PHYSIOLOGIC PRINCIPLES OF SURGERY

SECOND EDITION

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edited by

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PREFACE

The pace of advancement in the medical sciences has not slowed since the original edition of this book was projected. Indeed, if anything, the pace has accelerated. Therefore if this publication is to continue to serve its intended purpose, periodic revisions will be necessary. We believe the time for the first revision has now arrived.

In preparing this edition, we have attempted to bring the subject up to date without materially increasing the size of the book. Our contributors were requested to maintain the same critical attitude in selecting the matter to be covered as was urged in the initial version, and insofar as possible, to delete as much of the older material as was added in new information.

One additional chapter has been introduced—that on Oncology. The desirability of a section on susceptibility and resistance to tumor growth was considered for the first edition, but at that time it was not believed that such research embodied a sufficient degree of physiological investigation for inclusion in a book on surgical physiology. In the interim, we are convinced, this situation has changed, and we are indebted to Dr. J. T. Grace for his excellent presentation of this complex and rapidly expanding subject.

The amount of alteration required necessarily varied from chapter to chapter, depending on the advances that had been made. Because of the death of certain authors, and in a few instances for other reasons, some of the sections have been revised or rewritten by others. In general, however, the character of the book has been retained.

The acceptance of this work, calling for a new edition, is of course gratifying. In the original preface we stated that whatever merit the book possessed was due entirely to the efforts of its contributors. In this new version, the same recognition is gratefully expressed.

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CONTENTS

CHAPTER 1

METABOLIC CHANGES ASSOCIATED WITH INJURY

Stanley M. Levenson,
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The rapid nutritional deterioration that so often follows severe injuries or acute infections is one of the dramatic metabolic problems encountered clinically. For example, Figure 1-1 shows a young man about 8 weeks after he had been severely injured. His hospital course had been stormy. At the time the pictures were taken he weighed 100 pounds; his pre-injury weight had been 175. When such rapid nutritional deterioration develops in patients with severe injury (operative or traumatic), important complications ensue—weakness, easy fatigability, increased susceptibility to anesthesia, shock and infection, altered gastrointestinal and liver function, and impaired wound healing; as a consequence, convalescence is prolonged and mortality increased.

The postinjury nutritional problem, important in day-to-day civilian life, becomes magnified in time of war. Weight losses of 20 to 30 pounds in 2 to 4 weeks were common among a group of patients with severe battle wounds and renal failure treated during the Korean War in 1952-1953, and even losses up to 45 pounds within the same period occurred. Mortality was about 50 per cent.[1] The following case report is indicative of the complexity of the problem:

A 26-year-old American soldier was severely wounded by mortar shell fragments. His injuries included lacerations of the scalp, cerebral concussion, penetrating wounds of the abdomen, traumatic amputation of the right thigh, and multiple soft tissue injuries. He was in severe shock during the first 3 hours after injury despite the administration of 2500 cc. of blood, 350 cc, of 25 per cent albumin, and 150 cc. of

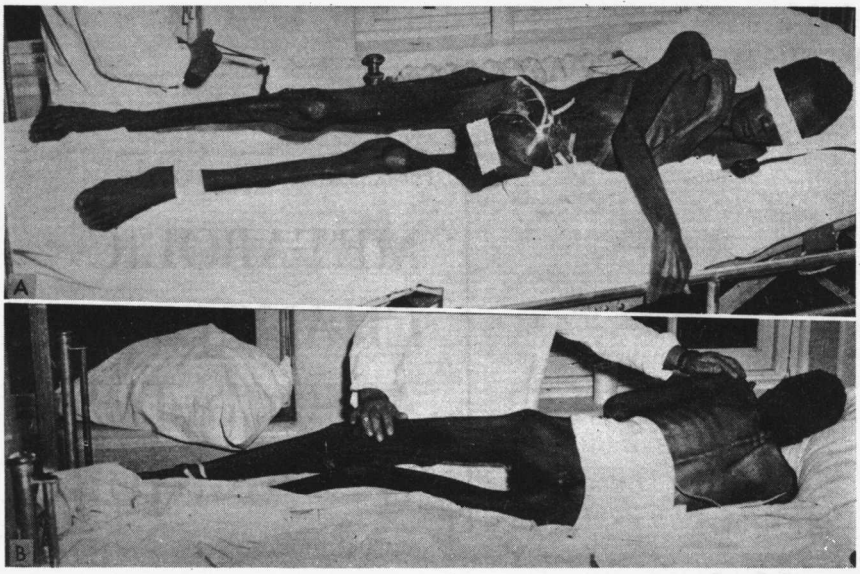

Figure 1–1. Nutritional deterioration in severely injured patient, 8 weeks following injury. (Armed Forces Institute of Pathology, Walter Reed Army Medical Center.)

isotonic saline solution. His right leg was amputated without anesthesia in the emergency room. In the next 4 hours, he was given an additional 2500 cc. of blood. His pressure gradually rose to normal, but his pulse remained rapid. Operation was delayed 3 more hours because the patient had not regained consciousness. During this time, he received another liter of blood. His blood pressure remained stable at about 130/80.

Operation, under atropine, thiopental sodium, and nitrous oxide anesthesia, was begun 10 hours after injury. The patient's blood pressure immediately fell and remained low throughout most of the operation despite the infusion of an additional 3500 cc. of blood. Estimated operative blood loss was 3000 cc. The operation lasted 3 hours and consisted of exploratory laparotomy, drainage of the liver and kidney perforations, re-amputation of the thigh, and debridement of any soft-tissue injuries.

The postoperative course was complicated by profound and persistent renal failure, jaundice, pneumonia, wound infections, and considerable weight loss. Extracorporeal hemodialysis by a Kolff-type artificial kidney was necessary on the fifth, ninth and fifteenth days because of progressive hyperkalemia and uremia. Temporary improvement followed each dialysis, but the course was progressively downhill. The patient died on the nineteenth postinjury day.

At autopsy, the entire body showed evidence of marked wasting. The amputation site of the right thigh showed extensive necrosis and infection of the skin flaps, muscle, and fascia. There was a severe bacterial pericarditis, focal edema and necrosis of the myocardium, bronchiolar pneumonia, severe central necrosis of the liver, acute tubular necrosis, hypertrophy of the parathyroids, and necrosis of the occipital poles of the cerebral cortex.

Nutritional disturbances that result from profound metabolic changes consequent to severe trauma may occur in any seriously injured patient. Lesser trauma is followed by less dramatic changes and by fewer complications, but the basic metabolic changes are qualitatively similar.

There are many similarities in the metabolic response of previously healthy subjects to acute infectious diseases and to injury, but these responses are not identical; it cannot yet be decided with certainty whether the differences are principally quantitative, or whether they are also qualitative. This is due in part to the virtual impossibility in comparing the severity or gravity of the two situations. Is a patient with an extensive third degree burn more severely "stressed" (from the metabolic and nutritional points of view) than a patient with overwhelming pneumonia?

It has been recognized for centuries, that malnutrition may result from serious injury or illness, but it was only

about 75 years ago that systematic investigation of this problem began with a few measurements of nitrogen excretion in patients with typhoid fever. Coleman, Shaffer, and Dubois[2,3] began their classic studies of the metabolic changes in patients with acute and chronic medical illnesses about 50 years ago, and about 35 years ago Cuthbertson[4] clearly described the increased urinary nitrogen excreted following fractures. It was not until the late 30's and early 40's, however, when the threat and then the occurrence of World War II aroused widespread interest in this problem. There have been hundreds of reports since then describing metabolic changes after injury, from which it appears that apparently all metabolites are affected.

Although considerable descriptive biochemical data have been accumulated, there are significant gaps in the available information, and many fundamental questions remain unanswered. We still do not fully understand the mechanisms underlying these metabolic changes, nor do we fully understand their physiologic consequences. How do these metabolic changes affect the clinical course and convalescence of the injured patients? Should attempts be made to modify them? Our intent in this chapter is, first, to present some of the known facts of the metabolic response to injury (with particular emphasis on the so-called "catabolic" period) and, second, to point out the gaps in our present knowledge of the mechanisms and significance of these responses with the hope of stimulating interest and investigation in these problems. Finally, we will make some recommendations for the nutritional care of the injured patient. The aspects of these matters that we have chosen to present in this chapter are to be regarded as a basis for thought and discussion, rather than an attempt to cover completely so vast a topic. This chapter is concerned principally with the responses of previously healthy adult males; it does not attempt any systematic coverage of the problem in infants, children, women, or the aged. And only a passing mention is made of the important influence of emotional factors on these problems. Also, since one of us (S.M.L.) has discussed in detail elsewhere[5] the metabolic changes associated with or due to shock, these matters are alluded to only briefly in this chapter. The matter is discussed more fully in Chapter 5 of this book.

METABOLIC REACTION TO INJURY

Normally, a complicated series of interrelated metabolic reactions proceeds in the young healthy adult in a manner that results in a relatively steady, albeit dynamic, state. Although it has been customary to talk of the metabolism of protein, carbohydrate, and fat separately, it is evident that this practice is one of historical and editorial convenience. Proteins, fats, and carbohydrates are so interconnected that any separation, in a sense, is artificial. Further, their reactions take place in water and are affected by vitamins, hormones, and minerals. Something happens after injury that disturbs these interrelationships and thereby modifies the steady metabolic state characteristic of the healthy adult; there are widespread disturbances in the behavior of all metabolites so far studied. These changes are in general qualitatively similar in patients with a wide variety of injuries or illnesses.[6-24]

The intensity and duration of this period of metabolic disturbances depend on a number of factors, among which are the extent of the injury and the state of the individual at the time of injury. The greater the injury, the greater the metabolic response. Moore[25] and Howard[26] have suggested a classification for grading the severity of uncomplicated traumatic and operative injuries. Shaffer, Coleman, and DuBois, as well as Grossman et al.,[27] have pointed out that the "gravity" of the acute infection determines the intensity and duration of the metabolic response. It is the previously healthy young man who shows the greatest upset; the reaction is less severe in women, children, the elderly, and the