Laura J. Moore
S. Rob Todd
Editors

Common Problems in Acute Care Surgery

Second Edition

EXTRAS ONLINE

Springer

Laura J. Moore • S. Rob Todd
Editors

Common Problems in Acute Care Surgery

Second Edition

Springer

Editors
Laura J. Moore
Associate Professor
Department of Surgery
The University of Texas McGovern Medical School - Houston
Chief, Section of Surgical Critical Care
Medical Director, Shock Trauma Intensive Care Unit
The Red Duke Trauma Institute, Memorial Hermann Hospital
Houston, TX, USA

S. Rob Todd
Professor of Surgery
Chief, Acute Care Surgery
Baylor College of Medicine
Chief, General Surgery and Trauma
Ben Taub Hospital
Houston, TX, USA

Videos can also be accessed at
http://link.springer.com/book/10.1007/978-3-319-42792-8

ISBN 978-3-319-42790-4 ISBN 978-3-319-42792-8 (eBook)

DOI 10.1007/978-3-319-42792-8

Library of Congress Control Number: 2016956003

Printed on acid-free paper

This Springer imprint is published by Springer Nature
The registered company is Springer International Publishing AG
The registered company address is: Gewerbestrasse 11, 6330 Cham, Switzerland

Foreword

It is a privilege to provide a foreword for the second edition of *Common Problems in Acute Care Surgery*. One of the editors (LJM) worked with me as a first-year medical student on a research project that won first prize and then later was my surgical critical care fellow. The other editor (SRT) is a former partner when we were both at one of the busiest trauma centers in the country. Both editors are highly committed to acute care surgery.

Acute care surgery is a continuously evolving specialty that encompasses trauma, emergency surgery, and surgical critical care. Trauma developed into an accepted specialty in the 1980s and 1990s. Initially trauma was a busy operative specialty associated with complex critical care, but, as our diagnostic modalities improved, and we realized that not every spleen or liver requires an operation, nonoperative care made the specialty less desirable to surgical trainees. At this same time, hospitals found it increasingly difficult to provide adequate coverage for emergency surgery. In response to fewer numbers of surgeons choosing trauma and surgical critical care as a career, a joint meeting of the American College of Surgeons, the American Association for the Surgery of Trauma (AAST), the Eastern Association for the Surgery of Trauma, and the Western Trauma Association was held in 2003 to address the problems of access to emergency surgical care and the future of trauma surgery. Later this same year, the AAST created an ad hoc committee to reorganize trauma, surgical critical care, and emergency surgery into what we now know as acute care surgery. The first formal AAST-accredited acute care surgery fellowship program began in 2008.

As acute care surgery has matured, it has continued to evolve and expand as the need for urgent or emergent surgical disease care continues to increase, especially as our population ages. The acute care surgeon is uniquely able to provide not only operative expertise but care from admission to discharge including critical care and, even when necessary, end-of-life care. Minimally invasive techniques are an increasing part of their operative lexicon. They must be adept at changing diagnostic modalities and critical care monitoring as these continue to change with technological advances.

Common Problems in Acute Care Surgery, 2nd edition, provides an evidence-based review of common problems that are encountered by the acute care surgeon. The target audience for this book includes trainees, physician extenders, and practicing surgeons who care for patients requiring emergency surgical care. It is organized in the same fashion as the first edition, in three parts: (1) general principles, (2) specific disease states, and (3) ethics, legal, and administrative issues. All of the topics in the first edition have returned and have been updated by experts from the acute care surgery community. In addition, a number of topics have been added. In the section on general principles, perioperative management of the cirrhotic patient, hemodynamic monitoring in the intensive care unit, and principles of vascular access are now included. In the section on specific disease states, management of intra-abdominal infections has been added. In the third section that includes administrative issues, a chapter outlining the development of an acute care surgery program is included.

I would like to acknowledge and thank the distinguished group of authors who participated in writing this book. I would also like to extend special thanks to the editors for taking on the task of updating a book on a constantly evolving field, acute care surgery.

Sacramento, CA, USA — Christine S. Cocanour

Preface

Acute care surgery continues to evolve as a specialty. With the growing patient need for access to emergency surgical services, acute care surgeons are providing much needed care for patients with urgent or emergent surgical disease. The field of acute care surgery has continued to evolve and expand in response to this need for reliable access to emergency surgical care. The care of the emergency surgical patient presents a complex set of challenges for surgeons. An understanding of the technical aspects of an operation combined with the presence of severe physiologic derangements presents a unique set of challenges to the surgeon. In order to deliver optimal care, the acute care surgeon must have expertise in both surgery and critical care. They must be facile with both open and laparoscopic surgical techniques, be familiar with the various diagnostic modalities available, be able to understand optimal resuscitation strategies, and be able to coordinate the care team in order to deliver rapid, evidence-based care for these challenging patients.

Common Problems in Acute Care Surgery, 2nd edition, addresses the common surgical emergencies encountered by acute care surgeons. The purpose of this text is to provide both trainees and practicing surgeons a comprehensive, evidence-based review of the most common clinical problems encountered by acute care surgeons. This second edition of the textbook includes updates to all of the topics from the first edition, as well as several new topics including the management of intra-abdominal infections, the management of the open abdomen, and hemodynamic monitoring of the critically ill surgical patient. The book is organized into three main sections. The first section focuses on general principles of acute care surgery including the initial evaluation and resuscitation, the perioperative management of the hemodynamically unstable patient, and common critical care issues encountered in the management of these patients. The second section focuses on specific disease states that are commonly encountered by acute care surgeons. Each chapter in this section addresses a specific clinical problem by describing the epidemiology, clinical presentation, diagnosis, management (including pertinent operative techniques), potential complications, and follow-up. The third and final section focuses on ethics and legal issues frequently encountered in acute care surgery.

Each of the authors in this text was selected for their expertise in the field of acute care surgery. We are grateful to the many surgeons who devoted countless hours in the preparation of this text. The end result is a resource that we hope will assist acute care surgeons in delivering compassionate, evidence-based care to the emergency surgical patient.

Houston, TX, USA

Laura J. Moore
S. Rob Todd

Contents

Part II Common Diseases in Acute Care Surgery

Contributors

Amit Agarwal, MD Department of Surgery, The University of Texas Health Science Center at Houston, Houston, TX, USA

Patricia Ayoung-Chee, MD, MPH, FACS Department of Surgery, New York University School of Medicine, New York, NY, USA

Seth A. Bellister, MD Department of Surgery, UT Health, Memorial Hermann Hospital Red Duke Trauma Institute, Houston, TX, USA

Walter L. Biffl, MD Acute Care Surgery, The Queen's Medical Center, Honolulu, HI, USA

Department of Surgery, John A. Burns School of Medicine, University of Hawaii, Honolulu, HI, USA

Shanda H. Blackmon, MD, MPH, FACS Division of General Thoracic Surgery, Mayo Clinic, Rochester, MN, USA

Marc I. Brand, MD Section of Colon and Rectal Surgery, Department of General Surgery, Rush University Medical Center, Chicago, IL, USA

Clay Cothren Burlew, MD, FACS Department of Surgery, Denver Health Medical Center, University of Colorado School of Medicine, Denver, CO, USA

Gregory E. Catlett Jr., MD Department of Orthpedic Surgery, McGovern Medical School at University of Texas Health Science Center at Houston, Houston, TX, USA

Edie Chan, MD Department of Surgery, Rush University Medical Center, Chicago, IL, USA

Winston M. Chan, MD Department of Surgery, The University of Texas Health Science Center at Houston, Houston, TX, USA

Chistine S. Cocanour, MD, FACS, FCCM Professor of Surgery, Surgical Critical Care Fellowship Program Director, The University of California - Davis Medical Center, Sacramento, CA, USA

Bryan A. Cotton, MD, MPH Department of Surgery, The University of Texas Health Science Center, Houston, TX, USA

Marie Crandall, MD, MPH Department of Surgery, University of Florida College of Medicine, Jacksonville, FL, USA

Benjamin L. Davis, MD FACU Surgeon, Second Medical Battalion, Second Marine Logistics Group, Camp Lejeune, NC, USA

Claire de Crescenzo, MD School of Medicine, UC Davis Medical Center, Sacramento, CA, USA

Millard Andrew Davis, MD Department of Surgery, Baylor College of Medicine, Houston, TX, USA

Karen J. Dickinson, MBBS, BSc, MD, FRCS Division of General Thoracic Surgery, Mayo Clinic, Rochester, MN, USA

Jon D. Dorfman, MD Department of Surgery, University of Massachusetts Medical School, Worcester, MA, USA

James K. Dzandu, PhD Trauma, Services, HonorHealth John C. Lincoln Medical Center, Phoenix, AZ, USA

Soumitra Eachempati, MD Department of Surgery, New York Presbyterian-Weill Cornell Medicine, New York, NY, USA

Bridget N. Fahy, MD, FACS Division of Surgical Oncology, Department of Surgery, University of New Mexico, Albuquerque, NM, USA

Kevin W. Finkel, MD, FACP, FASN, FCCM Division of Renal Diseases and Hypertension, University of Texas Health Science Center at Houston – McGovern Medical School, Houston, TX, USA

Joshua L. Gary, MD Department of Orthopaedic Surgery, McGovern Medical School at University of Texas Houston Science Center at Houston, Houston, TX, USA

Ramyar Gilani, MD, FACS Division of Vascular Surgery, Michael E. DeBakey Department of Surgery, Ben Taub Hospital, Houston, TX, USA

Stephanie Gordy, MD, FACS Michael E. DeBakey Department of Surgery, Ben Taub Hosptial, Baylor College of Medicine, One Baylor Plaza, Houston, TX, USA

David C. Gochnour, MD Department of Surgery, College Station Medical Center, College Station, TX, USA

Tim Gutierrez, MD Department of Surgery, Methodist Dallas Medical Center, Dallas, TX, USA

Maamoun A. Harmouch, MD Department of Surgery, The University of Texas Health Science Center at Houston, Houston, TX, USA

John A. Harvin, MD, FACS Division of Acute Care Surgery, Department of Surgery, University of Texas Medical School at Houston, Houston, TX, USA

Mark J. Hobeika, MD Department of Surgery and Internal Medicine, The University of Texas Health Science Center at Houston, Houston, TX, USA

John Holcomb, MD, FACS Division of Acute Care Surgery, Department of Surgery, The University of Texas Medical School at Houston, Houston, TX, USA

Alexzandra Hollingworth, MD, FACS Trauma Services, HonorHealth John C. Lincoln Medical Center, Phoenix, AZ, USA

Julie L. Holihan, MD Department of Surgery, University of Texas Health Science Center at Houston, Houston, TX, USA

Janeen R. Jordan, MD, FACS Department of Surgery, University of Florida Health, Gainesville, FL, USA

Lia Jordano, MD Department of Surgery, Rush University Medical Center, Chicago, IL, USA

Tien C. Ko, MD Department of Surgery, University of Texas Health Sciences Center at Houston, Houston, TX, USA

Lillian S. Kao, MD, MS Department of Surgery, University of Texas Health Sciences Center at Houston, Houston, TX, USA

Rosemary Kozar, MD, PhD, FACS Shock Trauma, University of Maryland, Baltimore, MD, USA

Laura A. Kreiner, MD Department of Surgery, MetroHealth Medical Center/Case Western Reserve University School of Medicine, Cleveland, OH, USA

John C. Kubasiak, MD Section of Colon and Rectal Surgery, Department of General Surgery, Rush University Medical Center, Chicago, IL, USA

Mike K. Liang, MD Department of Surgery, University of Texas Health Science Center at Houston, Houston, TX, USA

Ning Lu, MD University of Hawaii Residency Program in General Surgery, Honolulu, HI, USA

Alicia J. Mangram, MD, FACS Trauma Services, HonorHealth John C. Lincoln Medical Center, Phoenix, AZ, USA

Gary T. Marshall, MD Department of Surgery, New York University School of Medicine/ Bellevue Hospital Center, New York, NY, USA

James J. McCarthy, MD, FACEP Emergency Medical Services, Texas Medical Center, Memorial Herman Hospital, McGovern Medical School, University of Texas Health Science Center at Houston, Houston, TX, USA

Caitlin A. McIntyre, MD Department of Surgery, New York Presbyterian-Weill Cornell Medicine, New York, NY, USA

Michelle K. McNutt, MD, FACS Department of Surgery, UT Health, Memorial Hermann Hospital Red Duke Trauma Institute, Houston, TX, USA

Marc Mesleh, MD Department of Surgery, Rush University Medical Center, Chicago, IL, USA

Adam J. Meyers, MD Department of Surgery, University of California Davis Medical Center, Sacramento, CA, USA

David Evan Meyer, MD Department of Surgery, University of Texas Health Sciences Center at Houston, Houston, TX, USA

Alicia M. Mohr, MD, FACS, FCCM Department of Surgery, University of Florida Health, Gainesville, FL, USA

Frederick A. Moore, MD, FACS, MCCM Department of Surgery, University of Florida Health, Gainesville, FL, USA

Laura J. Moore, MD Associate Professor, Department of Surgery, The University of Texas McGovern Medical School - Houston, Chief, Section of Surgical Critical Care, Medical Director, Shock Trauma Intensive Care Unit, The Red Duke Trauma Institute, Memorial Hermann Hospital, Houston, TX, USA

Maureen D. Moore, MD Department of Surgery, New York Presbyterian-Weill Cornell Medicine, New York, NY, USA

Krislynn M. Mueck, MD, MPH Department of Surgery, University of Texas Medical School at Houston, Houston, TX, USA

Christian Perez, MD Department of Surgery, University of Texas Medical School at Houston, Houston, TX, USA

K. Shad Pharaon, MD Division of Trauma and Acute Care Surgery, Surgical Critical Care, PeaceHealth Southwest Medical Center, Vancouver, WA, USA

Division of Trauma, Critical Care, and Acute Care Surgery, Department of Surgery, Oregon Health and Science University, Portland, OR, USA

Heena P. Santry, MD, MS Department of Surgery, University of Massachusetts Medical School, Worcester, MA, USA

Diane A. Schwartz, MD Department of Surgery, The Johns Hopkins Bayview Medical Center, Baltimore, MD, USA

Michelle H. Scerbo, MD Department of General Surgery, University of Texas Health Science Center at Houston, Houston, TX, USA

Shinil K. Shah, DO Department of Surgery, University of Texas Medical School at Houston, Houston, TX, USA

Michael E. DeBakey Institute for Comparative Cardiovascular Science and Biomedical Devices, Texas A&M University, Houston, TX, USA

Sherry L. Sixta, MD Department of Surgery, Christiana Care Health System, Newark, DE, USA

Emmanuel Sonnaike, MD Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston, TX, USA

Robert Ellis Southard, MD Baylor College of Medicine, Houston, TX, USA

Jeffrey P. Spike, PhD McGovern Center for Humanities and Ethics, University of Texas Health Science Center, Houston, TX, USA

Anthony Tannous, MD School of Medicine, University of Maryland, Baltimore, MD, USA

Nirav C. Thosani, MD, MHA Division of Gastroenterology, Hepatology and Nutrition, Department of Internal Medicine, University of Texas Medical School at Houston, Houston, TX, USA

Michael S. Truitt, MD Department of Surgery, Methodist Dallas Medical Center, Dallas, TX, USA

Daniel Vargo, MD, FACS Department of Surgery, University of Utah School of Medicine, Salt Lake City, UT, USA

R. Mario Vera, MD Baylor College of Medicine, Houston, TX, USA

Peter A. Walker, MD Department of Surgery, University of Texas Medical School at Houston, Houston, TX, USA

Jeremy L. Ward, MD Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston, TX, USA

Justin J.J. Watson, MD Division of Trauma, Critical Care, and Acute Care Surgery, Department of Surgery, Oregon Health and Science University, Portland, OR, USA

Jennifer Marie Watters, MD Division of Trauma, Critical Care, and Acute Care Surgery, Department of Surgery, Oregon Health and Science University, Portland, OR, USA

Rebecca JoAnne Weddle, MD Division of Trauma, Critical Care, and Acute Care Surgery, Department of Surgery, Oregon Health and Science University, Portland, OR, USA

Andrea Weitz, MD Department of Surgery, University of Utah School of Medicine, Salt Lake City, UT, USA

Holly Whitt, BS Department of Surgery, The University of Texas Health Science Center, Houston, TX, USA

Curtis J. Wray, MD Department of Surgery, University of Texas Health Sciences Center at Houston, Houston, TX, USA

J. Davis Yonge, MD Department of Surgery, New York University School of Medicine, New York, NY, USA

Part I

General Principles

Initial Resuscitation and Management of the Hemodynamically Unstable Patient

1

Diane A. Schwartz and John Holcomb

Hemorrhagic and Hypovolemic Shock and Initial Stabilization Maneuvers

In 1946 hemorrhagic shock was induced in animal models and a stratification system emerged: simple hypotension, which was noted to always be reversible if identified and treated; impending shock, which was reversible if treated aggressively; and irreversible shock state, where hypotension, sustained by high-volume blood loss, correlated to notable metabolic derangement [1]. The authors concluded that hemorrhagic shock did not occur at a specific volume loss or blood pressure, but was rather a fluid state that required early recognition by the treating physician and immediate intervention during the reversible period.

Hemorrhagic shock is defined as a mismatch between cellular perfusion and metabolism. Strict adherence to the definition results in difficulty identifying compensated shock states, however, since compensated shock does not always have a straightforward clinical picture. Compensated and severe hemorrhagic shock occur on a spectrum of metabolic acidosis, blood loss, poor tissue perfusion, tissue injury, and ineffective oxygen extraction (Table 1.1 and Fig. 1.1).

Hemorrhage is commonly categorized by volume and percent blood loss with specific findings at defined losses [2]. Interestingly these categories are largely based on opinion rather than objective clinical data. Clinical parameters are not markedly different from baseline in phases one and two of shock, contributing to the difficulty in recognizing shock in its early stages. In providing care to the critically injured patient, it is of utmost importance to have the ability to diagnose impending or early hemorrhagic shock. It is rather easy to diagnose severe hemorrhagic shock; however, the affected patients have already undergone cardiovascular collapse and are near death. The astute clinician will prefer to intervene earlier, when the diagnosis is more obscure and reversal of the shock state is possible. Once recognized, directed treatment of imminent shock or ongoing hemorrhage begins.

During field resuscitation, patients receive treatments necessary to control bleeding. Several centers tout an integrated database or registry to incorporate pre-hospital data to analyze outcomes [3–5]. One pre-hospital intervention to consider in the management of hemorrhage is infusion of blood products. In patients who have the opportunity of survival, meaning that they are in severe hemorrhagic shock, but are able to resuscitate with source controlled prior to cardiopulmonary collapse and death, the first 6 h of resuscitation is an important time frame. This is because outcomes have been shown to improve with early and aggressive resuscitation during the first 6 h.

The Center for Translational Injury Research in Houston has shown improved outcomes and negligible waste associated with the use of blood and FFP in the pre-hospital setting [6]. The outcomes seem to be most notable in patients in severe hemorrhagic shock who survive to hospital arrival. This particular patient population faces imminent death within the first 6 h danger period of hemorrhagic shock. In receiving the products they need earlier, their shock process is mitigated faster and likely accounts for the outcome improvement. The Department of Defense is leading a prospective study for the use of thawed FFP in the pre-hospital setting, results of which are pending [7]. In centers where blood product cannot be transfused in the field or plasma cannot be kept in thawed state, protocols assist to determine hospital transfusion requirements based on field data [8].

Apparent blood loss in the field should be managed with application of direct pressure or a tourniquet. The use of tourniquets has turned some of the most life-threatening

D.A. Schwartz
Department of Surgery, The Johns Hopkins Bayview Medical Center, Baltimore, MD, USA

J. Holcomb (✉)
Division of Acute Care Surgery, Department of Surgery, The University of Texas Medical School at Houston, 6410 Fannin St., Houston, TX 77030, USA
e-mail: john.holcomb@uth.tmc.edu

L.J. Moore, S.R. Todd (eds.), *Common Problems in Acute Care Surgery*, DOI 10.1007/978-3-319-42792-8_1

Table 1.1 Estimated blood loss[a] based on patients' initial presentation

	Class I	Class II	Class III	Class IV
Blood loss (ml)	Up to 750	750–1500	1500–2000	>2000
Blood loss (% blood volume)	Up to 15	15–30	30–40	>40
Pulse rate (per minute)	<100	100–120	120–140	>140
Systolic blood pressure	Normal	Normal	Decreased	Decreased
Pulse pressure (mmHg)	Normal or increased	Decreased	Decreased	Decreased
Respiratory rate	14–20	20–30	30–40	>35
Urine output (ml/h)	>30	20–30	5–15	Negligible
Central nervous system/mental status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Initial fluid replacement	Crystalloid	Crystalloid	Crystalloid and blood	Crystalloid and blood

Reprinted with permission from American College of Surgeons. ATLS student manual. 9th ed. 2012
[a]For a 70-kg man

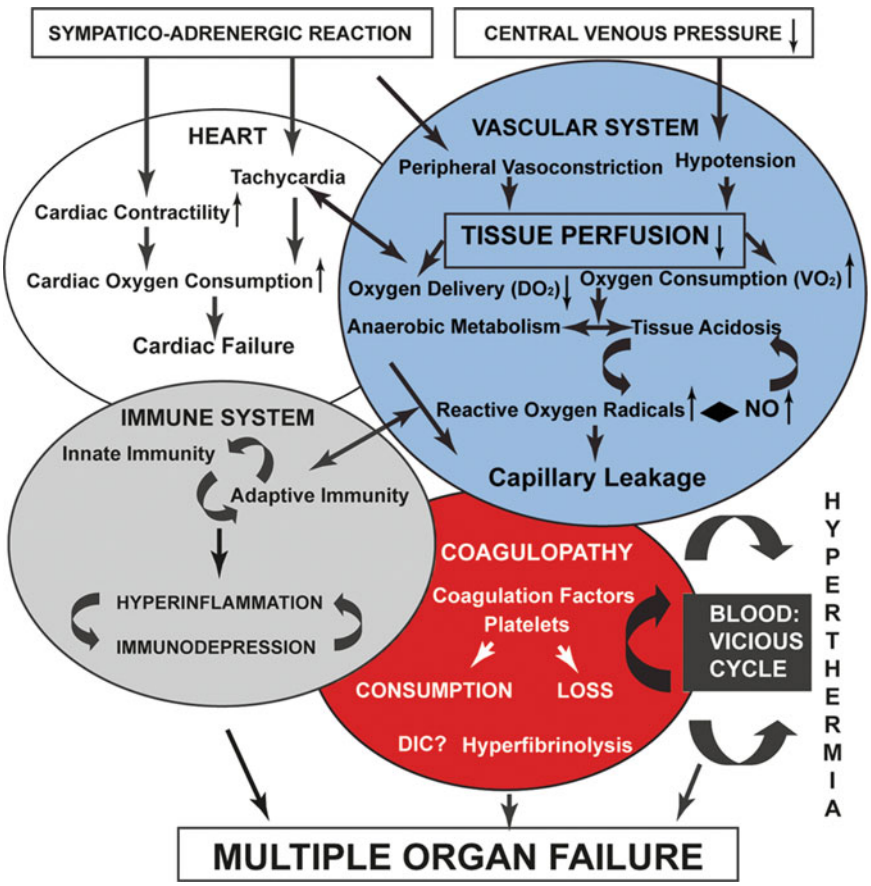

Fig. 1.1 Pathophysiology of hemorrhagic shock. (From Angele MK, Schneider CP, Chaudry IH. Bench to bedside review: latest results in hemorrhagic shock. Crit Care. 2008;12(4):218, with permission.)

injuries into ones where life and limb can be salvaged. The resurgent use of tourniquets has been overwhelmingly supported in the military data from the Iraq and Afghanistan experience, where it is shown that there are virtually no adverse effects of the tourniquet itself if left in place for less than 2 h [9]. Even in inexperienced hands, tourniquets have been shown to prevent life-threatening exsanguination and should be applied in any pre-hospital situation in which extremity hemorrhage exists and prior to the onset of exsanguination [10]. There are several commercial devices available and their purpose is to exert enough circumferential pressure to prevent blood from flowing into the extremity in question [11]. Contrary to older teaching, use of a tourniquet does not cause increased amputation rates [12]. Use of tourniquets is ubiquitous on the battlefield, and in many civilian centers use has become routine.

Massive Transfusion

Since no single injury, other than severe head injury, has ever been identified to correlate with non-survivability, massive transfusion protocols should not be held for an assumption of impending mortality [13]. According to this article, no lab

value, no injury severity score (ISS), no demographic data, and no vital sign, singly or grouped, accurately determine a mortality score. A second manuscript from the same group of authors discusses a potential model for predicting mortality at 30 days; however, still there are cautions against using such a model to withhold much-needed blood products during resuscitation [14]. Factors most predictive of 24-h mortality are pH, base deficit, and amount of blood transfused within the initial 6 h. Factors at 30 days that are of significance include age and ISS on admission.

At The Texas Trauma Institute at Memorial Hermann Hospital in Houston, Texas, the massive transfusion protocol is activated for any patient who is suspected to require substantial transfusion, based on any one of the following: prehospital administration of blood or blood products by Memorial Hermann Life Flight, heart rate on arrival of more than 120 beats per minute, systolic blood pressure on arrival of less than 90, a positive FAST exam, penetrating or blunt trauma mechanism, or having a requirement for uncrossmatched blood in the emergency room on arrival. These recommendations come from retrospective data comparing predictive scores for massive transfusion. Using these parameters a score of two or greater was found to be 75 % sensitive and 86 % specific, correlating relatively well without statistical significance to other published scoring systems [15]. The goal of this guideline is to make a continuous supply of six units of packed red blood cells (PRBC), six units of plasma (FFP), and one dose of a six-pack of platelets readily available. After 6 units of PRBC it is advised to check a fibrinogen level and if less than 150 mg/dl to administer ten units of cryoprecipitate. Serial labs are also drawn during the massive transfusion and include lactate, arterial blood gas, rapid thromboelastogram (TEG), coagulation panel, and complete blood count (CBC) with differential and platelet count. It should be noted that a TEG is available within minutes (5 min for a rapid TEG), whereas the coagulation panel and CBC take more than 45 min to process [16]. Additionally all level 1 trauma activations, which are the highest acuity patients at Memorial Hermann Hospital, are typed and crossed on arrival so that type-specific blood may be given when available.

There seems to be an advantage to maintaining ratio driven resuscitation initially, followed by goal-directed resuscitation once source control is achieved [17]. Data supporting the 1:1:1 FFP:platelet:PRBC ratio initially came from military literature dating from 2007, which shows an improvement in mortality for patients receiving such ratios [18]. This was later extrapolated in several studies to the civilian population and further propagated in several trauma centers as a new standard of care [19]. A review article from 2010 looked at nine additional observational studies that were published after the 2007 article [20]. There are now randomized trials comparing ratios and showing improvement in mortality within the crucial 3 h window for patients receiving 1:1:1 transfusions [21]. The majority of trauma centers now use this approach.

A goal-directed transfusion protocol is a seemingly attractive approach for trauma resuscitation once source control is achieved. Originally, massive transfusion protocols were designed to rapidly and reliably provide products to patients who had clinical evidence of substantial hemorrhage. Products and blood were given without a specific ratio until patients either expired or improved clinically. After introduction of the 1:1:1 ratio, which targets the coagulopathy that accompanies massive transfusion, surgeons began to question if transfusion should be automatic or rather if it should be guided by objective data and lab values. One of several manuscripts on goal-directed resuscitation expresses the idea that resuscitation may be more functional and cost effective if lab values, such as TEG, are used to guide decision making during the resuscitation [22]. This concept relies on laboratory reports being ordered, drawn, sent to, and returned from the laboratory in a clinically relevant time frame. While there have been questions raised regarding TEG's role in reducing mortality or improving transfusion-related outcomes, there is no question that TEG remains the fastest real-time data set available for coagulopathy assessment [1, 23]. Most clinicians have combined these two approaches. Using a ratio based approach is optimal when the patients are rapidly bleeding. As bleeding slows, TEG guided transfusion therapy is appropriate [24].

Acidosis

Acidosis is one component of the trauma triad of death that must be recognized as a propagator of coagulopathy and continued hemorrhage. Animal and human models have shown direct correlation of lactate and mortality risk [25]. Lactate levels are elevated in hemorrhagic shock and will persist in times of suboptimal tissue perfusion. pH correlates with overall resuscitation and tissue perfusion, maintaining statistical relevance even in cases where permissive hypotension is maintained [26]. Lactic acidosis is directly reflective of continued need for resuscitation but takes longer to normalize than other parameters, such as vital signs. In general patients will reach two separate resuscitative goals: source control, which happens first, and physiologic, which lags. Serum acid levels can be extrapolated from pH, lactate levels, CO_2 or bicarbonate on blood work; all are useful markers of cellular aerobic metabolism [27]. In using these other markers of acidosis, however, it is imperative to understand that not all acidosis is derived from lactate, and not all acidosis is directly related to ongoing anaerobic metabolism. For example, patients with severe hemorrhagic shock may have persistence of acidosis related to acute kidney injury despite

resuscitation being completed. In these cases lactate may be normal and acid levels are derived from other sources. There has been recent interest in non-invasive pH monitoring, since it seems to reliably track the success of resuscitation and risk of mortality [28, 29]. Non-invasive monitoring systems are desirable in both combat and civilian arenas where resources for invasive intervention may be lacking.

Lactate, serum bicarbonate, base deficit, hemoglobin, or tissue oxygenation are some of the most crucial lab values in determining metabolic acidosis, which occurs with poor tissue oxygen extraction and indicates shock at the cellular level [30–36]. Lactate, in the pre-hospital setting, may be more predictive of prognosis than are vital signs, which can be fairly stable until hemodynamic collapse ensues [37]. Lactate increases in under-perfused tissues and can be an early predictor of impending shock, and helps differentiate the stable patient from the one in a compensated shock state.

Base deficit is a reflection of metabolic acidosis secondary to unmeasured anions, which is typically assumed to be lactate in the trauma patient [38]. Base deficit, lactate, anion gap, and bicarbonate levels all correspond to metabolic acidosis and have all been shown to predict morbidity and mortality [39–42]. However, bicarbonate is only a single marker of acid–base status, whereas anion gap, base deficit, and lactate all have some dependence on electrolytes, pH, and buffer capacity of blood [43]. There does not seem to exist great consensus in the literature regarding which is the best predictor of mortality [44].

Up to a third of patients in the ICU show discordance between their base deficit and lactate, and in these situations it has been shown that lactate is more predictive of overall outcome, when it differs significantly from base deficit [45]. Authors from this source imply that base deficit on its own does not have the predictive capacity for mortality that lactate has. On the other hand, while lactate is the most helpful in the initial phase of resuscitation, it is not as accurate in determining the ongoing causes of metabolic acidosis in critical situations outside of trauma where lactate may not elevate, such as respiratory alkalosis and diabetic ketoacidosis.

Serum bicarbonate will correlate with base deficit only when the pH is constant, which has clinical implications in the patient whose standard chemistry is drawn from a venous line at a different time than the arterial blood gas is collected [46]. The fluctuating pH may affect the accuracy of either measurement when compared to the other. There may be a significant difference in base deficit when comparing arterial to venous samples. Venous samples may be more sensitive to changes in pH, pCO_2, and pO_2 resulting in earlier changes in base deficit [47].

Coagulopathy, acidosis, and hypothermia portend the downward spiral into fulminant hemorrhagic shock. The key to understanding hemorrhagic shock is to understand the interactions of the lethal triad and the human body's capacity to self-correct versus what must be medically and surgically repaired. Acidosis is a product of poor tissue perfusion and death at the cellular level [48]. Lactic acidosis is a finding associated with cellular anoxia. Free radical release during tissue hypoxia also contributes to overall organ dysfunction and further perpetuates the cascade [49]. The coagulopathy is secondary to dilution, platelet dysfunction, cellular damage, decreased hepatic synthesis of factors, and shunting of proteins away from creating coagulation factors and toward production of acute-phase reactants [50–53]. Hypothermia occurs secondary to decreased metabolism. It is also associated with infusion of cold or chilled blood products and crystalloid, and hypothermia itself contributes to continued perpetuation of coagulopathy [54]. Furthermore it is the mismatch between oxygen delivery and consumption with resultant organ dysfunction that defines the shock state [55]. All three elements of the lethal triad contribute and potentiate the death spiral after substantial bleeding. Interruption of this process is paramount to survival.

Hypothermia

Hypothermia, defined as a core temperature of less than 34°, is a definitive contributor to ongoing shock and ineffectiveness of resuscitation. It must be immediately corrected in the trauma patient who is coagulopathic [56]. Combat trauma physicians have experienced increased morbidity and mortality, likely due to the loss of function of platelets and coagulation factors, in hypothermic patients [57–60]. While the animal model has demonstrated survival benefit from mild hypothermia during initial resuscitation, coagulopathy in humans seems exacerbated by temperatures outside of physiologic range and is not recommended [61–63]. Moreover hypothermia does not have a role in the current resuscitation guidelines for hemorrhage and should not be employed, even if other clinical factors support its use. Heat loss is attributed to time spent in the field, exposure of the patient to external elements, chilled resuscitation fluid including blood product and crystalloid, air temperature in the emergency department and operating room, open body cavities during operation, and transport to a variety of locations without appropriate warming mechanisms. It is advised that the initial examination proceed with cognizance of the rapidity at which body heat is lost, and that patients be covered as quickly as possible with warm blankets. Bair huggers, ambient warming, institution of protocols that enforce use of hotlines or other mechanisms to warm fluid are all adjuncts to maintaining body temperature. Protocols for hypothermia avoidance are helpful in standardizing mechanisms for warming while bringing attention to this very important component of physiologic normalcy [64].

Crystalloid

There is no longer argument on the preferred resuscitation fluid for patients in hemorrhagic shock—it is maintained ratios of FFP, platelets, and blood—not crystalloid [65, 66]. Crystalloid should not be considered first line therapy for resuscitation in hemorrhagic shock in any facility where blood products are available. It seems intuitive that if a person is hemorrhaging, correction of that shock will be contingent on the repletion of blood, and that his or her coagulopathy will respond to transfusion of plasma and platelets. However, replacement of volume by crystalloid represents classical teaching and guidelines for correction of the initial phase of hemorrhagic shock [67]. Advanced Trauma Life Support (ATLS) discusses placing two large-bore IVs and bolusing 1 l of crystalloid for any patient assumed to be in hemorrhagic shock or any patient with significant blood loss [2]. However, recent data suggest that as little as 1.5 l of fluid has negative clinical implications and numerous sources are refuting the benefit of large-volume crystalloid resuscitation in hemorrhagic shock [68].

While many clinicians consider lactated Ringer's and normal saline interchangeable, they are not. Multiple studies in the swine model compare the use of various crystalloid solutions, focusing on lactated Ringer's solution and normal saline. The swine model demonstrates that if shock is induced and maintained for 30 min, followed by resuscitation with either normal saline or lactated Ringer's solution, the animals resuscitated with Ringer's lactate have better improvement in markers of shock, pH, and extracellular lung water [69]. In this study neutrophil activation contributes to cellular damage. Other studies support the neutrophil activation phenomenon; dextran is the biggest activator, followed by normal saline and then lactated Ringer's [70]. Colloid, plasma, and blood have also been implicated as morbid contributors to effects on neutrophil activation, mainly in the pulmonary system [71–73].

Lactated Ringer's, as a resuscitation fluid, yields less acidosis and less coagulopathy than seen with similar volumes of normal saline [74]. Normal saline causes a well-recognized metabolic hyperchloremic acidosis; patients resuscitated with lactated Ringer's do not achieve such levels of acidosis. Furthermore, normal saline-resuscitated patients demonstrate more blood loss than those resuscitated with lactated Ringer's [75]. This has been demonstrated also in the vascular literature. In a study of aortic repairs it was shown that there was more perioperative bleeding and acidosis when normal saline was used as opposed to lactated Ringer's [76]. There was no statistically significant difference in outcome however. Even despite the better physiologic results with lactated Ringer's resuscitation as compared to normal saline, lactated Ringer's still would not be the first choice for resuscitation in a patient with hemorrhagic shock as excessive bleeding is not well controlled with replacement of volume by crystalloid [77].

Another substitute for balanced crystalloid solution is Plasma-lyte, which has been shown to be cost effective and a potentially better crystalloid choice when compared to normal saline [78]. The enthusiasm for this fluid as a carrier and for maintenance may be due to the fact that it does not contain calcium and therefore can be infused with blood product without concern for crystallization of the line and less morbidity than other non-normal saline balanced fluids [79]. Importantly plasmalyte does not have the acidosis inducing profile of normal saline.

Permissive hypotension purposefully maintains mean arterial pressure as low as possible to ensure adequate organ perfusion. If the minimum mean arterial pressure is not exceeded with over-resuscitation, the delicate new clot formation should not be disrupted prior to operative intervention [80, 81]. These authors show that by purposefully maintaining mean arterial pressure no greater than 50 mmHg, the patients in these groups are not afflicted with coagulopathy to the same degree as controls that are resuscitated to a mean arterial pressure of greater than 65 mmHg. Earlier data from animal models show no difference in ultimate outcome when hypotension is maintained; end organ perfusion and prevention of metabolic perturbations that can occur when tissue oxygenation is inadequate are the goals of permissive hypotension [82]. That is to say that when metabolic acidosis is controlled and the mean arterial pressure is minimized on purpose, patients do not show any long-term adverse effects compared to patients whose resuscitation targets a higher mean arterial pressure. It is unclear how long patients can remain hypotensive without deleterious effects. The original descriptions of this concept date to World Wars 1 and 2. The original civilian studies on this topic show less intraoperative bleeding and overall fluid requirement and hence less postoperative morbidity when this strategy is applied [83]. Survival is improved by limiting crystalloid infusion. Furthermore overaggressive resuscitation to a physiologically normal blood pressure may contribute to ineffective hemostasis, termed "popping the clot," shown in an animal study where raising the blood pressure caused re-bleeding and increased mortality [84]. This cycle of repeated resuscitation and bleeding is ultimately detrimental to clot stability and to overall survival [85].

Hypertonic Saline

Crystalloid evaluation would not be complete without consideration of hypertonic saline. Hypertonic saline use is pervasive throughout the literature. Prior to the recent explosion of blood product-based resuscitation, crystalloid resuscitation was the standard of care. Hypertonic saline shows some improvement in blood pressure and arguable survival difference for patients

who receive it in the pre-hospital setting [86]. Interest also exists in combat medicine where space, weight, and facility of transport of medical devices remain an important consideration. There are studies showing decreased pre-hospital fluid requirements in patients who receive hypertonic saline during transport [87]. Immunomodulatory effects are enhanced with single administration of 250 ml of hypertonic saline in the initial phase of resuscitation of hemorrhagic shock, and this could have additional effects on patients with later discovered head injury [88, 89]. A large study of hypertonic saline showed statistical difference in outcome in pediatric head-injured patients when compared with isotonic fluid administration [90]. Hypertonic saline decreases interstitial pressure and consequently decreases bowel edema, which may be a potential benefit of using it on the patient whose abdomen is still open [91, 92]. Animal studies in the 1990s showed that there was no protective effect or difference in outcome for the patient in hemorrhagic shock with a head injury [93]. Since that time several studies examining hypertonic saline as a resuscitative fluid have been terminated secondary to futility and concerns for patient safety [94, 95]. It is still debatable that hypertonic has a physiologic or survival advantage when compared to other crystalloid formulations when used as a primary resuscitation fluid [96].

Table 1.2 Current indications and contraindications for EDT

Indications
Salvageable post-injury cardiac arrest:
Patients sustaining witnessed penetrating trauma with <15 min of pre-hospital CPR
Patients sustaining witnessed blunt trauma with <5 min of pre-hospital CPR
Persistent severe post-injury hypotension (SBP ≤60 mmHg) due to:
Cardiac tamponade
Hemorrhage—intrathoracic, intra-abdominal, extremity, cervical
Air embolism
Contraindications
Penetrating trauma: CPR >15 min and no signs of life (pupillary response, respiratory effort, or motor activity)
Blunt trauma: CPR >5 min and no signs of life or asystole

From Mears G, Glickman SW, Moore F, Cairns CB. Data based integration of critical illness and injury patient care from EMS to emergency department to intensive care unit. Curr Opin Crit Care. 2009;15(4):284–9, with permission

The Role of Cardiopulmonary Resuscitation

One of the great follies occurring during the treatment of hemorrhagic shock is to perform advanced cardiac life support (ACLS) or cardiopulmonary resuscitation (CPR) for patients where source control is not achievable. There will not be meaningful survival for hemorrhagic shock with the institution of CPR alone; CPR has no role in the definitive treatment of hemorrhagic shock [2]. It is costly, resource intense, and potentially dangerous for healthcare workers when it is used for patients without survivable injury pattern. Until the source of the hemorrhage is controlled and intravascular volume restored after hypovolemic arrest, there is no other effective treatment option.

Emergency Room Thoracotomy

Although residents often consider it a rite of passage to perform the emergency room thoracotomy (ERT), the mature surgeon realizes that the ERT has its place in very few clinical circumstances (Table 1.2) [97]. With only a 2% overall survival rate in blunt trauma, and a 35% survival rate for patients with a single penetrating, quickly controllable injury and no or brief loss of vitals, a selective approach to deciding which patient qualifies for such an invasive maneuver is mandatory. Patients who are found down with no signs of life should not be considered for ERT.

If a patient has spontaneous return of vital signs during the critical maneuvers of the EDT, then transportation to the operating room for more definitive surgical management is appropriate.

REBOA

Resuscitative endovascular balloon occlusion of the aorta, REBOA, has recently emerged as a potential adjunct to resuscitative cases of hemorrhagic shock below the diaphragm [98]. While REBOA is not applied universally, there are case series showing a potential benefit in its use [99]. Trauma surgeons are also engaging in training of endovascular techniques and even completing vascular fellowships, thus minimizing human resources for cases of hemorrhagic shock needing embolization. The REBOA is deployed in a similar fashion to an endovascular balloon via femoral access by cutdown or direct puncture, dilatation. In some examples the aortic occlusion time is decreased compared to thoracic aortic cross clamping and the mean arterial pressure is increased until definitive management can occur. Successful deployment of the balloon also can mitigate pelvic packing and violation of the thoracic cavity. In a recent multi-center study published by Moore and colleagues, REBOA was found to have a higher survival rate as compared to resuscitative thoracotomy for patient with non-compressible torso hemorrhage arising from below the diaphragm [100]. The appropriate patient for REBOA consideration is one with high mortality risk, intra-abdominal exsanguination source, pelvic instability, and sustained systolic pressures lower than 70; in essence, REBOA should be considered in a patient who does not require thoracotomy but who would benefit from aortic occlusion [101].

Thromboelastogram (TEG)

TEG is used to guide decisions in goal-directed resuscitation or correction of coagulopathy or fibrinolysis. TEG is a plotted graph of the effectiveness of clot formation and breakdown, and is considered more accurate to identify causes of coagulopathy in the trauma patient than is a coagulation panel [102, 103]. Several reviews have failed to show a mortality difference in patients who are resuscitated using TEG guidance versus those who follow a standardized massive transfusion protocol; however, the authors note poor power of that aggregated data [104]. They also note that TEG can potentially reduce the amount of transfusions if interpreted and applied during hemorrhagic shock, but that the data on this point is not definitive.

The TEG curves can provide information about all aspects of the clotting system, possibly even the interactions with the endothelium, which is currently an ongoing area of research [105]. The initial part of the TEG, which comprises the R time, or the activated clotting time (ACT), illustrates the amount of time to begin forming a clot (Fig. 1.2). The K time shows how long it takes to reach clot strength and quantitates the clot kinetics [106], whereas the alpha angle and the maximal amplitude (MA) show the rate of clot formation and the absolute clot strength indicating a relationship between fibrinogen and platelets, respectively. A low angle reflects a low fibrinogen concentration; a low MA means that the platelet count or function is reduced and the patient would benefit from platelet transfusion or desmopressin (DDAVP). The LY30 indicates the stability of the clot and the degree of fibrinolysis. The G value shows clot strength or firmness [107].

Normal ACT, R time, and K time indicate that clotting factors are intact and functional. Delays in any of these mean that the patient would most benefit from the administration of FFP or factor; additionally, it can reflect a patient on heparin or other medication that impairs clotting. The angle and MA reflect platelet function and an increase in either suggests hypercoagulable state, whereas a decrease in either means that the platelets may not be aggregating properly. In patients with an elevated MA there is argument for administration of a daily aspirin or placement of an IVC filter [108]. It has been shown that an MA greater than 68 correlates with an increase in coagulability, predisposing patients to thromboembolism [109]. LY30 greater than 3 % has significant consequences of increased mortality and use of antifibrinolytic therapy to target hyperfibrinolysis will be discussed below [110–113].

Damage Control Resuscitation

Damage control resuscitation is a term coined in the military [114, 115]. It is a reproducible strategy with reproducible results and it is automatic and continuous until a physician decides that the shock state has resolved and that hemostasis has been achieved. It describes a resuscitation that uses replacement blood product, rather than crystalloid, for hemorrhagic shock. By limiting the crystalloid infused in the initial resuscitation, patients appear to have less complications and morbidity [116, 117] There are fewer reports of compartment syndromes, a higher number of abdomens that can be closed after a damage control laparotomy, less acidosis, and less electrolyte disturbances.

PROPPR shows that using a 1:1:1 ratio for massive transfusion reduced mortality at 3 h in centers where product was immediately available [118]. By mitigating the onset of coagulopathy within the timeframe of predictable hemorrhagic death, mortality can be prevented. Many centers now utilize a strategy of blood product resuscitation and limitation of crystalloid allocation [119]. For example, Cotton and colleagues investigated the success of the trauma laparotomy when damage control resuscitation in a 1:1:1 ratio and limited crystalloid were implemented. This strategy of damage control resuscitation was found to be useful in the field. Patients in the damage control resuscitation group received

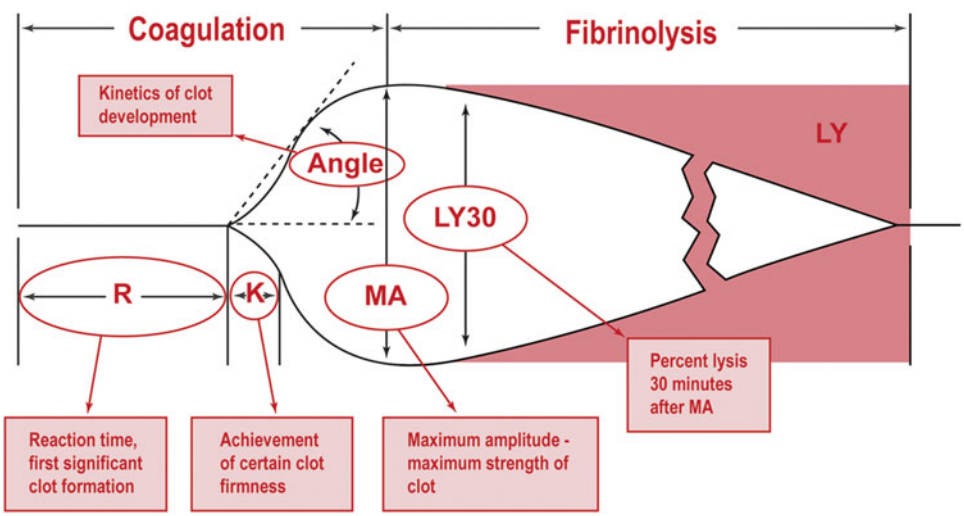

Fig. 1.2 Analytical software graphical representation of a TEG tracing. *R* initial time, *K* time it takes to reach clot strength, *MA* maximal amplitude, *LY* lysis. (From Mark H. Ereth, MD. Uncontrolled bleeding after thoracic aortic aneurysm repair: a case report and interactive discussion. http://www.bloodcmecenter.org, with permission.)

approximately 10 liters less of crystalloid in the first 24 h, had better short- and long-term survival, and showed signs of being less acidodic, less coagulopathic, and less hypothermic on arrival to the ICU than patients who received a traditional resuscitation. The study was a retrospective cohort that examined two similar groups of patients, finding improved morbidity and mortality rates in the group receiving better ratios and colloid. Secondary analyses showed statistically significant differences in multi-organ failure, acute lung and kidney injury, and their effects.

The length of time it takes to get access to FFP plays a role in the success of a massive transfusion protocol. Several studies have examined time factors in receiving product as a way to analyze the effectiveness of a massive transfusion protocol [120–122]. Thawed plasma improved availability and adherence to the 1:1:1 in 12 nationwide trauma centers participating in PROPPR [123]. These data showed an improvement in infusion time interval from 56 min to less than 5 min, which is associated with improved outcomes. Multiple centers are now using never frozen liquid plasma, which has up to 25 day storage at 4 °C [124]. This product combines rapid availability with much longer storage times.

Data is conflicting on the benefit of tranexamic acid (TXA) on outcome in patients with hyperfibrinolysis. While a Cochrane Review in 2012 showed that risk of death in bleeding patients with hypotension is reduced with early TXA, Harvin et al. showed no improvement in 30-day or in-hospital mortality [125, 126]. CRASH-2 showed that TXA was not specifically causative to thromboembolic events. Napolitano et al. review the current data and lingering questions regarding TXA in hemorrhagic shock [127].

Improved overall survival at 30 days and improved hemorrhage related survival without any difference in transfusion requirement was shown in the CRASH 2 trial. The antifibrinolytic was given despite the lack of laboratory data, and when infused more than three h after injury, death was increased. Based on the uncertainty created by the increased death rate, several leading centers have restricted the use of TXA to patients with increased fibrinolysis shown by TEG and presenting early after their traumatic event. Until further randomized data are published this seems like a reasonable approach.

Complications of Resuscitation

Data from the days when trauma patients were resuscitated with multiple liters of saline prior to receiving their first blood product shows complications related to the overwhelming volume of crystalloid infused [128–130]. These types of complications include compartment syndromes, high number of abdomens that cannot be closed, and grossly edematous bowel, all secondary to large volume resuscitation [131]. Complications of transfusion-related acute lung injury (TRALI) and transfusion-associated circulatory overload (TACO) are not seen frequently because the base resuscitative fluid is colloid at relatively lower volumes than a crystalloid based resuscitation [132–134]. Ileus, heart failure, and difficulty with wound healing have all additionally been attributed to over-resuscitation with crystalloid.

All trauma patients who receive a massive resuscitation remain at risk of abdominal compartment syndrome, but this complication appears better mitigated when low ratio of crystalloid to blood product is given. One study claimed that there would be an epidemic if crystalloid resuscitations are continued with such fervor and that patients were threatened by secondary compartment syndrome that occurs solely as the result of excessive crystalloid resuscitation during hemorrhagic shock [135]. Abdominal hypertension is defined as any pressure greater than 12 mmHg without evidence of multi-organ failure. Abdominal compartment syndrome is defined as any one of the following: pressure greater than 20 mmHg; progressive, identifiable organ dysfunction; and improvement following decompression. The trauma population is susceptible, even those who lack abdominal injuries and develop elevated pressures simply due to the amount of fluid they receive. In Houston during the late 1990s the resuscitations during the first 24 h for a group of 128 patients requiring decompression for organ dysfunction averaged the following volumes: (26 ± 2 units PRBC, 38 ± 3 l crystalloid). Seven of these cases required urgent non-abdominal operations, where they likely received several additional units of crystalloid or colloid [136].

It is recommended to check bladder pressures and peak inspiratory pressures routinely and aggressively in patients where massive transfusion has taken place [137]. This practice of serially checking bladder pressures, based on observational data, seems to help in the early identification of intra-abdominal hypertension, perhaps staving off the evolution to abdominal compartment syndrome [138]. Decompression can be done with placement of a temporary dressing and later planned closure with evidence of better results and earlier closure [139, 140].

Keeping the abdomen open after a damage control laparotomy also has its disadvantages. It has been shown that ileus and bowel edema prevent advancement of feeds and definitive closure, and that these phenomena are likely related to an ongoing inflammatory response that occurs as a result of the sustained acute resuscitative phase [141–143]. It is additionally unclear whether ileus is a cause or an effect of bowel edema and vice versa [144, 145]. Administration of 3 % hypertonic saline during the time that the abdomen is open has been shown in a small series to decrease bowel edema. The mechanism is thought to be due to hydrostatic gut edema induced by overaggressive resuscitation with crystalloid. The hypertonic saline gives a smaller volume of more concentrated solution, and pulls extra edematous fluid from the bowel wall. Success has been shown in the rat and subsequently in the human model.

Using Ultrasound to Determine Volume Status

Distinguishing compensated shock from impending complete cardiovascular collapse can be difficult. Understanding physiology and volume status on a global scale seems straightforward—it is the clinical application of these principles to the individual patient that creates a conundrum for identifying the degree of shock. While CVP, monitoring derived from arterial wave forms, intraesophageal echo, urine output and vital signs have all been described for assessment of fluid status, there is also potential benefit in using non-invasive US. Given the application of focused assessment with sonography in trauma (FAST) exam, there has been some interest in examination of the inferior vena cava (IVC) volume during the initial assessment. This is a non-invasive, accurate, and rapid way to assess the patient's overall volume status and is easy to repeat. The technique has been described as placing the patient in the supine position and angling the probe toward the right shoulder from a subcostal view. The IVC can be measured at the entrance of the hepatic veins. Measuring in expiration appears to yield the most accurate measurement. Several small studies demonstrate that measurements of IVC diameter are incredibly fast, non-invasive, accurate measures to determine if shock is present [146–149]. Of note there can be error in measuring the IVC diameter; when accounting for volume variability, the anterior–posterior measurement has been found to be less precise than measurements taken on the oblique axis. In this manuscript the minor axis was defined as the shorter axis when the IVC was viewed as an ellipse shape in horizontal orientation. Trauma patients were included in the study if they were noted to be hypovolemic on the initial ultrasound (minor axis measurement less than 15 mm, consistently measured one cm below the renal vessels) and if they received a computed tomography (CT) scan of their abdomen to further confirm results within 1 h of their diagnosis of hypovolemia. Expected expansion after fluid resuscitation was approximately 7 mm in the minor axis. It remains to be seen if this technique can be widely applied and reliably instituted as a means to identify patients who are volume depleted or dependent and guide resuscitation

Conclusion

Resuscitation is an art and requires attention to detail at all stages including pre-hospital, hospital, operating room, and ICU. The salient points from this chapter focus on understanding shock, providing deficient products, using TEG to guide resuscitation and identifying endpoints. Interested readers are encouraged to focus on several of the resources below to enhance their knowledge and perfect their resuscitation abilities.

References

1. Wiggers HC, Ingraham RC. Hemorrhagic shock: definition and criteria for its diagnosis. J Clin Invest. 1946;25(1):30–6.
2. American College of Surgeons. ATLS manual. 7th ed. Chicago, IL: American College of Surgeons; 2004.
3. Newgard CD, Zive D, Malveau S, Leopold R, Worrall W, Sahni R. Developing a statewide emergency medical services database linked to hospital outcomes: a feasibility study. Prehosp Emerg Care. 2011;15(3):303–19.
4. McGillicuddy DC, O'Connell FJ, Shapiro NI, Calder SA, Mottley LJ, Roberts JC, Sanchez LD. Emergency department abnormal vital sign "triggers" program improves time to therapy. Acad Emerg Med. 2011;18(5):483–7.
5. Mears G, Glickman SW, Moore F, Cairns CB. Data based integration of critical illness and injury patient care from EMS to emergency department to intensive care unit. Curr Opin Crit Care. 2009;15(4):284–9.
6. Holcomb JB, Donathan DP, Cotton BA, Del Junco DJ, Brown G, Wenckstern TV, Podbielski JM, Camp EA, Hobbs R, Bai Y, Brito M, Hartwell E, Duke JR, Wade CE. Prehospital transfusion of plasma and red blood cells in trauma patients. Prehosp Emerg Care. 2015;19(1):1–9.
7. Reynolds PS, Michael MJ, Cochran ED, Wegelin JA, Spiess BD. Prehospital use of plasma in traumatic hemorrhage (The PUPTH Trial): study protocol for a randomised controlled trial. Trials. 2015;16(1):321.
8. Weaver AE, Hunter-Dunn C, Lyon RM, Lockey D, Krogh CL. The effectiveness of a 'Code Red' transfusion request policy initiated by pre-hospital physicians. Injury. 2015;S0020-1383(15)00351-4.
9. Beekley AC, Sebesta JA, Blackbourne LH, Herbert GS, Kauvar DS, Baer DG, Walters TJ, Mullenix PS, Holcomb JB, 31st Combat Support Hospital Research Group. Prehospital tourniquet use in Operation Iraqi Freedom: effect on hemorrhage control and outcomes. J Trauma. 2008;64 Suppl 2:S28–37. Discussion S37.
10. Kragh JF, O'Neill ML, Beebe DF, Fox CJ, Beekley AC, Cain JS, Parsons DL, Mabry RL, Blackbourne LH. Survey of the indications for use of emergency tourniquets. J Spec Oper Med. 2011;11(1):30–8.
11. Taylor DM, Vater GM, Parker PJ. An evaluation of two tourniquet systems for the control of prehospital lower limb hemorrhage. J Trauma. 2011;71(3):591–5.
12. Peck MA, Clouse WD, Cox MW, Bowser AN, Eliason JL, Jenkins DH, Smith DL, Rasmussen TE. The complete management of extremity vascular injury in a local population: a wartime report from the 332nd Expeditionary Medical Group/Air Force Theater Hospital, Balad Air Base, Iraq. J Vasc Surg. 2007;45(6):1197–204. Discussion 1204–5.
13. Barbosa RR, Rowell SE, Diggs BS, Schreiber MA, Trauma Outcomes Group, et al. Profoundly abnormal initial physiologic and biochemical data cannot be used to determine futility in massively transfused trauma patients. J Trauma. 2011;71(2 Suppl 3):S364–9.
14. Barbosa RR, Rowell SE, Sambasivan CN, Diggs BS, Spinella PC, Schreiber MA, Trauma Outcomes Group, et al. A predictive model for mortality in massively transfused trauma patients. J Trauma. 2011;71(2 Suppl 3):S370–4.
15. Nunez TC, Voskresensky IV, Dossett LA, Shinall R, Dutton WD, Cotton BA. Early prediction of massive transfusion in trauma: simple as ABC (assessment of blood consumption)? J Trauma. 2009;66(2):346–52.
16. Cotton BA, Faz G, Hatch QM, Radwan ZA, Podbielski J, Wade C, Kozar RA, Holcomb JB. Rapid thrombelastography delivers real-time results that predict transfusion within 1 hour of admission. J Trauma. 2011;71(2):407–14. Discussion 414–7.
17. Johansson PI, Stensballe J, Oliveri R, Wade CE, Ostrowski SR, Holcomb JB. How I treat patients with massive hemorrhage. Blood. 2014;124(20):3052–8.

18. Borgman MA, Spinella PC, Perkins JG, et al. The ratio of blood products transfused affects mortality in patients receiving massive transfusions at a combat support hospital. J Trauma. 2007;63: 805–13.
19. Holcomb JB, Wade CE, Michalek JE, et al. Increased plasma and platelet to red blood cell ratios improves outcome in 466 massively transfused civilian trauma patients. Ann Surg. 2008;248:447–58.
20. Stansbury LG, Dutton RP, Stein DM, Bochicchio GV, Scalea TM, Hess JR. Controversy in trauma resuscitation: do ratios of plasma to red blood cells matter? Transfus Med Rev. 2009;23(4):255–65.
21. Holcomb JB, Tilley BC, Baraniuk S, et al. Transfusion of plasma, platelets, and red blood cells in a 1:1:1 vs. a 1:1:2 ratio and mortality in patients with severe trauma: the PROPPR randomized clinical trial. JAMA. 2015;313:471–82.
22. Kashuk JL, Moore EE, Sawyer M, Le T, Johnson J, Biffl WL, Cothren CC, Barnett C, Stahel P, Sillman CC, Sauaia A, Banerjee A. Postinjury coagulopathy management: goal directed resuscitation via POC thrombelastography. Ann Surg. 2010;251(4):604–14.
23. Da Luz LT, Nascimento B, Shankarakutty AK, Rizoli S, Adhikari NK. Effect of thromboelastography (TEG®) and rotational thromboelastometry (ROTEM®) on diagnosis of coagulopathy, transfusion guidance and mortality in trauma: descriptive systematic review. Crit Care. 2014;18(5):518.
24. Ho AM, Holcomb JB, Ng CS, Zamora JE, Karmakar MK, Dion PW. The traditional vs "1:1:1" approach debate on massive transfusion in trauma should not be treated as a dichotomy. Am J Emerg Med. 2015;33(10):1501–4.
25. Soller B, Zou F, Dale Prince M, Dubick MA, Sondeen JL. 2014 Military supplement: comparison of noninvasive pH and blood lactate as predictors of mortality in a swine hemorrhagic shock with restricted volume resuscitation model. Shock. 2015;44(1): 90–5.
26. Schmidt BM, Rezende-Neto JB, Andrade MV, Winter PC, Carvalho Jr MG, Lisboa TA, Rizoli SB, Cunha-Melo JR. Permissive hypotension does not reduce regional organ perfusion compared to normotensive resuscitation: animal study with fluorescent microspheres. World J Emerg Surg. 2012;22 (7 Suppl 1):S9.
27. Wilson M, Davis DP, Coimbra R. Diagnosis and monitoring of hemorrhagic shock during the initial resuscitation of multiple trauma patients: a review. J Emerg Med. 2003;24(4):413–22.
28. Ristagno G, Tang W, Sun S, Weil MH. Role of buccal PCO2 in the management of fluid resuscitation during hemorrhagic shock. Crit Care Med. 2006;34(12 Suppl):S442–6.
29. Soller B, Smith C, Zou F, Ellerby GE, Prince MD, Sondeen JL. Investigation of noninvasive muscle pH and oxygen saturation during uncontrolled hemorrhage and resuscitation in swine. Shock. 2014;42(1):44–51.
30. Dunham CM, Siegel JH, Weireter L, Fabian M, Goodarzi S, Guadalupi P, Gettings L, Linberg SE, Vary TC. Oxygen debt and metabolic acidemia as quantitative predictors of mortality and the severity of the ischemic insult in hemorrhagic shock. Crit Care Med. 1991;19(2):231–43.
31. Siegel JH, Fabian M, Smith JA, Kingston EP, Steele KA, Wells MR, et al. Oxygen debt criteria quantify the effectiveness of early partial resuscitation after hypovolemic hemorrhagic shock. J Trauma. 2003;54:862–80.
32. Siegel JH, Rivkind AI, Dala S, Goodarzi S. Early physiologic predictors of injury severity and death in blunt multiple trauma. Arch Surg. 1990;125:498–508.
33. Rixen D, Raum M, Holzgraefe B, Sauerland S, Nagelschmidt M, Neugebauer EA, Shock and Trauma Study Group. A pig hemorrhagic shock model: oxygen debt and metabolic acidemia as indicators of severity. Shock. 2001;16:239–44.
34. Bakker J, Coffernils M, Leon M, Gris P, Vincent JL. Blood lactate levels are superior to oxygen-derived variables in predicting outcome in septic shock. Chest. 1991;99:956–62.
35. Convertino VA, Ryan KL, Rickards CA, Salinas J, McManus JG, Cooke WH, Holcomb JB. Physiological and medical monitoring for en route care of combat casualties. J Trauma. 2008;64 Suppl 4:S342–53.
36. Vary TC, Siegel JH, Rivkind A. Clinical and therapeutic significance of metabolic patterns of lactic acidosis. Perspect Crit Care. 1988;1:85–132.
37. Jansen TC, van Bommel J, Mulder PG, Rommes JH, Schieveld SJ, Bakker J. The prognostic value of blood lactate levels relative to that of vital signs in the pre-hospital setting: a pilot study. Crit Care. 2008;12(6):R160. Epub 17 Dec 2008.
38. Balasubramanyan N, Havens PL, Hoffman GM. Unmeasured anions identified by the Fencl-Stewart method predict mortality better than base excess, anion gap, and lactate in patients in the pediatric intensive care unit. Crit Care Med. 1999;27:1577–81.
39. Smith I, Kumar P, Molloy S, et al. Base excess and lactate as prognostic indicators for patients admitted to intensive care. Intensive Care Med. 2001;27:74–83.
40. Husain FA, Martin MJ, Mullenix PS, et al. Serum lactate and base deficit as predictors of mortality and morbidity. Am J Surg. 2003;185:485–91.
41. Kincaid EH, Miller PR, Meredith JW, et al. Elevated arterial base deficit in trauma patients: a marker of impaired oxygen utilization. J Am Coll Surg. 1998;187:384–92.
42. Rutherford EJ, Morris JA, Reed GW, et al. Base deficit stratifies mortality and determines therapy. J Trauma. 1992;33:417–23.
43. Fidkowski C, Helstrom J. Diagnosing metabolic acidosis in the critically ill: bridging the anion gap, Stewart, and base excess methods. Can J Anaesth. 2009;56(3):247–56. Epub 13 Feb 2009.
44. Rocktaeschel J, Morimatsu H, Uchino S, Bellomo R. Unmeasured anions in critically ill patients: can they predict mortality? Crit Care Med. 2003;31(8):2131–6.
45. Martin MJ, FitzSullivan E, Salim A, Brown CV, Demetriades D, Long W. Discordance between lactate and base deficit in the surgical intensive care unit: which one do you trust? Am J Surg. 2006;191(5):625–30.
46. Lujan E, Howard R. Venous bicarbonate correlates linearly with arterial base deficit only if pH is constant. Arch Surg. 2006;141(1):105. Author reply 105.
47. Schmelzer TM, Perron AD, Thomason MH, Sing RF. A comparison of central venous and arterial base deficit as a predictor of survival in acute trauma. Am J Emerg Med. 2008;26(2):119–23.
48. Rossaint R, Cerny V, Coats TJ, Duranteau J, Fernandez-Mondejar E, Gordini G, Stahel PF, Hunt BJ, Neugebauer E, Spahn DR. Key issues in advanced bleeding care in trauma. Shock. 2006;26:322–31.
49. Hierholzer C, Billiar TR. Molecular mechanisms in the early phase of hemorrhagic shock. Langenbecks Arch Surg. 2001;386:302–8.
50. Hess JR, Brohi K, Dutton RP, Hauser CJ, Holcomb JB, Kluger Y, Mackway-Jones K, Parr MJ, Rizoli SB, Yukioka T, Hoyt DB, Bouillon B. The coagulopathy of trauma: a review of mechanisms. J Trauma. 2008;65(4):748–54.
51. Moore FA, McKinley BA, Moore EE, Nathens AB, West M, Shapiro MB, Bankey P, Freeman B, Harbrecht BG, Johnson JL, Minei JP, Maier RV. Inflammation and the host response to injury, a large-scale collaborative project: patient-oriented research core: standard operating procedures for clinical care. III. Guidelines for shock resuscitation. J Trauma. 2006;61:82–9.
52. Ayala A, Wang P, Ba ZF, Perrin MM, Ertel W, Chaudry IH. Differential alterations in plasma IL-6 and TNF levels following trauma and hemorrhage. Am J Physiol. 1991;260:R167–71.
53. Erber WN, Perry DJ. Plasma and plasma products in the treatment of massive haemorrhage. Best Pract Res Clin Haematol. 2006;19: 97–112.
54. Krause KR, Howells GA, Buhs CL, Hernandez DA, Bair H, Schuster M, Bendick PJ. Hypothermia-induced coagulopathy during hemorrhagic shock. Am Surg. 2000;66(4):348–54.

55. Peitzman AB, Billiar TR, Harbrecht BG, Kelly E, Udekwu AO, Simmons RL. Hemorrhagic shock. Curr Probl Surg. 1995;32: 925–1002.
56. Sharrock AE, Midwinter M. Damage control - trauma care in the first hour and beyond: a clinical review of relevant developments in the field of trauma care. Ann R Coll Surg Engl. 2013;95(3): 177–83.
57. Tsuei BJ, Kearney PA. Hypothermia in the trauma patient. Injury. 2004;35:7–15.
58. Martini WZ, Pusateri AE, Uscilowicz JM, et al. Independent contributions of hypothermia and acidosis to coagulopathy in swine. J Trauma. 2005;58:1002–9. Discussion 1009–1010.
59. Watts DD, Trask A, Soeken K, et al. Hypothermic coagulopathy in trauma: effect of varying levels of hypothermia on enzyme speed, platelet function, and fibrinolytic activity. J Trauma. 1998;44:846–54.
60. Sebesta J. Special lessons learned from Iraq. Surg Clin North Am. 2006;86:711–26.
61. Wu X, Stezoski J, Safar P, Nozari A, Tisherman SA. After spontaneous hypothermia during hemorrhagic shock, continuing mild hypothermia (34 degrees C) improves early but not late survival in rats. J Trauma. 2003;55(2):308–16.
62. Wu X, Kochanek PM, Cochran K, Nozari A, Henchir J, Stezoski SW, Wagner R, Wisniewski S, Tisherman SA. Mild hypothermia improves survival after prolonged, traumatic hemorrhagic shock in pigs. J Trauma. 2005;59(2):291–9. Discussion 299–301.
63. Prueckner S, Safar P, Kentner R, Stezoski J, Tisherman SA. Mild hypothermia increases survival from severe pressure-controlled hemorrhagic shock in rats. J Trauma. 2001;50(2):253–62.
64. Wooten C, Schultz P, Sapida J, Laflamme C. Warming and treatment of mild hypothermia in the trauma resuscitation room—an intervention algorithm. J Trauma Nurs. 2004;11(2):64–6.
65. Ho AM, Dion PW, Yeung JH, et al. Prevalence of survivor bias in observational studies on fresh frozen plasma: erythrocyte ratios in trauma requiring massive transfusion. Anesthesiology. 2012;116(3):716–28.
66. Holcomb JB, Jenkins D, Rhee P, Johannigman J, Mahoney P, Mehta S, Cox ED, Gehrke MJ, Beilman GJ, Schreiber M, Flaherty SF, Grathwohl KW, Spinella PC, Perkins JG, Beekley AC, McMullin NR, Park MS, Gonzalez EA, Wade CE, Dubick MA, Schwab CW, Moore FA, Champion HR, Hoyt DB, Hess JR. Damage control resuscitation: directly addressing the early coagulopathy of trauma. J Trauma. 2007;62(2):307–10.
67. Spinella PC, Holcomb JB. Resuscitation and transfusion principles for traumatic hemorrhagic shock. Blood Rev. 2009;23: 231–40.
68. Stern SA. Low-volume fluid resuscitation for presumed hemorrhagic shock: helpful or harmful? Curr Opin Crit Care. 2001;7(6):422–30.
69. Phillips CR, Vinecore K, Hagg DS, Sawai RS, Differding JA, Watters JM, Schreiber MA. Resuscitation of haemorrhagic shock with normal saline vs. lactated Ringer's: effects on oxygenation, extravascular lung water and haemodynamics. Crit Care. 2009;13(2):R30. Epub 4 Mar 2009.
70. Scultetus A, Alam HB, Stanton K, et al. Dextran and Hespan resuscitation causes neutrophil activation in swine after hemorrhagic shock. Shock. 2000;13(Suppl):52.
71. Rana R, Fernández-Pérez ER, Khan SA, Rana S, Winters JL, Lesnick TG, Moore SB, Gajic O. Transfusion-related acute lung injury and pulmonary edema in critically ill patients: a retrospective study. Transfusion. 2006;46(9):1478–83.
72. Eder AF, Herron R, Strupp A, Dy B, Notari EP, Chambers LA, Dodd RY, Benjamin RJ. Transfusion-related acute lung injury surveillance (2003–2005) and the potential impact of the selective use of plasma from male donors in the American Red Cross. Transfusion. 2007;47(4):599–607.
73. Plurad D, Belzberg H, Schulman I, Green D, Salim A, Inaba K, Rhee P, Demetriades D. Leukoreduction is associated with a decreased incidence of late onset acute respiratory distress syndrome after injury. Am Surg. 2008;74(2):117–23.
74. Todd SR, Malinoski D, Muller PJ, Schreiber MA. Lactated Ringer's is superior to normal saline in the resuscitation of uncontrolled hemorrhagic shock. J Trauma. 2007;62(3):636–9.
75. Kiraly LN, Differding JA, Enomoto TM, Sawai RS, Muller PJ, Diggs B, Tieu BH, Englehart MS, Underwood S, Wiesberg TT, Schreiber MA. Resuscitation with normal saline (NS) vs. lactated ringers (LR) modulates hypercoagulability and leads to increased blood loss in an uncontrolled hemorrhagic shock swine model. J Trauma. 2006;61(1):57–64. Discussion 64–5.
76. Waters JH, Gottlieb A, Schoenwald P, Popovich MJ, Sprung J, Nelson DR. Normal saline versus lactated Ringer's solution for intraoperative fluid management in patients undergoing abdominal aortic aneurysm repair: an outcome study. Anesth Analg. 2001;93(4):817–22.
77. Bickell WH, Bruttig SP, Millnamow GA, O'Benar J, Wade CE. The detrimental effects of intravenous crystalloid after aortotomy in swine. Surgery. 1991;110(3):529–36.
78. Smith CA, Duby JJ, Utter GH, Galante JM, Scherer LA, Schermer CR. Cost-minimization analysis of two fluid products for resuscitation of critically injured trauma patients. Am J Health Syst Pharm. 2014;71:470–5.
79. Shaw AD, Bagshaw SM, Goldstein SL, Scherer LA, Duan M, Schermer CR, Kellum JA. Major complications, mortality, and resource utilization after open abdominal surgery 0.9% saline compared to plasma-lyte. Ann Surg. 2012;255:821–9.
80. Cotton BA, Reddy N, Hatch QM, LeFebvre E, Wade CE, Kozar RA, Gill BS, Albarado R, McNutt MK, Holcomb JB. Damage control resuscitation is associated with a reduction in resuscitation volumes and improvement in survival in 390 damage control laparotomy patients. Ann Surg. 2011;254(4):598–605.
81. Morrison CA, Carrick MM, Norman MA, Scott BG, Welsh FJ, Tsai P, Liscum KR, Wall Jr MJ, Mattox KL. Hypotensive resuscitation strategy reduces transfusion requirements and severe postoperative coagulopathy in trauma patients with hemorrhagic shock: preliminary results of a randomized controlled trial. J Trauma. 2011;70(3):652–63.
82. Skarda DE, Mulier KE, George ME, Bellman GJ. Eight hours of hypotensive versus normotensive resuscitation in a porcine model of controlled hemorrhagic shock. Acad Emerg Med. 2008;15(9):845–52.
83. Bickell WH, Wall Jr MJ, Pepe PE, Martin RR, Ginger VF, Allen MK, Mattox KL. Immediate versus delayed fluid resuscitation for hypotensive patients with penetrating torso injuries. N Engl J Med. 1994;331(17):1105–9.
84. Sondeen JL, Coppes VG, Holcomb JB. Blood pressure at which rebleeding occurs after resuscitation in swine with aortic injury. J Trauma. 2003;54 Suppl 5:S110–7.
85. D'Alleyrand JC, Dutton RP, Pollak AN. Extrapolation of battlefield resuscitative care to the civilian setting. J Surg Orthop Adv. 2010;19(1):62–9.
86. Vassar MJ, Fischer RP, O'Brien PE, Bachulis BL, Chambers JA, Hoyt DB, Holcroft JW, The Multicenter Group for the Study of Hypertonic Saline in Trauma Patients. A multicenter trial for resuscitation of injured patients with 7.5% sodium chloride. The effect of added dextran 70. Arch Surg. 1993;128(9):1003–11. Discussion 1011–3.
87. Vassar MJ, Perry CA, Gannaway WL, Holcroft JW. 7.5% sodium chloride/dextran for resuscitation of trauma patients undergoing helicopter transport. Arch Surg. 1991;126(9):1065–72.
88. Rizoli SB, Rhind SG, Shek PN, Inaba K, Filips D, Tien H, Brenneman F, Rotstein O. The immunomodulatory effects of hypertonic saline resuscitation in patients sustaining traumatic

hemorrhagic shock: a randomized, controlled, double-blinded trial. Ann Surg. 2006;243(1):47–57.
89. Rhind SG, Crnko NT, Baker AJ, Morrison LJ, Shek PN, Scarpelini S, Rizoli SB. Prehospital resuscitation with hypertonic saline-dextran modulates inflammatory, coagulation and endothelial activation marker profiles in severe traumatic brain injured patients. J Neuroinflammation. 2010;7:5.
90. Simma B, Burger R, Falk M, Sacher P, Fanconi S. A prospective, randomized, and controlled study of fluid management in children with severe head injury: lactated Ringer's solution versus hypertonic saline. Crit Care Med. 1998;26(7):1265–70.
91. Radhakrishnan RS, Xue H, Moore-Olufemi SD, Weisbrodt NW, Moore FA, Allen SJ, Laine GA, Cox Jr CS. Hypertonic saline resuscitation prevents hydrostatically induced intestinal edema and ileus. Crit Care Med. 2006;34(6):1713–8.
92. Cox Jr CS, Radhakrishnan R, Villarrubia L, Xue H, Uray K, Gill BS, Stewart RH, Laine GA. Hypertonic saline modulation of intestinal tissue stress and fluid balance. Shock. 2008;29(5): 598–602.
93. Sheikh AA, Matsuoka T, Wisner DH. Cerebral effects of resuscitation with hypertonic saline and a new low-sodium hypertonic fluid in hemorrhagic shock and head injury. Crit Care Med. 1996;24(7):1226–32.
94. Bulger EM, Jurkovich GJ, Nathens AB, Copass MK, Hanson S, Cooper C, et al. Hypertonic resuscitation of hypovolemic shock after blunt trauma: a randomized controlled trial. Arch Surg. 2008;143(2):139–48. Discussion 149.
95. Bulger EM, May S, Kerby JD, Emerson S, Stiell IG, Schreiber MA, ROC Investigators, et al. Out-of-hospital hypertonic resuscitation after traumatic hypovolemic shock: a randomized, placebo controlled trial. Ann Surg. 2011;253(3):431–41.
96. Riha GM, Kunio NR, Van PY, Hamilton GJ, Anderson R, Differding JA, Schreiber MA. Hextend and 7.5% hypertonic saline with dextran are equivalent to lactated ringer's in a swine model of initial resuscitation of uncontrolled hemorrhagic shock. J Trauma. 2011;71(6):1755–60.
97. Cothren CC, Moore EE. Emergency department thoracotomy for the critically injured patient: objectives, indications, and outcomes. World J Emerg Surg. 2006;1:4.
98. Brenner M. REBOA and catheter-based technology in trauma. J Trauma Acute Care Surg. 2015;79(1):174–5.
99. Brenner ML, Moore LJ, DuBose JJ, Tyson GH, McNutt MK, Albarado RP, et al. A clinical series of resuscitative endovascular balloon occlusion of the aorta for hemorrhage control and resuscitation. J Trauma Acute Care Surg. 2013;75(3):506–11.
100. Moore LJ, Brenner ML, Kozar RA, Pasley J, Wade CE, Baraniuk MS, Scalea T, Holcomb JB. Implementation of resuscitative endovascular balloon occlusion of the aorta as an alternative to resuscitative thoracotomy for noncompressible truncal hemorrhage. J Trauma Acute Care Surg. 2015;79(4):523–32.
101. Saito N, Matsumoto H, Yagi T, Hara Y, Hayashida K, Motomura T, Mashiko K, Iida H, Yokota H, Wagatsuma Y. Evaluation of the safety and feasibility of resuscitative endovascular balloon occlusion of the aorta. J Trauma Acute Care Surg. 2015;78(5):897–903. Discussion 904.
102. Johansson PI, Stissing T, Bochsen L, et al. Thrombelastography (TEG) in trauma. Scand J Trauma Resusc Emerg Med. 2009; 17:45.
103. Plotkin AJ, Wade CE, Jenkins DH, et al. A reduction in clot formation rate and strength assessed by thrombelastography is indicative of transfusion requirements in patients with penetrating injuries. J Trauma. 2008;64:64–8.
104. Afshari A, Wikkelsø A, Brok J, Møller AM, Wetterslev J. Thrombelastography (TEG) or thromboelastometry (ROTEM) to monitor haemotherapy versus usual care in patients with massive transfusion. Cochrane Database Syst Rev. 2011;16(3), CD007871.
105. Johansson PI, Ostrowski SR. Acute coagulopathy of trauma: balancing progressive catecholamine induced endothelial activation and damage by fluid phase anticoagulation. Med Hypotheses. 2010;75(6):564–7. Epub 13 Aug 2010.
106. Reikvam H, Steien E, Hauge B, Liseth K, Gjerde Hagen K, Størkson R, et al. Thrombelastography. Transfus Apher Sci. 2009;40:119–23.
107. Stammers AH, Bruda NL, Gonano C, Hartmann T. Point-of-care coagulation monitoring: applications of the thromboelastography. Anaesthesia. 1998;53 Suppl 2:58–9.
108. Cotton B, Minei K, Radwan ZA, Matijevic N, Pivalizza E, Podbielski J, et al. Admission rapid thrombeolastography (r-TEG) predicts development of pulmonary embolism in trauma patients. J Trauma Acute Care Surg. 2012;72(6):1470–5.
109. McCrath DJ, Cerboni E, Frumento RJ, et al. Thromboelastography maximum amplitude predicts postoperative thrombotic complications including myocardial infarction. Anesth Analg. 2005;100: 1576–83.
110. CRASH-2 Collaborators, Roberts I, Shakur H, Afolabi A, Brohi K, Coats T, et al. The importance of early treatment with tranexamic acid in bleeding trauma patients: an exploratory analysis of the CRASH-2 randomised controlled trial. Lancet. 2011;377(9771):1096–101.
111. Henry DA, Carless PA, Moxey AJ, et al. Antifibrinolytic use for minimizing perioperative allogenic blood transfusion. Cochrane Database Syst Rev. 2011;3, CD001886.
112. Morrison JJ, Dubose JJ, Rasmussen TE, Midwinter MJ. Military application of tranexamic acid in trauma emergency resuscitation (MATTERs) study. Arch Surg. 2012;147(2):113–9.
113. CRASH-2 Trial Collaborators, Shakur H, Roberts I, Bautista R, Caballero J, Coats T, et al. Effects of tranexamic acid on death, vascular occlusive events, and blood transfusion in trauma patients with significant hemorrhage (CRASH-2): a randomized, placebo controlled trial. Lancet. 2010;376(9734):23–32.
114. Hess JR, Holcomb JB, Hoyt DB. Damage control resuscitation: the need for specific blood products to treat the coagulopathy of trauma. Transfusion. 2006;46:685–6.
115. Holcomb JB. Damage control resuscitation. J Trauma. 2007;62: S36–7.
116. Duchesne JC, Kimonis K, Marr AB, Rennie KV, Wahl G, Wells JE, et al. Damage control resuscitation in combination with damage control laparotomy: a survival advantage. J Trauma. 2010;69(1):46–52.
117. Duchesne JC, Barbeau JM, Islam TM, Wahl G, Greiffenstein P, McSwain Jr NE. Damage control resuscitation: from emergency department to the operating room. Am Surg. 2011;77(2):201–6.
118. Hess JR, Holcomb JB. Resuscitating PROPPRly. Transfusion. 2015;55(6):1362–4.
119. Duchesne JC, Hunt JP, Wahl G, Marr AB, Wang YZ, Weintraub SE, Wright MJ, McSwain Jr NE. Review of current blood transfusions strategies in a mature level I trauma center: were we wrong for the last 60 years? J Trauma. 2008;65(2):272–6. Discussion 276–8.
120. Ho AM, Karmakar MK, Dion PW. Are we giving enough coagulation factors during major trauma resuscitation? Am J Surg. 2005;190:479–84.
121. Dente CJ, Shaz BH, Nicholas JM, et al. Improvements in early mortality and coagulopathy are sustained better in patients with blunt trauma after institution of a massive transfusion protocol in a civilian Level I trauma center. J Trauma. 2009;66:1616–24.
122. Zink KA, Sambasivan CN, Holcomb JB, et al. A high ratio of plasma and platelets to packed red blood cells in the first 6 hours of massive transfusion improves outcomes in a large multi center study. Am J Surg. 2009;197:565–70. Discussion 570.
123. Novak DJ, Bai Y, Cooke RK, et al. Making thawed universal donor plasma available rapidly for massively bleeding trauma patients: the experience of the Pragmatic, Randomized Optimal Platelets and Plasma Ratios (PROPPR) trial centers. Transfusion. 2015;55(6):1331–9.

124. Cao Y, Dua A, Matijevic N, Wang Y-W, Pati S, Wade CE, Ko TC, Holcomb JB. Never-frozen liquid plasma blocks endothelial permeability as effectively as thawed fresh frozen plasma. J Trauma Acute Care Surg. 2014;77(1):28–33.
125. Roberts I, Shakur H, Ker K, Coats T, CRASH-2 Trial Collaborators. Antifibrinolytic drugs for acute traumatic injury. Cochrane Database Syst Rev. 2012;12, CD004896.
126. Harvin JA, Peirce CA, Mims MM, Hudson JA, Podbielski JM, Wade CE, Holcomb JB, Cotton BA. The impact of tranexamic acid on mortality in injured patients with hyperfibrinolysis. J Trauma Acute Care Surg. 2015;78(5):905–9. Discussion 909–11.
127. Napolitano LM, Cohen MJ, Cotton BA, Schreiber MA, Moore EE. Tranexamic acid in trauma: how should we use it? J Trauma Acute Care Surg. 2013;74(6):1575–86.
128. Coimbra R, Porcides R, Loomis W, Melbostad H, Lall R, Deree J, Wolf P, Hoyt DB. HSPTX protects against hemorrhagic shock resuscitation-induced tissue injury: an attractive alternative to Ringer's lactate. J Trauma. 2006;60(1):41–51.
129. Hatoum OA, Bashenko Y, Hirsh M, Krausz MM. Continuous fluid resuscitation for treatment of uncontrolled hemorrhagic shock following massive splenic injury in rats. Shock. 2002;18(6):574–9.
130. Solomonov E, Hirsh M, Yahiya A, Krausz MM. The effect of vigorous fluid resuscitation in uncontrolled hemorrhagic shock after massive splenic injury. Crit Care Med. 2000;28(3):749–54.
131. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Valdivia A, Sailors RM, Moore FA. Supranormal trauma resuscitation causes more cases of abdominal compartment syndrome. Arch Surg. 2003;138(6):637–42. Discussion 642–3.
132. Popovsky MA. Pulmonary consequences of transfusion: TRALI and TACO. Transfus Apher Sci. 2006;34(3):243–4. Epub 26 July 2006.
133. Gajic O, Gropper MA, Hubmayr RD. Pulmonary edema after transfusion: how to differentiate transfusion-associated circulatory overload from transfusion-related acute lung injury. Crit Care Med. 2006;34 Suppl 5:S109–13.
134. Haupt MT, Teerapong P, Green D, Schaeffer Jr RC, Carlson RW. Increased pulmonary edema with crystalloid compared to colloid resuscitation of shock associated with increased vascular permeability. Circ Shock. 1984;12(3):213–24.
135. Balogh Z, Moore FA, Moore EE, Biffl WL. Secondary abdominal compartment syndrome: a potential threat for all trauma clinicians. Injury. 2007;38(3):272–9. Epub 15 Nov 2006.
136. Balogh Z, McKinley BA, Cocanour CS, Kozar RA, Holcomb JB, Ware DN, Moore FA. Secondary abdominal compartment syndrome is an elusive early complication of traumatic shock resuscitation. Am J Surg. 2002;184(6):538–43. Discussion 543–4.
137. Maxwell RA, Fabian TC, Croce MA, Davis KA. Secondary abdominal compartment syndrome: an underappreciated manifestation of severe hemorrhagic shock. J Trauma. 1999;47(6):995–9.
138. Sugrue M, Buhkari Y. Intra-abdominal pressure and abdominal compartment syndrome in acute general surgery. World J Surg. 2009;33(6):1123–7.
139. Hatch QM, Osterhout LM, Ashraf A, Podbielski J, Kozar RA, Wade CE, et al. Current use of damage-control laparotomy, closure rates, and predictors of early fascial closure at the first takeback. J Trauma. 2011;70(6):1429–36.
140. van Boele HP, Wind J, Dijkgraaf MG, Busch OR, Carel GJ. Temporary closure of the open abdomen: a systematic review on delayed primary fascial closure in patients with an open abdomen. World J Surg. 2009;33(2):199–207.
141. Bueno L, Ferre JP, Ruckebusch Y. Effects of anesthesia and surgical procedures on intestinal myoelectric activity in rats. Dig Dis. 1978;23:690–5.
142. Livingston EH, Passaro Jr EP. Postoperative ileus. Dig Dis Sci. 1990;35:121–32.
143. Bauer AJ, Schwarz NT, Moore BA, Türler A, Kalff JC. Ileus in critical illness: mechanisms and management. Curr Opin Crit Care. 2002;8(2):152–7.
144. Balogh Z, McKinley BA, Cox Jr CS, et al. Abdominal compartment syndrome: the cause or effect of postinjury multiple organ failure. Shock. 2003;20:483.
145. Drake RE, Teague RA, Gabel JC. Lymphatic drainage reduces intestinal edema and fluid loss. Lymphology. 1998;31:68.
146. Lyon M, Blaivas M, Brannam L. Sonographic measurement of the inferior vena cava as a marker of blood loss. Am J Emerg Med. 2005;23(1):45–50.
147. Yanagawa Y, Sakamoto T, Okada Y. Hypovolemic shock evaluated by sonographic measurement of the inferior vena cava during resuscitation in trauma patients. J Trauma. 2007;63(6):1245–8. Discussion 1248.
148. Stawicki SP, Braslow BM, Panebianco NL, Kirkpatrick JN, Gracias VH, Hayden GE, Dean AJ. Intensivist use of hand-carried ultrasonography to measure IVC collapsibility in estimating intravascular volume status: correlations with CVP. J Am Coll Surg. 2009;209(1):55–61. Epub 1 May 2009.
149. Murphy EH, Arko FR, Trimmer CK, Phangureh VS, Fogarty TJ, Zarins CK. Volume associated dynamic geometry and spatial orientation of the inferior vena cava. J Vasc Surg. 2009;50(4):835–42. Discussion 842–3; Epub 6 Aug 2009.

The Evaluation of the Acute Abdomen

2

Marie Crandall

Introduction and Epidemiology

Abdominal pain is one of the most common reasons for visits to the emergency room, comprising 7 % of all visits [1]. Although for the majority of patients, symptoms are benign and self-limited, a subset will be diagnosed with an "acute abdomen," as a result of serious intra-abdominal pathology necessitating emergency intervention [2].

An expeditious workup and Epidemiology is necessary when evaluating patients presenting with acute abdominal pain to determine the most likely cause of their symptoms and determine whether or not emergent operative intervention is necessary. The most appropriate therapy should then be initiated with the patient's clinical status optimized. The workup should first include a thorough but efficient acquisition of the patient's history and physical examination followed by the judicious use of laboratory and radiologic studies. The evaluation of patients with acute abdominal pain can pose a diagnostic challenge for physicians as patients may present with atypical symptoms that interfere with the usual pattern recognition that often guides decision making. These atypical presentations may help account for the over 25 % of abdominal pain cases labeled as "nonspecific" or "undifferentiated" [2].

Additionally, physicians must take into account the patient's age, gender, and comorbidities as conditions associated with the acute abdomen may vary accordingly. Specifically, gastroenteritis, acute appendicitis, and abdominal trauma are common causes of the acute abdomen in children and young adults [3], whereas biliary disease, intestinal obstruction, diverticulitis, and appendicitis are among the most common causes in middle-aged adults and the elderly [4]. Furthermore, pelvic pathology accounts for approximately 12 % of acute abdominal pain presentations and should therefore be considered when evaluating female patients [2].

Finally, there are a variety of nonsurgical causes of abdominal pain that are cardiovascular, metabolic, and toxic in origin that should be considered when evaluating these patients.

Clinical Presentation

A thorough, yet expeditiously obtained, history and physical exam is paramount to developing the differential diagnosis for patients presenting with an acute abdomen. Various laboratory and imaging studies may subsequently be used as adjuncts to help guide decision making.

History

When obtaining a patient history, the physician should avoid questions that are leading and should focus on details of the pain. This includes information on the onset, character, duration, and location of pain as well as the presence of radiation of pain.

Regarding onset, pain that develops suddenly may be suggestive of a perforated viscus or ruptured abdominal aortic aneurysm (AAA). Pain that gradually worsens over time may be the result of conditions characterized by the progressive development of infection and inflammation such as acute appendicitis and cholecystitis.

With regard to character, pain described as "burning" may implicate the pain of a perforated peptic ulcer while a "ripping" or "tearing" sensation typically represents the pain of an aortic dissection. Pain that is intermittent or colicky should be distinguished from pain that is continuous in nature. Colicky pain is typically associated with obstructive processes of the intestinal, hepatobiliary, or genitourinary tract, while pain that is continuous is usually the result of

M. Crandall (✉)
Department of Surgery, University of Florida College of Medicine, 655 W. 8th Street, Jacksonville, FL 32209, USA
e-mail: marie.crandall@jax.ufl.edu

L.J. Moore, S.R. Todd (eds.), *Common Problems in Acute Care Surgery*, DOI 10.1007/978-3-319-42792-8_2

underlying ischemia or peritoneal inflammation. The latter may occur primarily or following an initial episode of colicky pain when an obstructive process is complicated by the development of ischemia. Examples of this include cases of biliary colic that progresses to acute cholecystitis or an incarcerated loop of intestine that becomes strangulated and ischemic.

The location of pain is important to consider as various pathologic conditions tend to occur in specific regions or quadrants of the abdomen (Fig. 2.1a, b). Therefore, if the physician is knowledgeable of the disease processes that cause pain in these areas, they may be able to significantly narrow down their differential. This holds true for those with the understanding that certain conditions may result in pain that radiates or is referred to an area beyond the site of disease due to shared innervation. Classic examples of this include biliary pain that is referred to the right subscapular region, the pain of acute pancreatitis that radiates to the back, and genitourinary pain that radiates from the flank down to the groin. Finally, it is important to note any chronological variation in the pain as this may provide helpful clues to the diagnosis. One of the best examples of this is in the case of acute appendicitis, in which pain is initially perceived in the periumbilical region before localizing to the right lower quadrant (RLQ). This phenomenon reflects the transition from visceral to parietal pain as appendiceal inflammation progresses to involve and irritate the peritoneal lining.

The majority of patients presenting with acute abdominal pain have associating symptoms (e.g., nausea, vomiting, diarrhea, constipation, hematochezia) that are often helpful in making a diagnosis. Chronology of nausea is important to consider as vomiting that occurs after the onset of abdominal pain is more likely to be surgical in nature as a result of medullary vomiting centers that are stimulated by pain impulses traveling via secondary visceral afferent fibers. Additionally, constipation or obstipation may point towards an intestinal obstruction, while diarrhea (especially if bloody) is associated with gastroenteritis, inflammatory bowel disease, and intestinal ischemia.

Aggravating or alleviating factors may also provide diagnostic clues. Depending on the underlying etiology, patients may maintain certain positions to help alleviate their pain. For example, patients with peritonitis may find some relief when lying still with their knees bent, while patients suffering from a bout of acute pancreatitis prefer to sit upright and lean forward. The effect of food is also important to consider as eating may alleviate the pain of a peptic ulcer while worsening the pain of an intestinal obstruction, acute cholecystitis, or acute pancreatitis [5, 6].

The patient's past medical and surgical histories may also help to narrow down the differential. A remote history of abdominal surgery may indicate that intestinal obstruction secondary to adhesive disease is the source of a patient's complaints. Furthermore, it is important to consider the impact that coexistent medical conditions, such as diabetes, chronic obstructive pulmonary disease, and atherosclerosis, may have on patient outcomes. The fact that elderly patients are more likely to have significant comorbidities places them at increased risk for end organ damage incited by gastrointestinal emergencies [7].

Physicians should also take into account the effects of medication use. Anticoagulants may predispose to the development of rectus sheath hematomas and precipitate the gastrointestinal bleeding that is a component of the patient's underlying illness or complicating the patient's postoperative or posttreatment course. Chronic use of nonsteroidal anti-inflammatory drugs (NSAIDs) may also promote bleeding episodes along with the development of peptic ulcer disease (PUD) and its complications.

A detailed social history should also be obtained to determine if there is any significant history of tobacco, alcohol, or illicit drug use, as such behaviors can be a source of the patient's symptoms as well as complicate the patient's hospital course. Notably, a history of cocaine abuse may point towards a diagnosis of mesenteric ischemia as the underlying reason for the patient's symptoms.

The social history should consist of a detailed gynecologic history, including the date of the last menses, the presence of any vaginal bleeding or discharge, and any history of unprotected sexual activity or intercourse with multiple partners. Such information could indicate pregnancy complications, salpingitis or pelvic inflammatory disease, and other gynecologic conditions as the cause of the patient's acute abdominal complaints. Physicians should also take note of any history of recent travel to implicate infectious enterocolitis. Any exposure to environmental toxins should be determined, as lead and iron poisoning are two well-known, extra-abdominal sources of acute abdominal pain [5, 6].

Finally, the patient's family history may ascertain whether a patient's symptoms are hereditary in origin, as seen in the case of inherited hypercoagulable states, which can cause acute mesenteric ischemia secondary to mesenteric venous thrombosis.

Physical Examination

Examination of the patient presenting with acute abdominal pain should initially begin with overall appearance of the patient and vital signs. Patients who appear diaphoretic, pale, and anxious often suffer from a condition of vascular origin, including dissecting AAA, mesenteric ischemia, or atypical angina. The patient who is lying particularly still on the exam table often has peritonitis from perforated viscus or pancreatitis. Vital signs should always be interpreted knowing the status of the patient's pain, or the influence of any home