SECOND EDITION

CRITICAL SURGICAL ILLNESS

Edited by

JAMES D. HARDY, M.D., F.A.C.S.

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W. B. SAUNDERS COMPANY 1980

PHILADELPHIA LONDON TORONTO

W. B. Saunders Company: West Washington Square
Philadelphia, PA 19105

1 St. Anne's Road
Eastbourne, East Sussex BN21, 3UN, England

1 Goldthorne Avenue
Toronto, Ontario M8Z 5T9, Canada

Library of Congress Cataloging in Publication Data

Hardy, James D 1918–

Critical surgical illness.

Includes bibliographies.

1. Surgical emergencies. I. Title. [DNLM: 1. Surgery. WO140 C934]

RD93.H37 1980 617'.026 79-92611

ISBN 0-7216-4511-9

Critical Surgical Illness ISBN 0-7216-4511-9

Last digit is the print number: 9 8 7 6 5 4 3 2 1

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PREFACE TO SECOND EDITION

This Second Edition is a natural sequel to the solid success achieved by the First Edition not only in the United States but abroad as well. As before, the objective has been to provide authoritative information regarding the best current management of a wide variety of serious disorders and problems commonly met on a busy surgical service.

The chapters which are retained from the previous edition have been appropriately revised and brought up to date, and new chapters dealing with head injury, extremity injuries, and the acute abdomen in infants have been added. All thirty-two chapters have been developed on the solid rock of rich clinical experience.

The editor expresses his sincere appreciation to each contributor. These outstanding surgeons extracted from their heavy schedules the time necessary to provide discussions which in every instance comprise truly significant contributions.

The editorial assistance of Mrs. Virginia W. Keith and Mrs. Wanda W. Kenney is also warmly acknowledged. Finally, the contributions of Mr. Robert B. Rowan, Carroll C. Cann and Daniel Ruth have been continuous, and the result is a book which we feel extends the high standard set by the First Edition.

JAMES D. HARDY, M.D.

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CHAPTER 1

POST-TRAUMATIC PULMONARY INSUFFICIENCY: ACUTE RESPIRATORY FAILURE IN ADULT SURGICAL PATIENTS

by **NICHOLAS E. O'CONNOR, M.D., and FRANCIS D. MOORE, M.D.**

Nicholas E. O'Connor was born in Kingston, Ontario, and attended McGill University Medical School. He served his surgical residency at Peter Bent Brigham Hospital and then joined the staff of the Peter Bent Brigham Hospital and the faculty of the Harvard Medical School. He has done outstanding metabolic research related to traumatic injury and has developed a particular interest and expertise in the management of respiratory problems in surgical patients.

Francis D. Moore was born in Illinois and received his college and medical education at Harvard. Almost immediately after serving his residency at the Massachusetts General Hospital he was appointed Moseley Professor of Surgery at Harvard and Surgeon-in-Chief to the Peter Bent Brigham Hospital. Thus he was early recognized as a superior clinical surgeon, teacher, and investigator, and subsequent years have proved this recognition even more prophetic. He has gone on to become possibly the foremost surgical biologist of his time, and several of his books and many articles have become standard reference works throughout the world. His ability to present basic surgical physiology to postgraduate surgical audiences is unexcelled. His leadership in all phases of surgical activity is recognized throughout the world. Dr. Moore was among the first fully to recognize the large role played by various types of respiratory insufficiency in surgical morbidity and mortality, and the following discussion attests his deep learning and understanding of this field.

CLASSIFICATION AND INCIDENCE

Post-traumatic pulmonary insufficiency, or acute respiratory failure, in the adult is a common critical complication in surgical patients. There are relatively few data on the statistical incidence of this syndrome. It was originally estimated from the material in our Intensive Care Unit that about one third of those patients who died exhibited this syndrome (Moore et al., 1969). The other two thirds were approximately equally divided between patients who died of primary visceral failure other than of the lungs (usually of the heart or kidneys) and those who died of acute overwhelming infection, usually a bacteremia with gram-negative bacilli. With certain steps in prevention and treatment (as indicated in the later sections of this chapter), the incidence of this syndrome in our Intensive Care Unit has decreased markedly. Other hospitals in this country that have isolated critically ill patients in an intensive care unit have likewise reported a high incidence of pulmonary failure, with improvement upon adoption of better procedures in respiratory care. This is noteworthy in the material of Weil and Shubin (1967) and MacLean and coworkers (1967); several reports on studies of the Vietnam casualties also emphasize the importance of this syndrome (Conference on Pulmonary Effects of Nonthoracic Trauma, 1968). None of these reports offers any simple statistical analyses. Fatalities are rare in the group most severely ill after extensive injury or operation unless there is some component of pulmonary failure. Before the development of effective methods for dialysis, death from pure renal

TABLE 1–1. Classification of Primary Causes of Acute Respiratory Failure

1. *Pneumonia*
   - Aspiration
   - Viral
   - Bacterial
   - Fungal
2. *Capillary Leak Syndrome (Low Pressure Pulmonary Edema)*
   - Interstitial pneumonia (allergic or drug)
   - Thoracic blunt trauma
   - Fat embolus
   - Amniotic fluid embolus
   - Inhalation of toxic gases, burn injury
   - Septic shock
   - Hypovolemic shock
   - Metabolic—uremia
3. *High Pressure Pulmonary Edema*
   - Congestive heart failure
   - Cardiogenic shock
   - Fluid overload
4. *Thromboemboli*
   - Pulmonary embolus
   - Pulmonary infarct
5. *Chronic Obstructive Pulmonary Disease*
   - Acute exacerbation of COPD

failure was commonplace, with little pathologic pulmonary change other than pulmonary edema. As renal failure has been effectively treated for longer periods of time and as cardiopulmonary resuscitation has become more effective in early phases of injury, post-traumatic pulmonary insufficiency has emerged as a more important cause of death. The frequent combination with renal failure is particularly noteworthy because of the high mortality of renal failure when it occurs as a complication of extensive injury, massive surgery, or burns. In this group of cases, recovery from renal failure is the exception rather than the rule; dialysis serves to prolong life for days or weeks, but the combination with severe tissue injury (or the need for continuing massive transfusions, as in ruptured aortic aneurysm) makes recovery most unusual. In all these cases, pulmonary insufficiency enters as a critically important lethal factor commencing three to five days prior to death.

A National Task Force on Respiratory Failure in 1972 estimated an annual incidence of 80,000 patients with 40,000 deaths from acute respiratory failure. The portion of surgical patients in this estimate was not mentioned. Further, a recent study of the Natural History of Acute Respiratory Failure in the Adult, extrapolating from data collected in nine intensive care units around the United States, estimated there were 150,000 patients with acute respiratory failure per year resulting in 40,000 deaths (Murray, 1977; Bartlett et al., 1980). These estimates serve to highlight the frequency of the problem, especially as seen in tertiary care referral hospitals.

Post-traumatic pulmonary insufficiency is defined as a pulmonary problem in a surgical patient requiring intubation and positive airway pressure for more than 24 hours and an inspired oxygen concentration (FIO_2) of at least 50 per cent to maintain an acceptable arterial oxygen concentration. The presence of extensive venoarterial shunting (and the associated arterial hypoxemia, which is insensitive to increases in FIO_2) is so characteristic as to be a diagnostic indicator, though of course it is not unique to this syndrome. Excluded from this definition are patients who require mechanical ventilation for neuromuscular disorders, for routine postoperative support, or for large airway disease. The diagnostic categories included in the syndrome are listed in Table 1–1. In Table 1–1,

TABLE 1–2. Incidence of the Various Diagnostic Categories as a Per Cent of the Total Number of Patients Studied

	Number	*Per Cent*
Pneumonia	248	36
Capillary leak	272	40
High pressure edema	126	18
Pulmonary emboli	22	3
COPD	18	3
Patients	686	100

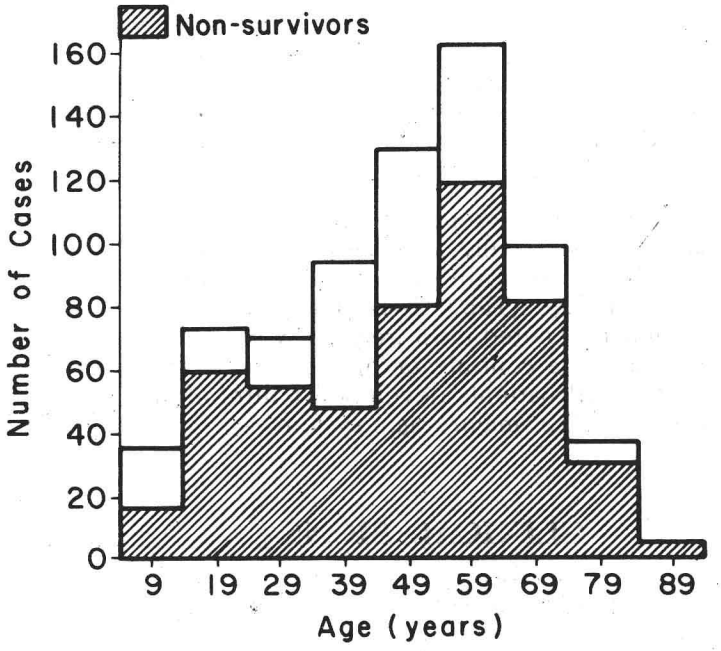

Figure 1–1. Incidence and mortality of post-traumatic pulmonary insufficiency.

capillary leak syndrome refers to pulmonary edema occurring at low left atrial pressures, that is, without fluid overload or left ventricular failure. Pulmonary embolus from deep vein thrombosis is a rare cause and is listed for completeness. An acute exacerbation of chronic obstructive pulmonary disease is likewise rare unless it is precipitated by one of the other causes of respiratory failure. At death most patients with this syndrome have evidence of "super infection," that is, alveolar and bronchiolar leukocytic infiltrates with positive bacterial cultures. This represents a secondary phenomenon, whereas the term pneumonia refers to the primary disease. Thus, the first three major classifications account for over 95 per cent of the cases (Table 1–2).

NATURAL HISTORY

From this same study something of the natural history of post-traumatic pulmonary insufficiency may be learned. As shown in Figure 1–1, the incidence and the mortality rise with each decade up to age 70 years. The survival rate was the same for both sexes (60 per cent of the patients in the study were males) (Table 1–3). The overall mortality for the whole group was 66 per cent, and the mortality in patients over age 65 was 85 per cent.

Respiratory failure will frequently be associated with failure of other organ systems. As a complication, respiratory failure alone had a lower mortality than did respiratory failure associated with failure of other organ sys-

TABLE 1–3. Sex and Survival in Post-Traumatic Pulmonary Insufficiency

	Male		*Female*	
Age	Number	Survival	Number	Survival
<65	286	114	204	75
>65	124	21	72	17
Total	410	135 (33%)	276	92 (33%)

TABLE 1–4. Increase in Mortality with Failure of Other Organ Systems

Organ System Involved	*Patients*	*Incidence (%)*	*Expired*	*Mortality (%)*
Respiratory Failure Alone	210	31	102	49
+1*	193	29	121	63
+2	140	21	128	91
+3	90	13	78	87
+4	35	5	34	97
Total number of patients:	668			

*Denotes number of systems that failed in addition to respiratory system.

tems, namely, the renal, central nervous, hepatic, cardiovascular, or coagulation system. Mortality rose with each additional organ system failure (Table 1–4). The two organ systems that failed most frequently were the renal and the central nervous systems. Two thirds of all patients developed failure of at least one other major organ system.

CLINICAL SIGNS

FOUR CLINICAL STAGES

INJURY PHASE. This is the initial period after injury or operation. Therapy in this phase is directed toward initial resuscitation and in all cases includes intravenous infusion of liberal quantities of blood, salt solution, and colloid. As the patient emerges from this period, the circulation is stabilized (as indicated by good perfusion of the central organs and the extremities), there is a return of urine output to normal or high levels, and there is restoration of the normal mental state. Cardiac output is normal or high. There is mixed respiratory and metabolic alkalosis.

In this apparently resuscitated period (leading to the "free interval") danger signals can arise, indicating the possibility of future severe pulmonary difficulty. These are maintained by spontaneous hyperventilation (as indicated by carbon dioxide tensions below 33 mm. Hg), spotty areas of pathologic change in the lung (on auscultation or x-ray), or the historical fact that the patient's injury involved sudden compression or decompression of the lung itself or inhalation of toxic gases. This results from nearby explosions, from smoke or fire, or from steering wheel injuries to the anterior thorax (even though rib fractures are absent); it also occurs in sudden blows to the abdomen, even though no viscera are ruptured. This type of injury, even without pulmonary changes in the first 36 hours, is regularly followed by a delayed-onset pulmonary lesion. When the chest wall has lost its integrity, either through multiple rib fractures or rupture of the diaphragm, or by penetrating missile wounds, then the likelihood of pulmonary insufficiency is greatly increased.

FREE INTERVAL. This period usually occupies from one to five days following the initial resuscitation. The patient's recovery now appears superficially to be progressing nicely. There is good blood pressure, cardiac output (which is often grossly increased), urine flow, peripheral perfusion, color, and mentation. Indeed, the patient may go on to complete recovery from this phase with no serious pulmonary impairment.

In those for whom further trouble is in store, there is a subtle progression characterized by maintained spontaneous hyperventilation, with an inappropriately low carbon dioxide tension occurring too late in the course for pain, apprehension, or direct pulmonary injury to be regarded as causative factors. There is a beginning difficulty in oxygenation, initially very mild, with oxygen tensions above 80 mm. Hg on room air. Test inhalation of 100 per cent oxygen for 20 to 30 minutes, using some type of mask to assure good airway closure (or using a cuffed tracheostomy or endotracheal tube if such has already been necessary because of the original injury) will demonstrate an alveolar-arterial oxygen difference that is greater than normal (see the following section). Thus, though the patient is not yet cyanotic or severely anoxic, early manifestations of the characteristic respiratory lesion may already appear in the form of hypocarbia with venoarterial admixture (Ayres et al., 1964). As this becomes more severe, some cyanosis is observed, hyperventilation is more marked, and

hypocarbia is more profound. The respiratory situation now becomes much more worrisome, not only because of these physiologic findings but also because of increasingly widespread rales and rhonchi (sometimes with marked evidence of bronchospasm) on auscultation and an increasingly widespread fluffy soft infiltrate visualized by x-ray.

PROGRESSIVE PULMONARY INSUFFICIENCY. As the pulmonary problem begins to predominate in the clinical picture, the patient enters the third phase, progressive post-traumatic pulmonary insufficiency. Before its recognition and during the free interval, many patients have been transferred to other parts of the hospital. In some instances this syndrome is doubtless recognized for the first time when fully developed, during its third phase; in such cases greater vigilance in anticipating the subtle early signs might have brought the problem to light several days earlier.

Difficulty in oxygenation now becomes the predominant clinical feature. Under no circumstances should tracheostomy be carried out for anoxia alone until a suitable trial of endotracheal intubation has indicated that direct access to the lower airway will yield an improvement that justifies its hazard. If the difficulty is purely that of venoarterial admixtures (i.e., passage of venous blood through the lungs without ventilation), then great increases in oxygen tension in the airway yield a disappointing increase in arterial oxygen tension. One must be satisfied with "safe" airway oxygen concentrations in the vicinity of 60 to 75 per cent to achieve arterial oxygen tensions (P_{O_2}) in the general neighborhood of 70 mm. Hg.

The relation of inhaled oxygen concentration to achieved arterial oxygen tension is the basis of the "oxygen tolerance test," which we first used as a rough quantification of venoarterial admixture in patients at this stage. In fact, giving the patient 100 per cent oxygen to breathe is a very sensitive measurement of shunt because when the arterial P_{O_2} is high, a small depression of arterial oxygen content with venous admixture causes a relatively large fall in arterial P_{O_2} owing to the almost flat slope of the oxyhemoglobin dissociation curve in this region. This test, repeated daily or more often as required, enables one to tailor the oxygen therapy to the patient's needs; changes in its response over the course of time are a guide to prognosis.

Tracheostomy can often be avoided. Endotracheal intubation is far preferable and is tolerated for periods of a week or more; withdrawal of the tube restores to the lower airway its normal anatomic relationship, a restoration that is never easily or quickly done after tracheostomy. When endotracheal suction is necessary, it can safely be done through the endotracheal tube.

Survival of patients who have sustained the severe anoxia of the third phase is still possible if the airway is managed with restraint and if certain promoting or maintaining factors of systemic therapy that further hazard the lungs are avoided. In most cases, however, in spite of the most elaborate measures to avoid damage to the lungs and airway, the lesion becomes progressive; there are progressive and diffuse infiltrates and widespread pulmonary infection, which is manifested both by a change in the x-ray appearances and by a febrile course. The aspiration of purulent material from the lower airway discloses very large numbers of a single organism, in many cases an organism that has previously been identified in cultures of wounds, septic surgical incisions, or blood.

TERMINAL PHASE. Oxygenation now becomes ever more refractory to any increase in airway oxygen tension as the shunting defect becomes more severe. Increases in end-expiratory pressure will improve oxygenation for only a short time, a day or two at most, as the lungs become progressively stiffer and require still higher ventilatory pressures. Finally, as ventilation and perfusion abnormalities in the lung become more widespread, with some areas perfused but not ventilated at all and others evidently ventilated but poorly perfused, there is an increase in dead space and a rise in carbon dioxide tension of the blood from its chronically hypocarbic levels. This presages death. It is frequently accompanied by other evidence of deterioration, particularly a return of circulatory failure with hypotension, oliguria, and coma and the resumption of a severe low-flow state with mounting lactic acidosis. During the last day or two of this disease the auscultatory and x-ray findings in the lungs change but little and are of remarkably little assistance to the therapist. Pulmonary infection is severe and blood-stream infection is commonplace. A few warning changes in the electrocardiogram may suggest subendocardial ischemia; slowing to asystole may be abrupt and with