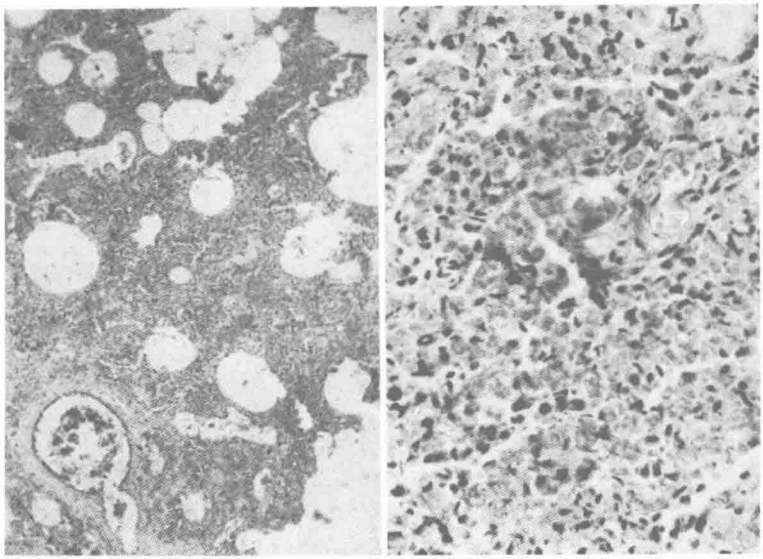

Figure 1–1. First clinical description and pathology of the respiratory distress syndrome of shock and trauma. From Jenkins, M. T., Jones, R. F., Wilson, B., and Moyer, C. A.: Congestive atelectasis in man. Ann. Surg., *132*:344, 1950. *Left:* Magnification × 35; *right:* magnification × 140.

Moon's classic studies of the pathologic changes in shock extended over many years and pointed out the frequent finding of a lung lesion in patients dying after trauma and burns.[14, 15] Mallory's review of the Armed Forces Institute of Pathology material from World War II focused attention on the frequency of pulmonary lesions. His cases represented late deaths, and pulmonary changes were found in every autopsy.[13]

The clinical syndrome that we now recognize as "shock lung" was first described by Jenkins, Jones, Wilson, and Moyer in 1950.[10] They documented eight cases of respiratory insufficiency, three of which were due to trauma. The first trauma case consisted of a 36 year old man admitted October 1, 1948, with a shotgun wound to the right buttock with perforation of the rectum and sigmoid colon and a retroperitoneal hematoma.

He was in profound peripheral circulatory failure. The blood pressure was unobtainable. On October 3 (two days after resuscitation and definitive surgery) he showed resistant cyanosis. Pulse 120, temperature 98°. Venous pressure elevation was manifested by prominent neck veins. Marked expiratory effort was present. There was a paradoxical pulse which equalled 10 mmHg. There were crepitant rales in the right lower lung field. The patient had passed no urine since operation so an indwelling catheter was inserted and about 200 ml of dark brown urine was obtained. Death occurred (on the third postopera-

tive day) suddenly without any unusual event. Respiration ceased first, then the pulse and heartbeat ceased in about two minutes.

Post mortem findings revealed diffusely red pulmonary parenchyma with slate blue color of dependent portions. A very hemorrhagic mass of pulmonary tissue was found in the lower posterior lobular segments of the right lung. This looked like an infarct, but obstruction to the respective pulmonary arterial segments could not be found. The left lung was grossly similar to the right. Only the anterior medial segments of both lungs contained any air. Edema fluid could be expressed from scattered areas on the cut surface. Microscopically all sections of the lungs showed intense capillary congestion, massive intra-alveolar hemorrhage, patchy areas of pulmonary edema superimposed upon the congestion and hemorrhage and incomplete expansion of the pulmonary parenchyma.[10]

The second case in Jenkins' series consisted of a gunshot wound of the pharynx, liver, and colon. The clinical findings and course were similar as was a third case which consisted of traumatic rupture of the duodenum with pancreatitis.

E. E. Muirhead suggested the application of the term "congestive atelectasis" for the appearance of the lungs;[10] this terminology was originally coined by Fegler and Banister to describe changes seen in the lungs of rabbits and other animals subjected to low barometric pressure. It was noted that these pathologic changes were also similar to the description previously given by Moon and Mallory.

Jenkins pointed out that the symptoms in signs of congestive atelectasis differ from those associated with obstructive atelectasis in only a few respects.

The congestive type is attended by a cyanosis that is generally deeper and does not clear as readily when oxygen is breathed. The mediastinal structures do not shift appreciably with congestive atelectasis, but do with obstructive. The presence, location, and extent of obstructive atelectasis are fairly determinable with roentgen rays but the determination of the presence and extent of congestive atelectasis cannot be made early with them. In other words, obstructive atelectasis produces organic pulmonary changes, detectable with roentgen rays and early congestive atelectasis does not. In this series, there is no correlation between the roentgenologic, the physical, and the post mortem examinations. The proved extent of pulmonary involvement is greater than the physical signs indicate, and the physical signs are more indicative of the extent of the process than the roentgen signs are.[10]

Jenkins then described a series of animal experiments designed to elucidate the problem.

"The circulation in the hind legs of dogs was cut off for an hour and a half by the application of tourniquets. Through a chest wall window the development of hyperemia of the lung was observed. After death, lungs of several dogs contained air only in the anterior segments. The middle and posterior segments showed a macroscopic and microscopic appearance suggestive of congestive atelectasis. The conclusions are that the volume of blood in the lungs increased

rapidly following hemorrhage and asphyxia. The rapid injection of saline solution following tourniquet shock after the lung has become stiff causes changes in vascular pressures that are compatible with acute heart failure and the severity of shock is increased thereby."

He concluded that the very rapid breathing which attends the onset of congestive atelectasis is due evidently to the congestion and is reflex in origin.

> The labored breathing was most likely a manifestation of stiffening of the lungs. Resistant cyanosis was manifestly caused by the continuation of a fairly rapid flow of blood through areas rendered airless by the positive increment of blood in the pulmonary capillaries and alveoli.
>
> The involved parts of the lung are carnified but when inflated with air under pressure, they inflate and appear grossly normal. Microscopically, the process is characterized by capillary congestion, intra-alveolar hemorrhage with little edema, incomplete expansion of alveoli and compression closure of bronchioles.[10]

The article concluded that the clinical picture is characterized by the rather sudden onset of dyspnea, labored expiratory breathing, tachypnea, tachycardia, fever, and hypotension that is often aggravated by the transfusion of blood. There is restriction of thoracic motion, dullness over the involved portions of the lung, and resistant cyanosis. The condition was associated with early absence of roentgen ray signs.

Very little information has been added to the description of the clinical and pathologic characteristics of shock lung since Jenkins' article. Review of the literature in the 1950's reflects little awareness on the part of clinicians of the significance of respiratory failure after shock and trauma, and little or nothing appears from the Korean War reports.

In the late 1950's and early 1960's, recognition of this syndrome in the literature appears primarily related to cardiopulmonary bypass. In the early period of open heart surgery, the oxygenators were crude, had extensive foreign surfaces, and the bypass introduced a period of shock (perfusion pressures of 60 to 70 millimeters of mercury being common). In 1960, Baer and Osborn reported that 41 patients died after open heart surgery, and indicated that 70 per cent of these patients had had pulmonary changes.[2] The description of these changes resembles those lesions described by Jenkins.

The documentation of the frequency of pulmonary problems in cardiac surgery resulted in the increased use of arterial blood gas monitoring. Practical and relatively inexpensive means of measuring arterial blood gas tensions became available during the early 1960's. This assessment extended to other critical surgical problems, thus documenting that respiratory problems existed in these patients as well.

Studies such as those of Berry and Sanislow reemphasized that "congestive atelectasis" was present in many critical surgical conditions

including trauma and complicated, prolonged operations which required transfusion.[3] They suggested that positive pressure ventilation with increased increments of oxygen was advisable and reported one survivor so managed.

Gradually the use of positive pressure ventilation became more prevalent. This created the requirement for more sophisticated monitoring and support and encouraged the development of special care units (ICUs).

With the progressive escalation of the Viet Nam conflict between 1960 and 1968, the United States involvement became heavier and more medical units were required. New problems were recognized, and interest in the study of shock was once more revived by the challenge generated by war injuries. Rapid helicopter evacuation to base hospitals, prompt fluid resuscitation, and massive transfusion were the basic principles which were introduced. These resulted in survivals from injuries which were universally fatal previously; consequently, a host of new and challenging syndromes were recognized.

A unit for the study and intensive treatment of refractory shock in human patients was created in late 1965 at Walter Reed General Hospital.[11] This unit's initial report concerned 19 patients admitted with refractory shock. Nine patients had respiratory insufficiency following correction of a hemodynamic crisis. In eight patients, respiratory insufficiency was considered the major cause of death by both the Shock Unit and the autopsy pathologist. The cause of death of all patients who survived a shock period and subsequently died was respiratory failure. Only two patients of the 19 had had any pulmonary disease prior to the shock episode. As Hardaway's group noted:

> This respiratory lesion has been called 'shock lung' or acute pulmonary failure. It is characterized by pulmonary congestion, hemorrhage, atelectasis, edema, and capillary thrombi. This lesion seems to develop as time goes by and may progress even though blood pressure, lactic acid, urine output, and other indices are brought back to normal. More and more pressure may be required to insure a given volume of respiratory exchange. Pulmonary surfactant decreases or disappears. pO_2 progressively falls and the patient dies. The possible role of oxygen therapy in the development of this lesion is unknown.[11]

It was pointed out that the arterial oxygen tension was often extremely low and required immediate treatment as part of resuscitation. This was accomplished by the administration of oxygen nasally or by mask in four cases, but in others tracheal intubation or tracheostomy and the use of a ventilator was required.

In 1967, Ashbaugh and Petty recognized the significance of respiratory failure in civilian trauma.[1] They described 12 cases of acute respiratory distress in adults, seven of which occurred in patients with major trauma, and they coined the term "adult respiratory distress

syndrome." They had introduced blood gas monitoring of seriously ill patients on a routine basis and recognized the frequent occurrence of critical respiratory failure. The clinical course and pathologic changes described were identical to those noted previously by Jenkins. They once again called attention to the significance of respiratory failure after shock and trauma. All 12 of their patients were managed with positive pressure ventilation. The authors called attention to the fact that three out of five patients who were the most critically ill survived, whereas only two of the seven not treated with continuous positive pressure lived. They felt that corticosteroids contributed to salvage of these patients. They ruled out overinfusion of fluids and oxygen toxicity as the cause of lung changes and concluded that the lung changes were consistent with a decrease in surface active agent (surfactant).

In addition to the studies of the Walter Reed Shock Unit, which by 1968 had transferred their clinical research activity to Viet Nam, more and more reports from the Southeast Asia conflict indicated that respiratory failure was the lesion responsible for a large percentage of the deaths after initial successful resuscitation from shock. "Da Nang Lung," "shock lung," and "progressive pulmonary failure" were terms used to describe the pulmonary lesion which, with the adoption of routine blood gas monitoring, was being universally recognized.

In 1968, the National Research Council sponsored a meeting on the pulmonary effects of nonthoracic trauma. The magnitude of the problem in Viet Nam casualties was aired, and the true significance of the lesion was dramatically documented.[8] Simeone, in an excellent review of the problem, presented the historical development of the recognition of the changes of shock lung and raised the question as to the nature of the lesion.[16]

He presented 15 cases of pulmonary complications after trauma which he classified into five types of respiratory failure: fat embolism, flagrant fluid overload, post traumatic atelectasis, pneumonia, and congestive atelectasis.

Our recognition of the significance of respiratory failure in shock developed gradually in the period between 1958 and 1964. Many patients with critical surgical illness appeared to die suddenly in the postoperative period. We ascribed this to the "stress of operation or illness." Respiratory distress was not recognized unless frank cyanosis was manifest. In retrospect, many of our deaths occurred in patients who were tachypneic and no doubt represented deaths whose basic etiology was respiratory failure. In the early period of cardiopulmonary bypass surgery, many patients whom we saw required critical care and were felt to represent examples of "low cardiac output syndrome." These cases, however, almost universally had pulmonary changes at the time of autopsy.[2] Arterial blood gas tension monitoring became prac-

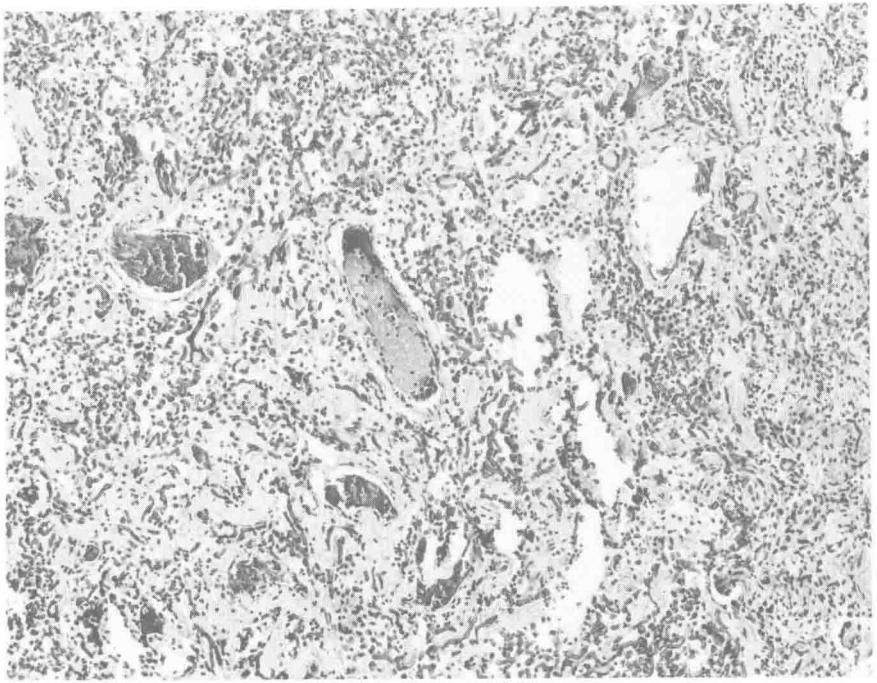

Figure 1–2. Our first description of the pathologic changes of the respiratory distress syndrome. This patient died 24 hours following the surgical treatment of a ruptured abdominal aortic aneurysm. Microscopic sections of the lung (H & E × 100) revealed atelectasis with fibrin-platelet aggregates filling pulmonary arterioles 25 to 100μ in diameter. From Blaisdell, F. W. et al.: Pulmonary microembolism: A cause of morbidity and death after major vascular surgery. Arch. Surg., *93*:776, 1966.

tical and was incorporated into our surgical intensive care unit monitoring in 1963. Originally, our blood gas tension monitoring was limited to cardiac surgery cases. This experience led us subsequently to use it in other critical types of postoperative surgical problems.

In 1966, we called attention to the deaths from cardiopulmonary complications following major vascular surgery, especially those related to ruptured aneurysms.[4] We felt this was due to pulmonary microembolism because in those patients dying immediately after the clinical event, autopsies usually disclosed microaggregates filling pulmonary arterioles 25 to 250 μ in diameter. Clinical findings in these patients were identical to those described by Jenkins and subsequently by Ashbaugh and Petty, and the pathologic changes resembled those of congestive atelectasis.

Subsequently, a similar clinical and pathologic pattern was noted in patients with major trauma at San Francisco General Hospital. Efforts were made to reproduce this syndrome in the laboratory. Robert C. Lim, Jr. succeeded in developing a shock model in the dog utilizing