THE ILLNESS OF TRAUMA

GARRETT PIPKIN, M. D.
VICTOR BUHLER, M. D.

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By

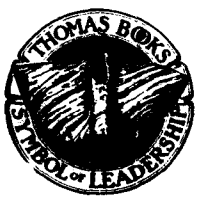
GARRETT PIPKIN, M.D.

Clinical Professor of Orthopedic Surgery
Kansas University Medical Center
Kansas City, Kansas

and

VICTOR BUHLER, M.D.

Associate Clinical Professor of Pathology and Oncology
Kansas University Medical Center
Kansas City, Kansas



CHARLES C THOMAS · PUBLISHER
Springfield · Illinois · U.S.A.

Published and Distributed Throughout the World by
CHARLES C THOMAS • PUBLISHER
BANNERSTONE HOUSE
301–327 East Lawrence Avenue, Springfield, Illinois, U.S.A.
NATCHEZ PLANTATION HOUSE
735 North Atlantic Boulevard, Fort Lauderdale, Florida, U.S.A.

Library of Congress Catalog Card Number: 70–135941

Printed in the United States of America
K-8

PREFACE

THIS MONOGRAPH reflects a heritage of scientific knowledge and clinical experience in the illness of trauma. It was inherited by the authors from certain members of the Jackson County Medical Society, who in their day were members of the medical staff of the Kansas City General Hospital.

In the first half of the twentieth century, this municipal hospital, the second largest hospital in the state of Missouri, was the clearing center for all cases of trauma occurring on the streets of greater Kansas City which were of such severity as to require transportation by ambulance. Since the city of Kansas City, Missouri, maintained the primary ambulance system, traffic officers were instructed to contact the Kansas City General Hospital for needed ambulances. Along this route came all the injured for treatment, regardless of age, race or social or financial responsibility. Those of financial means who had mere serious injuries were admitted and subsequently transferred to private hospitals as their status permitted; the indigent were kept at the Kansas City General Hospital. Thus a great many acutely traumatized patients were available for study.

During the 1930's two major changes were instituted. One change was accomplished under the directorship of Frank Dickson, M.D., when the fracture service was made a part of the Department of Orthopedics. This department instituted an annual instructional program in the care and handling of the injured for ambulance drivers, i. e. splint them where they lay. The second change consisted of the medical management of the acutely traumatized patient. Robert Koritschorner, M.D., Pathologist at Menorah Hospital, Kansas City, Missouri, brought from Germany the concept of fat embolism. Abraham Sophian, M.D., applied this knowledge, and clinically reported the first cases of fat embolism to be recognized antemortem, and treated the syndrome with oxygen. It is of great interest, after these

many years, numerous reports in medical literature, and many aliases, that the illness of trauma has completed the cycle and is now again frequently referred to as nonthoracic trauma—a syndrome resulting from hypoxia whose treatment is oxygen.

Meanwhile, during the 1930's, the attending internists at Kansas City General Hospital, particularly Paul Stuckey, M.D., and Hubert Parker, M.D., taught the mechanical minded orthopedists to recognize and treat the syndrome of fat embolism and demanded that the pathologist demonstrate the pathology. They relied on oxygen, transfusions, and heparin. In the late 1930's, after Hermann's paper from Cincinnati General Hospital was published, intravenous alcohol was added.

In the post-World War II period, the authors were thrown into juxtaposition by Kansas City General Hospital staff appointments. Victor Buhler, M.D., was chief of pathology from 1936 to 1940. Garrett Pipkin, M.D., was on the orthopedic staff from 1936 to 1961, and chief of orthopedics from 1951 to 1960. Their overlying interests resulted in the publishing of several papers on the illness of trauma.

In the 1960's, Buhler and Pipkin continued their studies at St. Joseph Hospital, Kansas City, Missouri, aided by a grant from the American Fracture Association. The authors, now referred to as arteriosclerotic senior citizens, have a compulsion to record the passing scene of their fortuitous experience for what value it may be to future workers in the field of trauma.

GARRETT PIPKIN
VICTOR BUHLER

ACKNOWLEDGMENTS

We would like to acknowledge the assistance of

Leonard Peltier, M.D., Ph.D., Professor of Orthopedic Surgery, University of Kansas Medical Center, on whose theoretical work we have leaned heavily.

Joseph F. Farney, D.V.M., who boarded our menageries and unselfishly recorded much routine and tiresome data.

Buck Martin, Chief of the Art Department, *Kansas City Star,* for our illustrations.

Roger O'Dneal (alias "Roger the Lodger"), Kansas City General Hospital Audio-Visual Department, for his microphotography.

Mrs. Margaret Lawler, former librarian, Jackson County Medical Society, for her many contacts with the Library of Congress.

Mr. Payne Thomas for his stimulation and editorial counsel.

Mrs. Marjorie J. Ray, secretary.

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THE ILLNESS OF TRAUMA

Chapter I

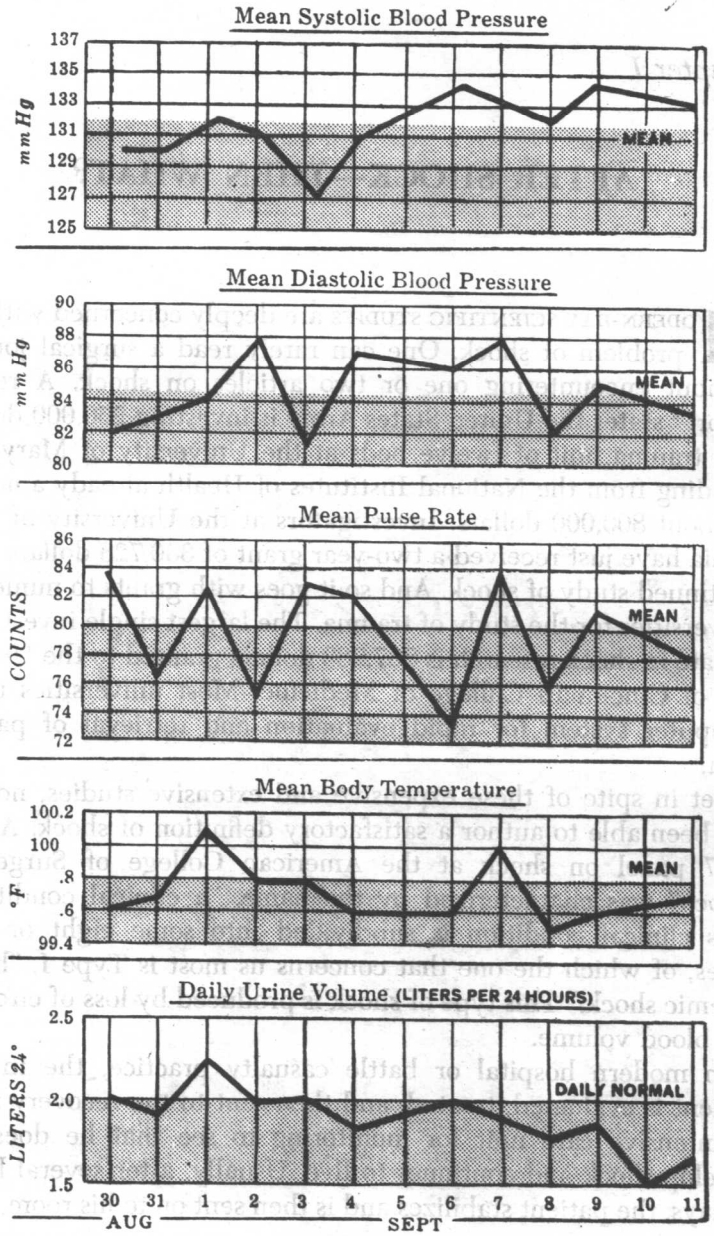
AFTER SHOCK—THEN WHAT?

MODERN-DAY SCIENTIFIC STUDIES are deeply concerned with the problem of shock. One can rarely read a surgical journal without encountering one or two articles on shock. A recent report [2] states the United States Army is investing 250,000 dollars in a trauma unit of twelve beds at the University of Maryland. Funding from the National Institutes of Health already amounts to about 800,000 dollars. Investigators at the University of California have just received a two-year grant of 330,728 dollars for a continued study of shock. And so it goes with grants to numerous universities for the study of trauma. The largest single investment to date in shock research is 337,283 dollars granted to the University of Cincinnati College of Medicine. Most universities use a computer system for rapid evaluation and retrieval of patient data.

Yet in spite of these expensive and extensive studies, no one has been able to author a satisfactory definition of shock. At the 1967 panel on shock at the American College of Surgeons,[4] "shock" was characterized by the name, "a clinical condition." This clinical condition is subdivided into some eight or nine types, of which the one that concerns us most is Type I, "hypovolemic shock." This type of shock is produced by loss of circulating blood volume.

In modern hospital or battle casualty practice, the injured patient is evaluated, treated, and then sent to the recovery room or intensive care unit for monitoring to see that he does not develop shock and continues to live. Usually, after several hours or days, the patient stabilizes and is then sent on to his room, only

VITAL SIGNS



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to become ill the second posttraumatic day (or postsurgical day) with fever, increased pulse and respiratory rates, and usually with symptoms of pneumonic congestion. It is this period of illness that comes on after accident or operation, starting about the forty-eighth hour and lasting six days, with which this book is concerned.

Recently a conference on pulmonary effects of nonthoracic trauma was held in Washington, D. C., February 29 to March 2, 1968, by a group of experts to study the pulmonary insufficiency that occasionally follows nonthoracic injury. The work of this conference comprises the entire September, 1968, issue of *The Journal of Trauma.* "Pulmonary effects of nonthoracic trauma," as we shall develop later, is but another alias for our thesis "the illness of trauma." At this conference a panel of experts in thoracic physiology, chemistry, and pathology received a selected number of fatal case reports from Vietnam. These experts evaluated these clinical reports against their own sophisticated background of research models. In such reports the alphabet is commonly used as shorthand or jargon such as $p_1 - O_2 - PaO_2$; pa CO_2, PVR and PAP to select a few at random. So to the clinical surgeon this excellent volume presents areas of hard reading.

Dr. Alfred P. Fishman,[1] in summarizing the work of this panel, expressed these comments:

> *Probably the most direct way to monitor the basic disturbances of serious and multiple injuries is by use of the conventional clinical indices.* (See Fig. 1) Granted that there may be little opportunity for relaxed observation, the vital signs are still familiar and available. A change in the respiratory frequency may herald the advent of an embolus to the lungs or the onset of pulmonary edema better than the ventilatory measurements that we have been discussing. Cold clammy skin may be a useful sign of intense autonomic activity. Distended neck veins may indicate overtransfusion. A decreasing urine flow in conjunction with systemic hypotension and tachycardia may be an ominous sign. If possible, serial x-rays of

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FIGURE 1. The clinical indices as recorded in a two-week study of a case of caisson disease. (Reproduced from *Nutrition Today,* p. 8, March, 1966.) Can you evaluate the findings of the first three days so as to successfully treat the patient? Or do you need a computer?

the chest are remarkably informative about heart and accumulation of fluid in the lungs during intensive treatment. *These indices have not yet received a pre-eminent place in our deliberations.*

The clinical indices, alias the vital signs, alias the graphic record on the patient's chart are at the present time the method of handling the bulk of the sick and injured. Only a few sample cases can receive computerized care in a nine (9) lead physiological and chemical analysis from a million-dollar-a-bed shock input. Granting that unlimited number of such highly specialized beds were available, we do not have enough nurses or doctors and paramedical personnel to man them. So the bulk of our injuries in the foreseeable future are of necessity going to be processed by the clinical indices.

Since the beginning of modern medicine, shock has been measured by the state of the blood pressure and one of its parameters, the palpation of the peripheral pulse.

So we have attempted to present in this monograph on the illness of trauma evaluations based on the more routine clinical findings and laboratory tests such as are available in our smaller general hospitals. When we stray from conventional clinical evaluations, it is only in hopes of translating some of the concepts of circulatory dynamics back to the level of common everyday clinical usage.

Chapter II

WHAT IS SHOCK—WHEN DOES IT END?

HEMORRHAGIC SHOCK has been a subject of concern among scores of investigators. Studies of its causes and proper treatment constitutes a continuing discussion which has not been fully resolved.

Stahl [21] defines shock as "a condition in which oxygen is unavailable to the tissue cell in sufficient amounts to allow the cell to function normally." He states:

> Under ordinary conditions the normal supply of oxygen to the cell allows sufficient energy for maintenance of cell integrity and is accomplished by constant removal of sodium ions from the cell by the so-called "sodium pumping" in order to maintain the normal distribution of sodium in the extracellular fluid, and potassium within the cell. Also each specialized type of cell requires energy to perform its particular function.
>
> Deprivation of sufficient oxygen results in anerobic metabolism, which is much less efficient than aerobic metabolism. It results in release of lactic acid causing metabolic acidosis, and sodium ion water imbalance. It is clear that such oxygen lack at the cellular level may be produced by a number of factors, viz:
>
> I. As physical shock—or block.
> II. Transport shock
> 1. Heart (pump)
> 2. Volume—circulating blood
> 3. Capacitance or tone of the vascular tree.
>
> Failure of the fine tuning or interchange of these three factors can result in vascular collapse.

The vascular collapse characterized by a weak and thready pulse and a cold, moist patient has been the usual measurement of shock, a clinical phantom when one attempts to record it.[16]

$$\text{But } \frac{\text{Cardiac Output} + \text{Circulating Blood Volume}}{\text{Total Peripheral Resistance}} = \text{Blood Pressure}$$

How shock can arise from sudden cardiac failure or just loss of circulating blood volume is easily comprehendible. Traumatic shock is a more complex variant of volume loss, hemorrhage, loss of extra cellular fluid, and continued psyche and physical trauma. Surgical shock has these factors, plus the added toxicity of the anaesthetic agent.

Thus, shock is concerned with total resistance of the microcapillary system. The capillary network is the body's largest organ, covering an estimated total length of 60,000 miles. It is supposed to be of single cell layer in thickness, covered with a layer of surfactant, supposedly dipalmityl lecithin, on which floats a layer of oxygen.[5, 8]

The capillary wall permits passage of fluids under normal circumstances. Trauma radically increases capillary permeability. In simple English, the lining of the vessel (oxygen) leaks, fluid loss is rapid, and red cells clump (Fig. 2). This is called sludging.[10,22]

As the fluid is depleted, hydrostatic pressure increases relative to osmotic pressure. Blood elements and protein are forced

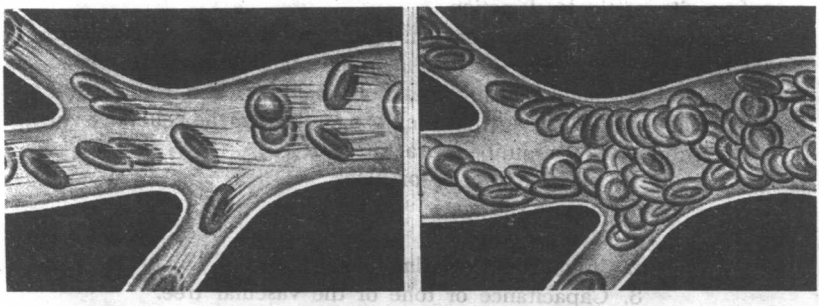

FIGURE 2. The concept of Sludging, after Gelin. *Left:* Normal microcirculation. *Right:* Red cell aggregation. (Redrawn from a colored plate in *Surgery, Gynecology and Obstetrics,* p. 64, March, 1968. Attributed to Gelin, L. E.: Conference of Low Molecular Weight Dextran in Shock. National Academy of Science, National Research Conference, Washington, D. C., 1963, pp. 6–24.)

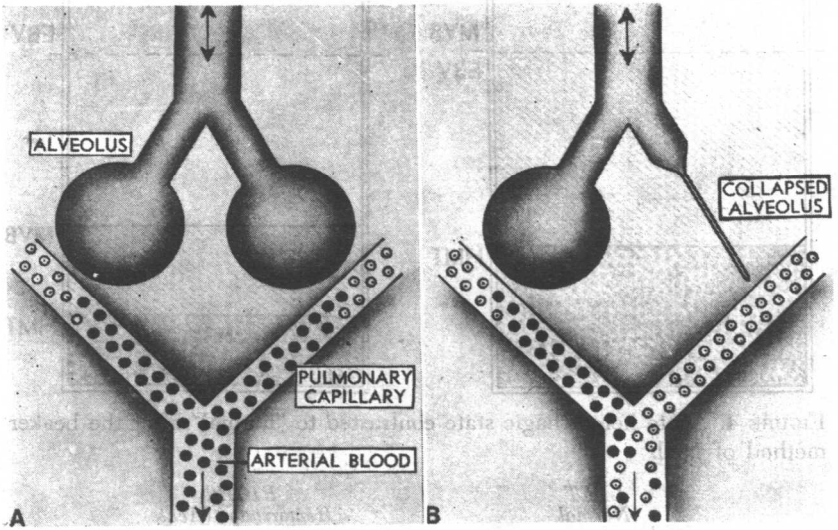

FIGURE 3. A. Simplified normal interaction between alveolus and capillary venous blood (gray discs) with low oxygen tension and high carbon dioxide tension is brought to the alveoli. Oxygen diffuses into the capillaries, combines with hemoglobin, and returns oxygen tension to arterial levels (black discs) carbon dioxide is removed. *B.* Collapsed alveoli are common after major surgery or injury. The blood that passes a collapsed alveolus does not take part in gas exchange. Low oxygen tension results. Such situations are termed shunts. (Redrawn from Winter, Peter M. and Lowenstein, E.: Respiratory failure. *Scientific American, 221*:25, November, 1969).

through capillary walls. Oxygen supply and elimination of cellular metabolic wastes are compromised, adding chemical insult to the injured area. Shunting of the pulmonary vascular bed further diminishes oxygenation (Fig. 3). Or, as Haldane [11] has put it, "lack of oxygen not only stops—but wrecks the machine."

Shock by our definition [17] is loss of vessel permeability resulting in hemoconcentration. The blood pressure falls, the hemoglobin value tends to become elevated, and the red cell volume rises.

Using the beaker method of Neil,[15] these changes may be illustrated as shown in Figure 4.

Assuming that the injured person survives his injury (and that his blood loss is not replaced by transfusion or packed cells), he is faced with a refill problem which becomes a state of hemodilu-

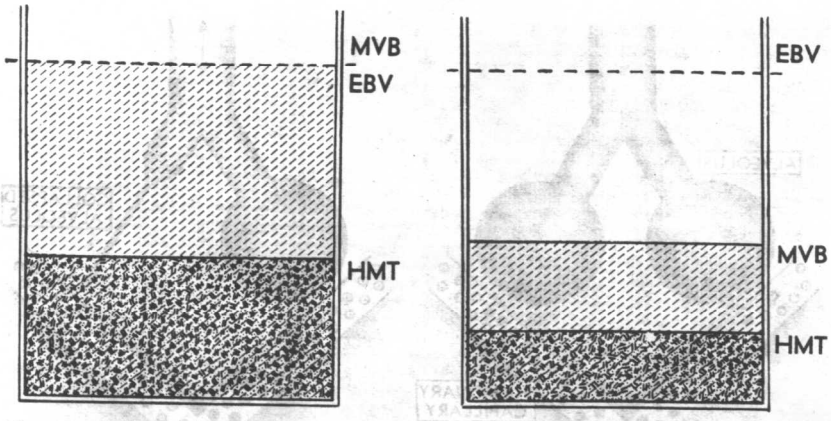

FIGURE 4. Acute hemorrhagic state contrasted to "normal" after the beaker method of Neil.

LEFT *Normal*		*RIGHT* *Hemorrhage (Acute)*	
MBV	5000	MBV	2500
EBV	5000	EBV	5000
HMT	40%	HMT	20%
RBC	2000	RBC	1000

(Redrawn from Neil, C. M.: The role of the blood volume in surgery. *International Surgery, 46*(5):452–460, November, 1966.)

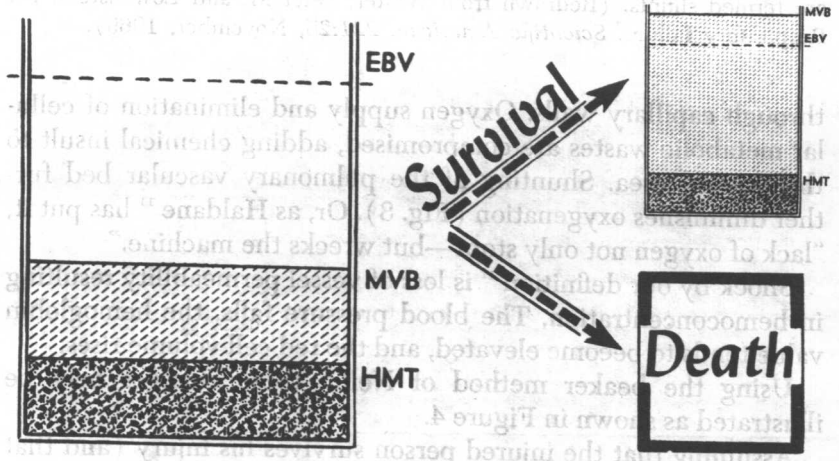

FIGURE 5. Shock is a state of hemoconcentration which moves rapidly to a state of hemodilution or death. Based on beaker technique of Neil.