POST-TRAUMATIC PULMONARY INSUFFICIENCY

PATHOPHYSIOLOGY OF RESPIRATORY FAILURE AND PRINCIPLES OF RESPIRATORY CARE AFTER SURGICAL OPERATIONS, TRAUMA, HEMORRHAGE, BURNS, AND SHOCK

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Post-Traumatic Pulmonary Insufficiency

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Introduction

These studies began as an effort to define processes ultimately responsible for the death of young patients who are healthy and socially effective prior to injury: terminal mechanisms in salvageable patients.

We seem to be living in an era of disappearing surgical mortality analogous to the reduction in maternal mortality which took place after 1925. But, unlike the obstetrician, the surgeon must treat a great variety of patients some of whom are in an older age group, suffering from advanced vascular disease, late malignancy, or chronic visceral failure. Among the elderly, failure of therapy is usually due to some combination of age, malignaney, or chronic tissue degeneration. For our purposes here it was more important to concentrate on the salvageable group whose illness is of special social significance: patients in the prime of life. It is in this group, which includes all of the military and most of civilian trauma, that surgical mortality seems to be falling. Many patients still are lost; their salvage depends on a clear definition of the terminal mechanisms that threaten them.

One of the commonest of these terminal pathways is a peculiar, progressive, and highly lethal form of respiratory failure.

When an injured person is resuscitated from a low-flow state, and dies a week later under intensive care with progressive anoxic coma under oxygen treatment, conventional terms used to describe this death are often meaningless. The patient did not die of the trauma alone, nor will terms such as "bronchopneumonia," "shock," "shock lung," or "anesthesia death" adequately describe the events. One must look beyond such clinical platitudes to determine the precise impact of the initial injury, the low-flow state, its response to treatment, the challenges to pulmonary ventilation and perfusion posed both by injury and treatment, the role of oxygen inhalation and mechanical ventilation, and the immunologic injury suggested by declining resistance to ambient flora of low virulence.

Awareness of this pulmonary problem goes back many years to that period after World War II when, with increased use of blood transfusion, and effective methods of treatment of renal failure, patients lived long enough to develop new complications of injuries previously lethal. During the period from 1955 to 1960 there was a marked increase in the use of special respiratory apparatus and mechanical ventilators. Many patients were taken care of in intensive care units originally established for patients undergoing aortic, cardiopulmonary, or cerebral surgery. It soon became evident that, despite the variety of devices and methods available in an intensive care unit, respiratory failure was still a common cause of death.

Post-traumatic pulmonary insufficiency is always a mixed lesion as to pathogenesis, bacteriology, and treatment. Preexisting pulmonary disease is a rarity in most of these patients, although heavy smoking seems to make the patient more vulnerable. The disease presents a characteristic syndrome. It can often be prevented by recognition of the early danger signals, identification of causative factors, and avoidance of systemic or local measures that are a further hazard to the lungs.

This is not a textbook of respiratory physiology or of techniques for intensive care of inhalation therapy. It is designed instead to supplement a number of excellent works on those subjects, many of which describe specialized techniques or approaches, yet give insufficient attention to the rest of the clinical setting.

It is our purpose to bring together the many facets of patients we have studied, and to assemble the clinical syndrome as it is seen in patients after resuscitation from tissue injury. Such an assembly must include all aspects of the patient's care, including the nature of the initial injury and its surgical treatment, disordered pulmonary physiology, pathogenesis, and, above all, prevention. This is a lesion which develops under the eyes of the therapist. It is amenable to prevention in most instances.

These observations were initiated in the summer of 1963 with a series of longitudinal studies of critically ill patients. We would like to acknowledge our indebtedness to the surgeons and physicians of the Peter Bent Brigham Hospital and the Harvard Medical School who participated in the care of these patients. We would like particularly to indicate our gratitude to Dr. Leroy D. Vandam, Dr. David E. Leith, and Dr. Milton H. Alper of the Anesthesia Division, who have collaborated with us in the care and study of these patients; to Dr. Dwight E. Harken, Dr. Jack Matloff, and Dr. Warren Taylor of the Thoracic-Cardiac Division, whose techniques and insights into the problems of these patients have always been helpful; to Dr. Eric Milne of the Radiology department, who has made the most of films taken under adverse circumstances with portable equipment.

We are indebted to Dr. Eleanora Galvanek of the Department of Pathology, who has made the electron micrographs from lungs of pa-

tients studied here; to Dr. Gary L. Huber of the Harvard School of Public Health, who assisted in their interpretation; and to Dr. Carl Teplitz of the Department of Biology at Harvard College, who has supplied us with the electron micrographs of the lung in experimental animals.

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This book was originally planned as a journal article of the review type. The bibliography, numerous charts, and color plates made this impractical. We are indebted to Mr. John Dusseau and the W. B. Saunders Company for making it possible to publish this monograph on the same schedule as would have been possible in a periodical.

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Our thanks go out, as in all our previous publications, to those many patients who, wittingly or unwittingly, have contributed to our knowledge. In this instance many have gained in security and healing through the development of new concepts and techniques of management.

Finally, the authors wish to indicate their debt of gratitude to their wives and families who have been of such assistance and shown such patience during the preparation of this work.

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Clinical Setting and Incidence

CLINICAL SETTING

Patients exhibit several distinct patterns of clinical behavior after their initial resuscitation from severe injury, hemorrhage, or shock. In most instances the restoration of blood volume, blood pressure, and blood flow is readily accomplished, and is followed by prompt recovery without residual damage.

In a second group of patients, blood pressure and flow are only precariously reestablished, and the patient becomes increasingly refractory to complete resuscitation. A much more intensive program of care is necessarily commenced. Most of the fatalities will be found among these patients with a refractory low-flow state, who are difficult to resuscitate and who require large volumes of replacement solution, usually blood or blood products (Smith and Moore, 1962). A significant fraction of patients who later succumb pursue the course of progressive pulmonary insufficiency.

Among adult patients who have been severely injured, approximately one-third of the deaths are due to direct involvement of the heart, brain, or kidney by the initial injury or primary disease. About one-third die from a process that is primarily septic and involves bloodstream infection, usually with gram-negative bacilli or yeasts. Finally, about a third of the fatalities are due primarily to progressive respiratory failure. Some of the patients in the first two categories also exhibit important elements of pulmonary insufficiency. Disease in the lungs therefore becomes of importance in the death of 30 to 50 per cent of adult patients who die under intensive care today, following an initial episode of trauma, hemorrhage, major operation, burn, or shock.

During their hospital course, these patients demonstrate a gradual transition from the primary manifestations of the injury itself and the subsequent low-flow state, to secondary problems of pulmonary insufficiency. After a free interval of several days following the initial episode of injury, arterial oxygen tension falls and becomes progressively unresponsive to increase in the oxygen tension in the inspired gas mixture. Terminally, there is recurrent failure of tissue perfusion, severe anoxia, and a mixed acidosis due to lacticacidemia with superimposed hypercarbia, finally leading to bradycardia and asystole.

Patients who demonstrate this syndrome are the survivors of an insult so severe that in a former decade – or in a later year of their life – they might have succumbed far earlier in their illness. During and after World War II extensive blood transfusion enabled patients to survive long enough to unmask acute tubular necrosis as a complication of severe injury, hemorrhage, and shock; extensive blood transfusion was responsible for these long survivals, yet it also contributed to the renal failure if the blood was poorly matched or preserved. The syndrome under consideration here appears to be a pulmonary analogue: current methods of resuscitation and ventilatory support enable patients to survive long enough to develop a pulmonary lesion, while certain features of the therapy itself (tracheostomy and mechanical ventilation at high oxygen tensions) appear to contribute to its severity.

INCIDENCE

The experience of our unit is not unique in identifying pulmonary insufficiency as a major cause of death in patients under intensive care after severe injury. Respiratory insufficiency is reported by Hardaway et al. (1967) as a major contributing cause of death in a group of patients analyzed and studied in detail at the Walter Reed Army Institute for Research. He reported that, of 19 patients who were admitted in severe shock, respiratory insufficiency developed in 9 following correction of the hemodynamic defect. In 8 patients respiratory insufficiency was a major cause of death. Only 2 patients of the 19 had demonstrable pulmonary disease prior to the episode of injury. Hardaway acknowledges the possible role of high oxygen tensions in the production of this pulmonary lesion; his report does not indicate how many patients had tracheostomies or in what fraction high oxygen tensions were required for any length of time. The same author mentions progressive pulmonary insufficiency as a major cause of death among those lost after initial resuscitation in the Vietnam War.* Shoemaker* and Weil* also report that pulmonary insufficiency accounts for a significant fraction of the late

*These are personal communications from Dr. Hardaway, Dr. Shoemaker, and Dr. Weil based on their experience, either in the military (Hardaway) or in civilian shock units in Chicago (Shoemaker) and Los Angeles (Weil).

deaths observed in their Intensive Care Units, among patients initially resuscitated. In some instances these were deaths among patients whose resuscitation had appeared sufficiently complete to permit their transfer to other portions of the hospital.

A report by Nash et al. (1967) notes a lesion of this general type in patients receiving prolonged mechanical ventilation at increased oxygen tensions. The report by Nash described 70 patients who were examined after death following prolonged artificial ventilation. As a control group, 70 patients were studied who had never received mechanical ventilatory assistance. Lung weights and histologic studies were used as indicators of the severity of the parenchymatous pulmonary process, which the authors associated in large part with oxygen therapy. The report does not

TABLE 1. TOTAL ADMISSIONS AND LETHAL MECHANISMS
Intensive Care Unit—PBBH—Twelve Months

PRESENTING PROBLEMS ON ADMISSION OR TRANSFER TO INTENSIVE CARE

	NUMBER OF CASES	DEATHS NUMBER	DEATHS RATE (PER CENT)
Advanced peritonitis	4	4	100
Postoperative complications	30	13	43
Massive trauma	12	3	25
Primary respiratory failure	22	7	31
Postoperative open cardiac repair	93	28	30
Cerebral disease (stroke, head injury, barbiturate overdosage)	7	1	14
Myocardial revascularization (postoperative)	26	2	8
TOTAL	194	58	30

PREDOMINANT LETHAL MECHANISMS

	NUMBER OF DEATHS	PER CENT OF TOTAL DEATHS
Lungs: progressive pulmonary insufficiency	18	31
Heart: myocardial failure, infarct, or arrhythmia	15	26
Infection	4	7
Hemorrhage	4	7
Kidney: acute tubular necrosis	7	13
Brain: barbiturate overdosage, head injury, and stroke	10	16
TOTAL	58	100

INCIDENCE OF CERTAIN MEASURES

	NUMBER OF CASES	DEATHS NUMBER	DEATHS RATE (PER CENT)
Mechanical ventilation over six hours	119	50	42
Tracheostomy	51	30	60
Tracheostomy unnecessary	143	28	20
Hemodialysis	8	7	88

list in detail the prior injury or disease states of these patients, but there is a remarkable correlation of such indices as lung weight, hyaline membrane, and alveolar cell hyperplasia with the duration of therapy under high oxygen tensions. All authors recognize the fact that some patients treated with high oxygen tensions in the airway survive uneventfully. This is particularly true of patients who require respiratory therapy for causes other than acute trauma (such as poliomyelitis, polyneuritis, barbiturate overdosage, and myasthenia gravis). This contrast makes it particularly important to examine acute trauma and its treatment, to define those components that render the lung vulnerable to damage during inhalation therapy and mechanical ventilation.

In Table 1 is shown the experience of the Intensive Care Unit at this hospital over a representative 12-month period. This provides an example of the incidence of pulmonary problems among a group of critically ill patients in a general hospital. The mortality among these 194 patients was 30 per cent. About one-third of the deaths were associated with progressive pulmonary insufficiency. Primary involvement of the heart, kidneys, and brain accounted for somewhat over one-third of the deaths. In this particular group, hemorrhage and sepsis were less prominent than usual, together accounting for only 14 per cent of the deaths. Forty-two per cent of the patients who later died received mechanical ventilatory assistance for periods longer than six hours, and the mortality among those who required tracheostomy was 60 per cent, whereas it was only 20 per cent in the much larger group whose injury did not require tracheostomy. The impressive mortality of renal failure, when it complicates surgical operations, severe compound injuries, or infection, is evident.