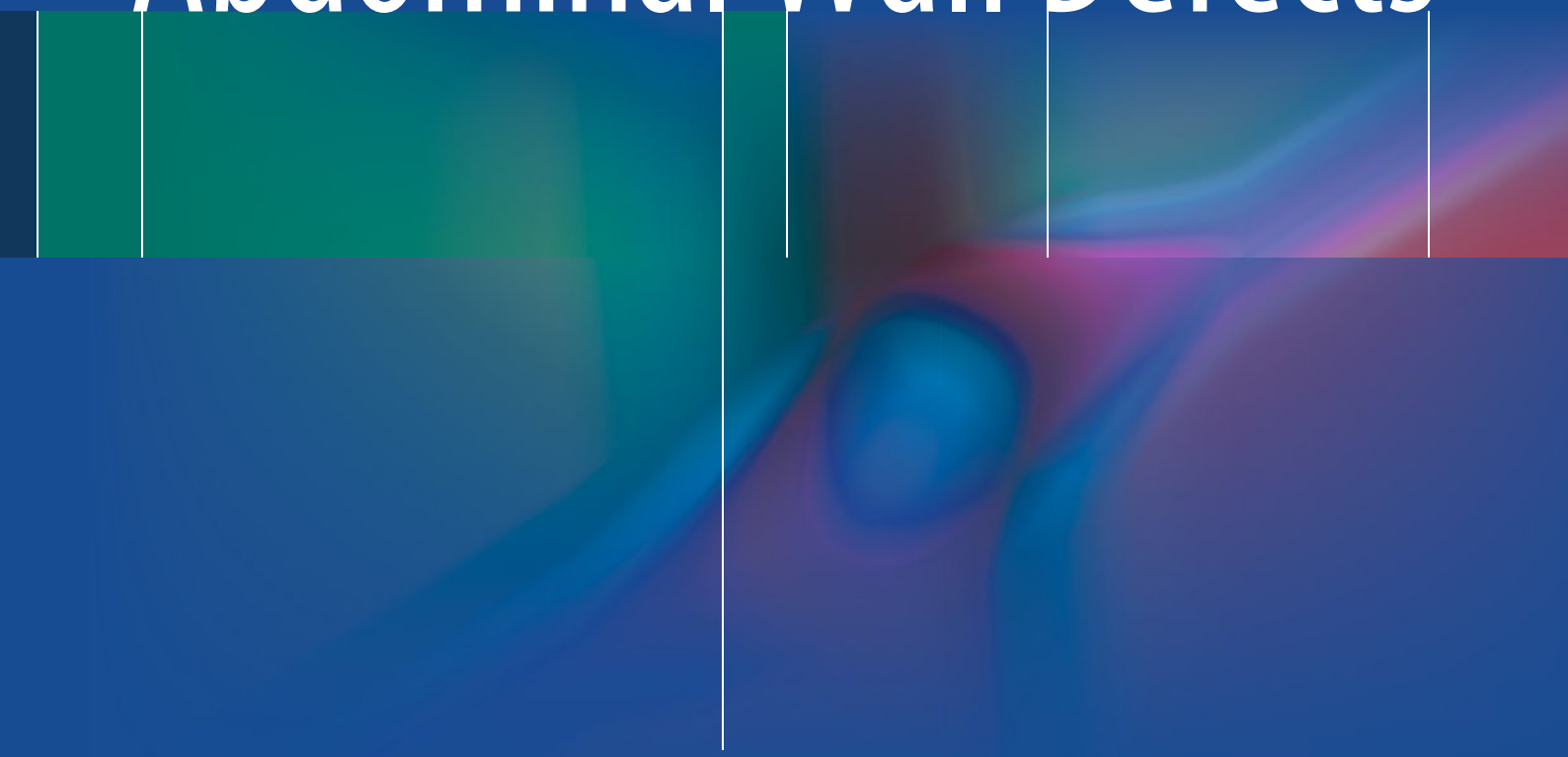
Rifat Latifi
Editor

Surgery of Complex Abdominal Wall Defects



Springer

Rifat Latifi
Editor

Surgery of Complex Abdominal Wall Defects

Springer

Editor
Rifat Latifi, MD, FACS
Department of Surgery
The University of Arizona
Tucson, Arizona
USA

Trauma Section, Department of Surgery
Weill Cornell Medical College
Hamad General Hospital
Doha, Qatar

ISBN 978-1-4614-6353-5 ISBN 978-1-4614-6354-2 (eBook)
DOI 10.1007/978-1-4614-6354-2
Springer New York Heidelberg Dordrecht London

Library of Congress Control Number: 2013939780

Printed on acid-free paper

Springer is part of Springer Science+Business Media (www.springer.com)

To Dr. Stanley J. Dudrick, the father of modern surgical nutritional support, my mentor, teacher, and friend, who taught me reoperative complex surgery and how to care for most complex and difficult surgical patients, and whose extraordinary personal attributes and professional, educational, and scientific contributions will endure into future generations of surgeons all over the world.

Foreword

A surgeon can do more for the community by operating on hernia cases and seeing that his recurrence rate is low than he can by operating on cases of malignant disease.

Sir Cecil Wakely, 1948, president of the Royal College of Surgeon

Oh, if only it were that simple! Certainly, Sir Wakely was referring to inguinal and perhaps umbilical hernias in this well-known quotation from the middle of the last century. I wonder if at the time he could have imagined the true complexity of the problem: our limited understanding of the dynamic physiology of abdominal wall tension and the need for more refined surgical techniques to manage abdominal wall defects. Perhaps most shocking of all to Sir Wakely might have been the ability for patients to withstand and survive catastrophic illness resulting in complex abdominal wall defects. Even as recently as 25 years ago, it was hard to believe that a patient who had lost integrity of the abdominal wall as a result of injury, abdominal sepsis, or gastrointestinal failure could even survive, let alone return to functional status. However, with the evolution of resuscitation, operation, and surgical critical care for patients with devastating abdominal injury and illness, a high survival rate is now a reality. With this, our ability to manage the attendant complications, including complex abdominal wall defects with and without intestinal fistula, has improved dramatically. This has happened because the clinical circumstances have demanded it, and our zeal to improve care is no less ardent than that of Sir Wakely over a half century ago.

The editor, Dr. Rifat Latifi, and contributors to this work have produced what I believe is the quintessential and seminal resource on this vexing and challenging topic. *Surgery of Complex Abdominal Wall Defects* is the first textbook of its kind to provide a comprehensive review of modern management of abdominal wall problems. It eloquently reviews the anatomy and physiology of the abdominal wall and the pathophysiology of abdominal wall defects. It provides a valuable history of abdominal wall repair and then systematically provides the latest approach to operative repair, including preoperative preparation, acute management of the open abdomen, the approach to the hostile abdomen in the intermediate term, critical strategies in long-term reconstruction, and the full spectrum of special circumstances that arise along the way. Nowhere will you find a more comprehensive and practical guide for the management of these patients. If nothing else, this text provides the fundamental context in which these problems will be discussed and in which future advances are made.

I commend the authors on this accomplishment, and I encourage the readers to pay close attention to the content. Herein lies the state-of-the-art surgical management for patients with complex abdominal wall defects. Sir Wakely would be proud to know how far the art and science of the approach to these patients have come.

Greenville, NC, USA — Michael F. Rotondo, MD, FACS

Disclosure

Dr. Rifat Latifi is a speaker for LifeCell Corporation and has received a grant through International Virtual e-Hospital Foundation, a not-for-profit organization that made possible administrative support and secretarial help for this book.

Instead of Prologue

Eight Reasons This Book Is in Your Hands

When I conceived the idea to put together this book, I was fully cognizant of the huge task ahead of me. But, the biggest motive was that this book will help us as surgeons take better care of our patients. Finalizing this book has been a great, albeit difficult journey. Many times during this process, I have asked myself these questions: Why another book? Will this one make a significant contribution? Will it change patient care for the better? Do practicing surgeons need this book to take care of patients with complex surgical problems?

Obviously, I decided in the end that this book would indeed help interested surgeons in this subfield. And now, seeing it complete, I do think it will add to our knowledge and improve our practice. I hope that you, the reader, will find a positive answer to these questions as well.

Here are the main reasons that drove me to produce this book that you now hold in your hands.

Reason 1: Surgeons' Need

Admittedly, a number of well-written textbooks focus on hernias, a number of great surgical textbooks touch on abdominal wall reconstruction, and a number of books deal with surgical complications. However, in all my years of taking care of seriously ill patients with complex abdominal wall defects (with or without associated fistulas, stomas, and loss of abdominal wall domain), I have not been able to find a real reference textbook that reflects the latest advances in biologic and synthetic meshes, especially when we deal with open abdomen and abdominal wall reconstruction. In my surgical practice—initially in Richmond, Virginia, and now, for nearly a decade, at the University of Arizona—I have longed for such a book to keep on my desk and refer to daily, something written by actual practicing surgeons for actual practicing surgeons.

I hope that my collaborators and I have now filled this gap. This was my main motive for taking on this project. As an editor of this book, I have read every word in this book and have carefully looked over every illustration and every figure. Every line represents a patient or a group of patients, offering practical evidence of bona fide surgical opinions and treatments. Real-world know-how is the power of this book, helping us to truly help patients with complex abdominal wall defects, patients who often see us as their last chance.

Reason 2: Patients' Need

Patients with complex abdominal wall defects are not eligible for same-day surgery; they are not among those who can undergo an operation in the morning and then go home in the afternoon—not at all. In fact, far from it. Such patients will be in the hospital for a long time postoperatively; most of them have already been with us for a long time, having survived a number of previous operations. Most of them have battled, for months or even years, the consequences of major trauma or the abdominal catastrophes, cancer, or necrotizing infections that left them without an abdominal wall (a part of the anatomy that we all take for granted until we lose it).

This monstrous defect, or set of defects, results in a foul-smelling odor most of the time; it severely limits patients' ability to work, to exercise, to have a sex life, and even to be in public. So, the need to know how to take care of these patients is enormous; as we continue to make progress in medicine and surgery, this need will be even bigger.

Reason 3: Need to Share Knowledge and the Existing Expertise

I asked some of the best practicing surgeons in the world who deal almost daily with this problem to help me put this book together. The topic is not a simple one, just as it is not a simple endeavor to take care of patients with complex abdominal wall defects. I asked the contributors to say something new, something that they think will help other practicing surgeons help their patients. We are not discussing small umbilical hernias, but rather giant abdominal defects that are often associated with fistulas, stomas, obesity, and the lack of an abdominal wall. These defects pose enormous problems for patients and surgeons alike. Specific medical and physiologic expertise, complicated surgical interventions, and a well-coordinated team approach are required. In each of our chapters, we share what we have learned, with an emphasis on current principles and practices and an eye toward new strategies.

Reason 4: Frequency of Abdominal Wall Defects

Currently, complex abdominal wall defects are more common than in the past: a larger number of patients are surviving serious injuries and intra-abdominal catastrophes, thus living longer with significant comorbidities. As surgeons, we have made significant progress—in terms of technology, knowledge, and skills—in caring for patients with open abdomens. Often, the end result is a patient who has survived an initial insult and now has an open abdomen, with a temporary cover, that requires delayed reconstruction of an abdominal wall defect; a giant ventral hernia; or in the worst-case scenario, a frozen abdomen with enteric fistulas. Preventing or managing complications is of utmost importance.

Reason 5: Complexity of Most Abdominal Wall Defects

When complex abdominal wall defects are associated with fistulas, the complexity increases significantly. A strategic operative plan is imperative, ideally using a multidisciplinary approach. Those of us who treat such patients know firsthand that the more operations an individual undergoes, the more complications potential complications can develop. However, at some point, we as surgeons must make a decision and perform what we hope will be that individual's "final" surgery, the one that will definitively complete the abdominal wall reconstruction and return them to normal life.

Reason 6: Three Principles of Surgical Care

Before definitive surgical intervention, the cornerstone goal is to prevent, or at least to treat successfully, the well-recognized characteristic sequelae of fistulas and complex abdominal defects (such as sepsis, malnutrition, and fluid and electrolyte disturbances), muscle wasting, and overall stamina. This goal has not changed significantly since the advances in nutritional support, promulgated by Dr. Stanley Dudrick in the 1960s.

In our Focus Issue "Current Management of Enterocutaneous Fistulas," published in the *European Journal of Trauma and Emergency Surgery* (2011) and in the International Association for Trauma Surgery and Intensive Care (ATSIC) symposium "Management of Abdominal Defects and Enterocutaneous Fistulas in the Era of Biologic Mesh," published in *World Journal of Surgery* (2012), we summarized the need for three new treatment modalities for these complex patients: first, complete nutrition and metabolic support using TPN (total parenteral nutrition) or enteral nutrition for as long as it takes; second, application of complex surgical techniques to provide skin coverage through tissue transfer techniques and biological mesh; and third, the use, in both inpatients and outpatients, of wound VAC (vacuum-assisted closure) [1, 2]. These three modalities have now become part of our armamentarium for caring for patients with complex abdominal wall defects, including those with stomas or fistulas.

Reason 7: New Technologies

The explosion in new proposed strategies and meshes, as a result of recent strides in technology and biomedical research, has made available for today's surgeons choices unheard of in previous generations. Sometimes, though, all these choices are confusing, if not overwhelming. As surgeons, we need to evaluate each new technological "miracle" painstakingly in the light of the research presented, much of it in the form of case series rather than large, randomized, double-blind studies that yield level I evidence.

In particular, one type of industry is on the rise, namely, the business of creating biologic mesh, be it from human sources or from different animals. This industry promotes the use of novel meshes and prostheses, each company claiming that its products are better than the competitors'. Given the significant comorbidities of most patients with complex abdominal wall defects, biologic meshes are nearly their only alternative, especially when wound infections are present or probable. The ability of certain biologic prostheses to support revascularization and to become part of human tissue is a major advance, adding a new dimension to surgical repair.

Fortunately, the use of advanced surgical techniques and biologic materials may reduce the risk of recurrence of abdominal wall defects and the risk of surgical site infections. Biologic mesh that is both human and porcine in origin is especially useful in high-risk patients. Acellular dermal matrix (ADM) provides an advantage over the nonbiologic materials used as an adjunct to hernia repairs in that ADM allows implantation in infected fields. Of concern, however, is that no method of ADM use in abdominal wall reconstructions has been standardized, despite its daily use by a number of surgeons worldwide.

Reason 8: Need for a Multidisciplinary Approach

Our rule is to try to prevent major abdominal defects and to close the abdomen as early as possible. But, even when we succeed in doing so, patients then need long-term care, including abdominal wall reconstruction. In recent years, we have come to realize the importance of a multidisciplinary team as we try to prevent or control sepsis, manage any imbalance in fluids and electrolytes, provide specialized nutritional support (both parenterally or enterally), protect the skin, define the patient's individual anatomy, and plan the appropriate surgical intervention. No single surgeon, irrespective of the type of practice (whether private, academic, or group) can adequately take care of such patients alone. The surgeon is and should be the team leader, and he or she should direct the treatment, but many other clinicians also have a crucial role.

References

1. Latifi R, Turegano F, guest editors. Focus on current management of enterocutaneous fistulas. Eur J Emerg Surg 2011;37:207–67.
2. Latifi R, Leppaniemi A, guest editors. IATSIC symposium. Management of complex abdominal wall defects and enterocutaneous fistulae in the era of biological mesh. World J Surg 2012;36:495–538.

Tuscon, AZ, USA Rifat Latifi, MD, FACS

Acknowledgments

As with any other book, no single author or editor can take the entire credit. This book is a result of the hard work of so many people, and the credit should go to all of them. For the last year and a half, I have been working every day with the many wonderful contributors to this book from around the world as well as with Liz Corra of Springer's editorial office. I would like to express my deep gratitude to all of them.

Special thanks to my children, Kalterina, Qendresa, Kushtrim, Fortesa, and Lulejeta, and my wife, Drita, for all of their loving support, which makes it all worth it.

Contents

Contributors

Hassan Al Thani, MD Trauma Section, Department of Surgery, Hamad General Hospital, Doha, Qatar

Rahul J. Anand, MD, FACS Division of Trauma, Critical Care, and Emergency Surgery, Department of Surgery, Virginia Commonwealth University Medical Center, Richmond, VA, USA

Eva Barbosa, MD, MSc Department of Anatomy, ICBAS - University of Porto, Vila do Conde, Portugal

Department of Surgery: Colo-Rectal and Abdominal Wall Surgery, Hospital Pedro Hispano-Matosinhos, Sra da Hora, Portugal

Donald P. Baumann, MD Department of Plastic Surgery, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Charles E. Butler, MD, FACS Department of Plastic Surgery, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Albert Chi, MD Department of Surgery, Johns Hopkins Hospital, Baltimore, MD, USA

Michael Ditillo, DO Department of Surgery, Yale School of Medicine, New Haven, CT, USA

Stanley J. Dudrick, MD, FACS Department of Surgery, Yale University School of Medicine, Naugatuck, CT, USA

Fernando Ferreira, MD Department of Surgery, Upper Gastrointestinal and Abdominal Wall Surgery, Hospital Pedro Hispano, Sra da Hora, Portugal

Department of Medicine - University of Porto, Praça de Gomes Teixeira Porto, Portugal

Gustavo Pereira Fraga, MD, PhD Division of Trauma Surgery, Department of Surgery, University of Campinas, Campinas, SP, Brazil

Rainer W.G. Gruessner, MD, FACS Department of Surgery, The University of Arizona, Tucson, AZ, USA

Fahim Habib, MD, FACS Division of Trauma Surgery, Broward Health Medical Center, Lauderdale, FL, USA

Guillermo Higa, MD Department of Surgery, Willamette Valley Medical Center, McMinnville, OR, USA

Rao R. Ivatury, MD, FACS Division of Trauma, Critical Care, and Emergency General Surgery, Virginia Commonwealth University, Richmond, VA, USA

Bellal Joseph, MD Department of Surgery, Division of Trauma, Critical Care, and Emergency Surgery, University of Arizona/University Medical Center, Tucson, AZ, USA

Shigeki Kushimoto, MD, PhD Division of Emergency Medicine, Tohoku University Graduate School of Medicine, Sendai, Japan

Rifat Latifi, MD, FACS Department of Surgery, The University of Arizona, Tucson, AZ, USA

Trauma Section, Department of Surgery, Weill Cornell Medical College, Hamad General Hospital, Doha, Qatar

Ari Leppäniemi, MD, PhD Department of Abdominal Surgery, Meilahti Hospital, University of Helsinki, Helsinki, Finland

Department of Emergency Surgery, Meilahti hospital, University of Helsinki, Helsinki, Finland

David Mark Levi, MD Department of Surgery, Carolinas Medical Center, Charlotte, NC, USA

Ajai K. Malhotra, MBBS (MD), MS, FRCS, DNB, FACS Division of Trauma, Critical Care, and Emergency Surgery, Department of Surgery, Medical College of Virginia, Virginia Commonwealth University, Richmond, VA, USA

Andrés García-Marín, MD Department of General Surgery and Digestive Surgery, University Hospital San Juan de Alicante, Alicante, Spain

Antonio C. Marttos Jr., MD Department of Surgery, University of Miami, Miami, FL, USA

Ronald Merrell, MD Department of Surgery, Virginia Commonwealth University, Richmond, VA, USA

Maurice Y. Nahabedian, MD Department of Plastic Surgery, Georgetown University, Washington, DC, USA

Mayur Narayan, MD, MPH, MBA Program in Trauma, R Adams Cowley Shock Trauma Center, University of Maryland School of Medicine, Baltimore, MD, USA

Bartolomeu Nascimento, MD, MSc, CTBS Department of Surgery, University of Toronto/Sunnybrook Health Sciences Center, Toronto, ON, Canada

Alexander T. Nguyen, MD Department of Plastic Surgery, The University of Texas MD Anderson Cancer Center, Houston, TX, USA

Ruben Peralta, MD, FACS Trauma Section, Department of Surgery, Hamad General Hospital, Doha, Qatar

Bruno Monteiro T. Pereira, MD, MSC Division of Trauma Surgery, Department of Surgery, University of Campinas, Campinas, SP, Brazil

Jose M. Pimiento, MD Department of Surgery, Michigan State College of Human Medicine Lacks Cancer Center/Saint Mary's Health Care, Grand Rapids, MI, USA

Marcos Campos W. Reis, MD, TCBC Department of General Surgery, Baleia Hospital, Faculdade de Sociologia e Ecologia Humana (FASEH), Belo Horizonte, Brazil

Peter Rhee, MD, MPH, FACS, FCCM Division of Trauma, Critical Care, Burns and Emergency Surgery, Department of Surgery, The University of Arizona/University of Arizona Medical Center, Tucson, AZ, USA

Department of Surgery, The University of Arizona, Tucson, AZ, USA

Eduardo D. Rodriguez, MD, DDS Department of Plastic, Reconstructive, and Maxillofacial Surgery, University of Maryland School of Medicine, Baltimore, MD, USA

Thomas M. Scalea, MD Program in Trauma, R Adams Cowley Shock Trauma Center, University of Maryland School of Medicine, Baltimore, MD, USA

Gennaro Selvaggi, MD Department of Clinical Surgery, University of Miami, Miami, FL, USA

Department of Liver and GI Transplant, University of Miami, Miami, FL, USA

Fernando Turégano, MD, PhD Department of General Surgery and Emergency Surgery, University General Hospital Gregorio Marañón, Madrid, Spain

Andreas G. Tzakis, MD, PhD, Dhe Mult Division of Liver Transplant, Transplant Department, Cleveland Clinic Florida, Miami, FL, USA

Selman Uranues, MD Department of Surgery, Medical University of Graz/University Hospital, Graz, Austria

James F. Whelan, MD, FACS Division of Trauma, Critical Care, and Emergency General Surgery, Department of Surgery, Virginia Commonwealth University Medical Center, Richmond, VA, USA

Intraoperative Decision-Making Process: The Art and the Science

1

Rifat Latifi, Rainer W.G. Gruessner, and Peter Rhee

Introduction

How do we as surgeons make intraoperative decisions under what can be inauspicious conditions? That question has not been answered appropriately in the literature [1], despite a few attempts [2]. When a patient is dying from bleeding that we cannot control, when irreversible metabolic shock does not respond to anything that we do, when new problems emerge unexpectedly, when things go alarmingly wrong—in such dire moments during a carefully planned operation, how do we decide what to do next? Many surgeons make such decisions on the basis of "a gut feeling" or "intuition" or the "gray hair effect," among other techniques. In this chapter, we review theoretical as well as objective data that we as surgeons use to make intraoperative decisions. Most of the many theories and hypotheses in the literature have been created by individuals who are not surgeons. But, our collective firsthand experience as surgeons points to a combination of factors contributing to our intraoperative decision-making process, including education, clinical expertise, mentoring, and the creativity and excellence that come with long practice and with surgical strict discipline.

R. Latifi, MD, FACS (✉)
Department of Surgery, The University of Arizona,
1501 N. Campbell Ave, Tucson, AZ 85724, USA

Trauma Section, Department of Surgery,
Weill Cornell Medical College,
Hamad General Hospital, Doha, Qatar
e-mail: latifi@surgery.arizona.edu

R.W.G. Gruessner, MD, FACS
Department of Surgery, The University of Arizona,
1501 N. Campbell Ave, Tucson, AZ 85724, USA
e-mail: rguessner@surgery.arizona.edu

P. Rhee, MD, MPH, FACS, FCCM
Department of Surgery, The University of Arizona,
1501 N. Campbell Ave, Tucson, AZ 85724, USA

Division of Trauma, Critical Care,
Burns and Emergency Surgery, Department of Surgery,
The University of Arizona/
University of Arizona Medical Center,
Tucson, AZ, USA
e-mail: prhee@surgery.arizona.edu

The Anatomy of Surgeons' Intraoperative Decisions

Naturalistic and complex problem-solving theories [3] attempt to explain how high-risk professionals make decisions, but such theories lump surgeons with other high-risk professionals whose decisions demand superb accuracy, such as pilots and nuclear plant scientists. Indeed, it has become fashionable to compare pilots with surgeons. However, there are distinct differences between these professions. Pilots have in their hands the most sophisticated machines ever created by humans, but the pilots are backed by powerful computers and, frequently, have full support from the base on the ground. Although surgeons have a team with them in every operation, they themselves make the most important decisions; they are in charge of carrying out the procedures that may either save or kill the patient at hand. Although once in perfect condition, the human machine on the operating table might be in grave condition and may not respond to any intervention.

So, surgeons have to rely on their own experience and knowledge, on their grasp of the patient's clinical information, and, occasionally, on their assistants' help. A dynamic stepwise model of surgeons' intraoperative decision-making process involves monitoring and assessing the situation, taking appropriate actions, and reevaluating the patient's response [1, 2]. That model encompasses components such as intuition (also known as "recognition-primed decision-making" analytical ability) and creativity [4]. Nowhere is that model more applicable than in complex reoperative surgical procedures, which are often associated with an array of unanticipated problems. In our opinion, an important theoretical component that has not received sufficient attention and is beyond technical abilities [5] is the surgeon's leadership ability. Adroitly taking charge of a calamitous, often hopeless,

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_1,

situation—applying proper technical skills, assigning different team members to different tasks, and communicating in a timely, clear, and calm manner—can make a significant difference. In fraught intraoperative situations, few surgeons have reported that they make decisions through analytical, rational heuristics or through trial and error [6, 7]. Rather, studies among surgeons have shown that the basis of surgical decision-making process is primarily task visualization, communication, and the mental state of the surgeon, that is, on what is called a mental model [8]. Other critical factors influencing intraoperative surgical decision making have been described [9–11]. In addition to the surgeon's leadership ability and mental state, creativity might be the most important element of all. Historically, surgeons have shown solid creativity. It often has changed the way we practice medicine and surgery, defying the anatomy and physiology of the body and buying at least some time. The ideal virtues of any surgeon should include open-mindedness and flexibility. While respecting sound surgical principles, the surgeon must be ready to adapt to any new intraoperative challenge at any time. Creativity in the service of excellence does not come easily, however. It takes dedication. It takes a lifetime of continuously studying the art and science of surgery.

Intraoperative Endpoints of Resuscitation

Complex theoretical discussions, though intellectually and perhaps scientifically important, need to be backed by objective data. Intraoperatively, the surgical patient should be resuscitated in the same manner as any trauma patient. Adequate oxygen delivery is mandatory, as is maintenance of normal tissue perfusion and of adequate body temperature. Fluid status should be monitored. Hypotension should be avoided, especially if the patient underwent bowel preparation. Rigorous intraoperative assessment of the patient's status mandates the use of one or more global or regional endpoint of resuscitation. Standard hemodynamic parameters do not adequately reflect physiologic disturbances and do not accurately assess biochemical and cellular status [12, 13]. Arterial lactate, arterial base deficit, and gastric pHi have limitations, yet these endpoints of resuscitation predict development of multiple-organ failure and should be used to guide intraoperative care and the extent of surgery. Depending on the institution's setup, other endpoints might be used, such as oxygen delivery and mixed venous oxygen saturation, tissue oxygen and transcutaneous O_2 and CO_2, and near-infrared spectroscopy. If the patient becomes cold, coagulopathic, or acidotic, then surgery should be abbreviated and damage control performed. The patient should be resuscitated and warmed in the intensive care unit (ICU). In summary, in terms of objective data, the most important surgical decision-making signpost is complete and continuous awareness of the patient's physiology and anatomy (or distorted anatomy, in the case of reoperations).

Damage Control on Demand

Despite all the preoperative planning, extensive discussion with the patient and family, signed informed consent forms, time-out, and other preventive measures that we currently take for things to go right, things can go wrong, plans can change, and surgery can take longer than expected. Surgeons may need to consider stopping the surgery and returning the next day (or even later) to complete an anastomosis or to reconstruct the abdominal wall. The need to abbreviate a laparotomy might be the result of either the surgeon's or the patient's physiology. We call this necessary break "damage control on demand" [14, 15]. If definitive closure of the abdomen is impossible or ill advised at this time, the surgeon should implement a plan (ideally, a plan made preoperatively) for temporarily covering the viscera. During the interim period, until further surgery, the patient can be fully resuscitated, any coagulation problems or acidosis can be corrected, and the surgeon and surgical team can obtain some necessary rest. The surgery can be completed a few days later during the same hospitalization or at a later date.

Staged Operations

In reoperative intestinal surgery, especially if reestablishing the continuity of the GI tract is expected, surgeons should not promise patients that they will not have a stoma, temporary or otherwise. If the integrity of an anastomosis is questionable, it is reasonable to revise it. Or, a proximal diverting ostomy can be created, especially with two or fewer anastomoses or with an anastomosis deep in the pelvis [16]. If diversion is performed, a loop-diverting stoma is preferred to avoid entering the abdomen.

For the last few decades, to treat the most severely injured and physiologically compromised patients, the concept of damage control surgery—e.g., an abbreviated laparotomy followed later by a planned reoperation—has been accepted as a new paradigm [17]. Damage control surgery has been increasingly used in patients with nontraumatic abdominal emergencies, such as severe acute pancreatitis or secondary peritonitis [18–20].

In addition, increased intra-abdominal pressure, especially abdominal compartment syndrome, are now recognized as conditions requiring active monitoring and sometimes surgical decompression; as a result, the number of patients with an open abdomen with a temporary cover over the viscera has increased [21]. Open abdomen, also known as laparostomy, still has potentially severe consequences, even though

it is a lifesaving intervention [22]. Such patients often require delayed reconstruction of abdominal wall defects or of giant ventral hernias; the worst-case scenario is a frozen or hostile abdomen [23] with enteral fistulas.

Temporary Closure

With damage control on demand, the surgeon has several options for temporary closure of the abdomen, most notably an intestinal bag, wound vacuum-assisted closure (VAC), or a moist gauze that serves as the "poor person's wound VAC" [24]. However, if the patient has enough skin and subcutaneous tissue, then closing the skin offers the best temporary closure. Temporary closure of the fascia should be avoided for fear of injuring the edges of the fascia and subsequently creating a hernia and dehiscence. If wound VAC is used, just enough pressure should be applied to maintain closure; pressures higher than 70 mmHg must clearly be avoided, especially for long periods. High pressures have been associated with creation of new fistulas in patients with an open abdomen. If at all possible, final and definitive closure of the abdomen should be performed within 12–24 h after temporary closure. The final and definitive closure type is discussed in other chapters (see Chaps. 9, 10 and 11). Different techniques for abdominal wall reconstruction are described elsewhere in this book (see Chaps. 7, 9 and 16).

Summary

The intraoperative decision-making process can be difficult. It draws on the surgeon's education, clinical experience, leadership ability, mental state, physiology, and creativity, as well as objective data from the patient's physiology and anatomy. Flexibility and an open-minded approach, along with a respect for sound surgical principles, are important. Accommodating the physiology of both the patient and the surgeon is imperative. Still, most intraoperative decisions are made "on the fly" and are hard to theorize, quantify, or categorize. Additional work, especially from and on surgeons themselves, is needed to delineate further how we make life-changing intraoperative decisions.

References

1. Flin R, Youngson G, Yule S. How do surgeons make intraoperative decisions? Qual Saf Health Care. 2007;16(3):235–9.
2. Pauley K, Flin R, Yule S, Youngson G. Surgeons' intraoperative decision making and risk management. Am J Surg. 2011;202:375–81.
3. Orasnu J, Fischer U. Finding decisions in natural environments: the view from the cockpit. In: Zsambok C, Klein G, editors. Naturalistic decision making. Mahwah: Lawrence Erlbaum; 1997.
4. Klein G. A recognition-primed decision making (RPD) model of rapid decision making. In: Klein G, Orasanu J, Calderwood R, Zsambock C, editors. Decision making in action. New York: Ablex; 1993.
5. Bolotin G, Kypson A, Nifang W, et al. A technique for evaluating competitive flow for intraoperative decision making in coronary heart surgery. Ann Thorac Surg. 2003;76:2118–20.
6. Velanovich V. Operative decisions. Theor Surg. 1991;638–40.
7. Aziz F, Khalil A, Hall J. Evolution of trends in risk management. ANZ J Surg. 2005;75:603–7.
8. Czyzeweska E, Kicka K, Czarnecki A, et al. The surgeon's mental load during decision making at various stages of operations. Eur J Appl Physiol Occup Physiol. 1983;51:441–6.
9. Jalote-Parmar A, Badke-Schaub P. Critical factors influencing intra-operative surgical decision-making. International conference on systems, man and cybernetics SMC. 2008;1091–6.
10. Jalote-Parmar A, Badke-Schaub P. Workflow integration matrix: a framework to support the development of surgical information systems. Des Stud. 2008;29(4):338–68.
11. Crosskerry P. The theory and practice of clinical decision-making. Can J Anaesth. 2005;52:R18.
12. Tisherman SA, Barie P, Bokhari F, et al. Clinical practice guideline: endpoints of resuscitation. J Trauma. 2004;57:898–912.
13. Velhamos G, Demetriades D, Shoemaker W, et al. Endpoints of resuscitation of critically injured patients: normal or Supranormal? A prospective randomized trial. Ann Surg. 2000;232:409–18.
14. Latifi R, Leppaniemi A. Complex abdominal wall defects and enterocutaneous fistulae in the era of biological mesh: did we make any real progress? World J Surg. 2012;36(3):495–6.
15. Latifi R, Joseph B, Kulvatunyou N, Wynne JL, O'Keeffe T, Tang A, et al. Enterocutaneous fistulas and a hostile abdomen: reoperative surgical approaches. World J Surg. 2012;36(3):516–23.
16. Latifi R, Gustafson M. Abdominal wall reconstruction in patients with enterocutaneous fistulas. Eur J Trauma Emerg Surg. 2011;37:241–50.
17. Rotondo MF, Schwab CW, McGonigal MD, Phillips GR, Fruchterman TM, Kauder DR, et al. "Damage control": an approach for improved survival with exsanguinating penetrating abdominal injury. J Trauma. 1993;35(3):375–82.
18. Diaz Jr J, Cullinane DC, Dutton DW, et al. The management of the open abdomen in trauma and emergency general surgery: part 1—damage control. J Trauma. 2010;68:1425–38.
19. Midwinter MJ. Damage control surgery in the era of damage control resuscitation. J R Army Med Corps. 2009;155:323–6.
20. Smith BP, Adams RC, Dorasiwamy VA, et al. Review of abdominal damage control and open abdomens: focus on gastrointestinal complications. J Gastrointestin Liver Dis. 2010;19(4):425–35.
21. Stawiicki SP, Cipolla J, Bria C, et al. Comparison of open abdomens in non-trauma and trauma patients: a retrospective study. OPUS 12 Sci. 2007;1:1–8.
22. Scott BG, Feanny MA, Hirshberg A. Early definitive closure of the open abdomen: a quiet revolution. Scand J Surg. 2005;94:9–14.
23. Leppaniemi A. The hostile abdomen—a systematic approach to a complex problem. Scand J Surg. 2008;7:218–9.
24. Erdmann D, Drye C, Heller L, et al. Abdominal wall defect and enterocutaneous fistula treatment with the vacuum-assisted closure (VAC) system. Plast Reconstr Surg. 2001;108(7):2066–8.

History of Abdominal Wall Repair: In Search of New Techniques and Materials

2

Ronald Merrell

Introduction

The abdominal wall was not meant to be violated. An early description of defect closure came from Plutarch in his description of the suicide of Cato the Younger in 46 BCE. Cato, the stoic, had thrown in his lot with Pompeius Magnus against the imperial Caesar, and all had turned out badly for him. After the death of Pompey and the defeat of forces in Utica, Cato decided to end his life by sword to the abdomen. He was successful in opening the abdominal wall and apparently fainted. A brave physician was called, who recognized that the situation might be remedied by surgical skill and daring. "The physician … put in his bowels, which were not pierced, and sewed up the wound." This was successful, but when Cato regained consciousness and realized his global failure had even extended to his own suicide, he ripped open the wound and tore out the intestine, dying promptly as he always intended but now as a surgical complication [1].

Surgeons have struggled with the daunting task of restoring the abdominal wall despite its nature, their patients' intent, and personal inadequacy. There were many attempts at laparotomy that, despite best intentions, ended in peritonitis and death. The first success was that of Ephraim McDowell in 1809. In Danville, Kentucky, he removed an ovarian tumor without benefit of anesthesia or antisepsis. He was clean in his habits, which may explain why this procedure was followed by a 33-year survival for his patient [2]. Throughout the nineteenth century, there were many bold efforts at operating in the abdomen, and the successful reports did not seem to include any problem with healing of the abdominal wound.

R. Merrell, MD
Department of Surgery,
Virginia Commonwealth University,
Richmond, VA, USA
e-mail: rcmerrel@vcu.edu

Early Reports in the Annals of Surgery

The great prospect of laparotomy, with some caveats, was declared in the *Annals of Surgery* in 1886 [3]. Reports were duly made to the American Surgical Association for pistol shot [4], gunshot [5], and splenectomy [6], and all successes were reported without failure of the abdominal wall. Dixon reported concerning an appendectomy for purulent perforation and in the same issue reported a laparotomy for strangulating hernia [7, 8]. Not only could the pristine abdomen be treated but also potentially septic pathology could be managed. Reports of ventral hernia after laparotomy were slow to come. The first report in the *Annals of Surgery* was in 1901 from Eads [9]. The early problem was considered that of great difficulty, and the reports of hernia were notably lacking in the bravura of earlier reports of successful laparotomy. When the surgeon was confronted with massive protrusion of abdominal contents, which could be seen writhing in peristalsis just beneath the thin skin, there was a strong urge to repair the problem. Many of these efforts followed in the twentieth century and today.

The persistence in innovation for repair of the anterior abdominal wall strongly suggests that there is no good way to repair the problem even now. The current incidence of incisional hernia may be as high as 11 % across the board. One might implicate a poor effort at the closure of the original laparotomy. However, the surgeon who undertakes to remedy the earlier mess is rewarded with a 33 % likelihood of hernia recurrence. The second or third effort at repair of abdominal wall defects is associated with an even higher likelihood of recurrence [10].

The options to repair include movement of local tissue into a configuration that will restore wall integrity. This approach was the mainstay for most of the history of laparotomy. However, the inadequacy of this approach in general led to a relentless search for autologous, allogeneic biological material or prosthetic materials for over a century.

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_2,

Prosthetic Materials

In 1947, Koontz (Baltimore, MD) reported at the Southern Surgical Association on his work with tantalum gauze. Tantalum was an interesting choice based on the tragic story of the element's namesake, the mythological Tantalus, who was condemned to stand in a pool of water he could never reach to drink. Certainly, the identification of a prosthetic material to slake the knowledge thirst of frustrated surgeons did not end with this material; it continues to the present. Koontz had previously reported the use of preserved ox fascia but now moved on to a relatively inert metal that could span the defect of an abdominal hernia. Experimental repair preceded his clinical application, and he was insightful in recognizing that the strength of his experimental repair was because of the infiltration explained by the structural strength of the repair and not the mesh itself. Furthermore, he described the use of the material as a full replacement of the defect as well as an overlay for tenuous fascia approximation. He described the need to overlap the fascia and the material with generous sutures [11]. Koontz also described the desirability to divide the rectus fascia vertically to provide the abdominal fascia mobility in seeking midline union. His work followed a half century of difficult work with silver mesh. That material was not only antiseptic but also irritating, eventually dissolving.

The local tissue approach to reapproximate the anterior abdominal wall continued as an evolving challenge with a seminal development by Albanese in the 1950s and popularized by Ramirez in 1990. He described the elevation of subcutaneous flaps far lateral of the midline and the division of the external oblique fascia. This plane also became undermined, and the rectus fascia was divided just posterior to the midline to allow advancement of the rectus. This dissection and fascial division allowed advancement perhaps 10 cm to the side to provide a generous coverage of even huge hernias while relying on the redundancy of the abdominal wall layers to ensure structural integrity [12]. Regional flaps—such as tensor fascia lata, latissimus dorsi, and free flaps—have been applied for specific needs, but these are generally proposed for initial repair of large defects created in the resection of abdominal wall tumors.

Finding the Perfect Mesh

There has been great interest in finding a polymer that would approximate collagen in strength, durability, and flexibility. Such a polymer has not been found. In this search, a reasonable set of criteria was proposed by Cumberland [13] and Scales [14] in 1952 and 1953, respectively. They proposed eight characteristics for an ideal mesh; the ideal mesh should be noncarcinogenic, chemically inert, resistant to mechanical strain, suitable for sterilization, biologically inert, nonallergenic, limited foreign body tissue reaction, and amenable to production in useful form for surgery. Polypropylene was first synthesized in its crystalline isotactic form in 1954. Commercial production began in 1957, and Usher described the first use of polypropylene mesh for hernia in 1959 [15, 16]. The mesh was marvelously flexible, durable, and strong. It also harbored bacteria in its many interstices; an infection could flare many years after implantation. Undesirably, the material not only incorporated the invading fibrous tissue but also engendered adhesions to the intestine and created a prospective intestinal obstruction. Polyester has similar qualities.

Because of adhesions and infections, new expectations were placed on the ideal mesh. It would be desirable if the mesh resisted infection, presented a nonadherent face to the abdominal cavity, and could respond biologically in a manner similar to autologous tissue [17]. A review of prosthetics by Shankaran et al. was superb, timely, and scholarly.

Nonabsorbable Mesh

The polymer meshes include polypropylene (Prolene®, Ethicon, Somerville, NJ; Marlex®, C.R. Bard, Murray Hill, NJ); lightweight polypropylene (Vypro®, Ethicon; ProLite™, Atrium, Hudson, NH); polyester (Dacron, Mersilene™, Ethicon); and expanded polytetrafluorethylene (ePTFE, GoreTex®, W.L. Gore & Associates, Newark, Delaware). They differ by pore size; ePTFE has smallest and therefore has the least likelihood to harbor bacteria. They differ in tensile strength, but all exceed the necessary strength. They are of similar thickness. They differ in varying degrees in postoperative pain syndromes, and there are varying reports of recurrence. Generally, after a mesh repair of an incisional hernia, there is a recurrence rate of 2–30 % compared to open/primary repair failures of 18–62 % [18–22]. None of the polymer meshes achieve the ideals listed previously. A large number of coated or composite meshes have been introduced to address needs. Mesh has been coated with bioabsorbable but initially active agents such as poliglecaprone (Ultrapro®, Ethicon); carboxy-methylcellulose–Seprafilm on polypropylene (called Sepramesh™, C.R. Bard); omega-3 fatty acids (C-Qur™, Atrium); cellulose (Proceed®, Ethicon); and collagen-polyethylene-glycerol on polyester (Parietex™, Covidien, Dublin, Ireland). Each has great proponents and detractors, but a definitive advantage is not obvious. The mesh has been made double sided to address the special issue of reactivity next to the bowel on the peritoneal side. Lightweight or heavyweight polypropylene on ePTFE (Composix™, C.R. Bard) is dual sided, and there is dual-sided ePTFE with a different surface, resulting in a nonporous material (DualMesh®, W.L. Gore) as need proposed. The chemistry of the mesh occupied most of the discussion and progress in

understanding and treating massive abdominal trauma in the latter part of the twentieth century. The technique regarding placement of the mesh relative to the abdominal viscera has continued to add fuel to the debate, and the truth is still out there somewhere [23–27].

Absorbable Mesh

Absorbable mesh has also been considered in order to provide a temporary matrix and strength, with subsequent replacement with natural tissue. Polyglycolic acid (Dexon™, Covidien) and polygalactin (Vicryl®, Ethicon) had been used, but problems with failure to control infection and high recurrence rates have dimmed enthusiasm except in severe circumstances of sepsis for which a temporary barrier is all that is required [28].

Laparoscopic Repair

In 1982, laparoscopy was applied to ventral hernia for the first time with internal closure of a hernia sac [29]; a full description and result were published in 1993 by Le Blanc and Booth [30]. Full anatomic reconstruction of the abdominal wall by laparoscopy has been a growing trend because of its decreased injury and quicker return to function [31]. The data have been subject to the improved database registry of the American College of Surgeons National Surgical Quality Improvement Program [32]. Despite lower overall morbidity with laparoscopic technique, this 2011 report only accounted for 17 % of the procedures in a registry of over 71,000 ventral herniorrhaphies for the years 2005–2009. Laparoscopy for massive abdominal wall defects is considered difficult because of alternate entry ports, adhesions, and the disorientation of the surgeon confronted with terribly distorted anatomy. Comparison of open versus laparoscopic procedures examined ten randomized controlled trials in the Cochrane Database [33]. A general review of the dramatic progress in herniorrhaphy was published by Gray et al. in 2008 [34].

Conclusion

The next level of endeavor for the thousands of disabled patients threatened by abdominal hernia probably lies with improved skills in laparoscopy. Most likely, materials science is not going to offer the next frontier in hernia repair. The possibility of tissue engineering manufacturing a truly comparable dynamic tissue to substitute for the abdominal wall should be anticipated, however. Further improvement in results will certainly come from agreement on proper surgical indications, eliminating high-risk patients from the tally. Finally, better understanding of the biology of the marvelous structure, function, and plasticity of the abdomen may offer sound and new principles in the initial repair of this essential barrier to prevent such a prevalent and almost always iatrogenic scourge.

References

1. Plutarch's parallel lives. http://penelope.uchicago.edu/Thayer/E/Roman/Texts/Plutarch/Lives/home.html. Accessed 1 Jan 2012.
2. Othersen HB. Ephraim McDowell: the qualities of a good surgeon. Ann Surg. 2004;239:648–50.
3. Colles CJ. On laparotomy in cases of perforation of the stomach and intestines (editorial). Ann Surg. 1886;3:286–397.
4. Skelly JI. Laparotomy for perforating pistol-shot wound of the abdomen—recovery. Ann Surg. 1887;6:49–51.
5. Dalton HC. Gunshot wound of the stomach and liver treated by laparotomy and suture of visceral wounds, with recovery. Ann Surg. 1888;8:81–100.
6. Park R. A case of splenectomy for lucaemic enlargement. Ann Surg. 1888;8:380–3.
7. Nixon A. Laparotomy for inflammation of the vermiform appendix with ulcerative perforation, followed by recovery. Ann Surg. 1888;8:23–38.
8. Dixon A. Case of strangulated hernia: operation followed by laparotomy for intestinal obstruction. Ann Surg. 1888;8:371–80.
9. Eads BB. Ventral hernia following abdominal section. Ann Surg. 1901;33:1–12.
10. Mazzochi A, Dessy LA, Ranno R, Carlesino B, Rubino C. Component separation technique and panniculectomy for repair of incisional hernia. Am J Surg. 2011;201:776–83.
11. Koontz AR. Preliminary report on the use of tantalum mesh in the repair of ventral hernias. Ann Surg. 1948;127:1079–85.
12. Ramirez OM, Ruas E, Dellon AL. Components separation method for closure of abdominal wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86:519–26.
13. Cumberland VH. A preliminary report on the use of a prefabricated nylon weave in the repair of ventral hernia. Med J Aust. 1952;1:143–4.
14. Scales JT. Tissue reactions to synthetic materials. Proc R Soc Med. 1953;46:647–52.
15. Usher FC, Fries JG, Ochsner JL, Tuttle Jr LL. Marlex mesh, a new plastic mesh for replacing tissue defects. II. Clinical studies. AMA Arch Surg. 1959;78:138–45.
16. Usher FC, Gannon JP. Marlex mesh, a new plastic mesh for replacing tissue defects. I. Experimental studies. AMA Arch Surg. 1959;78:131–7.
17. Shankaran V, Weber DL, Reed RL, Luchette FA. A review of available prosthetics for ventral hernia repair. Ann Surg. 2011;253:16–24.
18. Lomanto D, Iyer SG, Shabbir A, et al. Laparoscopic versus open ventral hernia mesh repair. Surg Endosc. 2006;20:1030–5.
19. Burger JW, Luijendijk RW, Hop WCJ, Halm JA, Verdaasdank EGG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg. 2004;240:578–85.
20. Millikan KW. Incisional hernia repair. Surg Clin North Am. 2003;83:1223–34.
21. Luijendijk RW, Hop WC, van den Tol MP, De Lange DCD, Braaksma MMJ, Jzerman JMM, et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med. 2000;343:392–8.
22. Sauerland S, Schmedt CG, Lein S. Primary incisional hernia repair with or without polypropylene mesh: a report on 384 patients with 5-year follow-up. Langenbecks Arch Surg. 2005;390:408–12.

23. Iversen E, Lykke A, Hensler M, Jorgensen LN. Abdominal wall hernia repair with a composite ePTFE/polypropylene mesh: clinical outcome and quality of life in 152 patients. Hernia. 2010;14: 555–60.
24. Poelman MM, Langenhorst LAM, Schellekens JF, Schreurs WH. Modified onlay technique for the repair of the more complicated incisional hernias: single-centre evaluation of a large cohort. Hernia. 2010;14:369–74.
25. Abdollahi A, Maddah GH, Mehrabi BM, Jangoo A, Forghani MN, Sharbaf N. Prosthetic incisional hernioplasty: clinical experience with 354 cases. Hernia. 2010;14:569–73.
26. Jenkins ED, Melman L, Deeken CR, Greco SC, Frisella MM, Matthews BD. Biomechanical and histologic evaluation of fenestrated and nonfenestrated biologic mesh in a porcine model of ventral hernia repair. J Am Coll Surg. 2011;212:327–39.
27. Snyder CW, Graham LA, Gray SH, Vick CC, Hawn MT. Effect of mesh type and position on subsequent abdominal operations after incisional hernia repair. J Am Coll Surg. 2011;212:496–504.
28. Tuveri M, Tuveri A, Nocolo E. Repair of large abdominal incisional hernia by reconstructing the midline and use of an onlay of biological material. Am J Surg. 2011;202:e7–11.
29. Swanstrom LL. Laparoscopic herniorrhaphy. Surg Clin North Am. 1996;76:483–91.
30. LeBlanc KA, Booth WV. Laparoscopic repair of incisional abdominal hernias using expanded polytetrafluorethylene: preliminary findings. Surg Laparosc Endosc. 1993;3:39–41.
31. Heniford BT, Park A, Ramshaw BJ, Boeller G. Laparoscopic ventral and incisional hernia repair in 407 patients. J Am Coll Surg. 2000;190:645–50.
32. Mason RJ, Moazzez A, Sohn HJ, Berne TV, Katkhouda N. Laparoscopic versus open anterior abdominal wall repair: 30-day morbidity and mortality using the ACS-NSQIP database. Ann Surg. 2011;254:641–52.
33. Sauerland S, Walgenbach M, Habermalz B, Seiler CM, Miserez M. Laparoscopic versus open surgical techniques for ventral or incisional repair. Cochrane Database Syst Rev. 2011;3: CD007781.
34. Gray SH, Hawn MT, Itani KMF. Surgical progress in inguinal and ventral incisional hernia repair. Surg Clin North Am. 2008; 88:17–26.

Anatomy and Physiology of the Abdominal Wall: Surgical Implications

3

Ronald Merrell

Introduction

The abdominal wall forms a container filled with solid and hollow viscera. The volume is a function of pressure with a potential for vast distension in isobaric conditions or with little change in pressure. The normal pressure is less than 10 mmHg (13.6 cm H_2O) [1]. The pressure needs to be raised only to 15 mmHg to accommodate the entire 5 l or so needed for the distension of pneumoperitoneum in laparoscopy [2]. The cavity can distend to allow the growth of a full-term fetus with little change in pressure. Furthermore, the cavity can be distended with ascites to grotesque dimensions without organ compromise. The distensibility of the cavity is used to excellent advantage for peritoneal dialysis or to increase the tissue surface in preparation for hernia repair [3]. The tissues stretch. The hollow viscera are compressible, of course, but the noncompressible elements of solid viscera and vessels are well served by abdominal wall distensibility. Difficulties for the solid viscera and vessels are discussed in this chapter.

The abdomen is well designed to increase its volume with minimal change in wall tension. The problem for intraperitoneal physiology comes when the pressure rises rapidly and the abdominal wall cannot mitigate the pressure with volume. The first victim of pressure in the abdomen is the diaphragm, with displacement into the thorax and a rise in respiratory pressures. The next victim is the inferior vena cava, with reduction in right heart return and relative hypovolemia. Each of these can be compensated by resuscitative measures. However, the ureter and renal calices are also affected by increased intra-abdominal pressure, and resuscitative measures may not easily compensate; renal oliguria follows. The pressure in the abdominal wall is accurately reflected by pressure in the urinary bladder, which is easily measured through a urinary catheter. The problem of excess pressure caused by failure of the abdominal wall to distend is termed *abdominal compartment syndrome*, with a fall in renal function at 25 mmHg or higher [4, 5]. The problem is so urgent that drastic measures such as decompressive celiotomy or leaving the abdomen open have become standard practices in the last 30 years.

Although the abdominal wall customarily handles chronic pressure threats with distension, the wall can sustain enormous increases in pressure that are brief, such as in coughing or heavy lifting. In these circumstances, the pressure may reach 150 cm H_2O in reflex coughing but never at the risk of compromising the normal abdominal wall over many decades of life and many thousands of cough strains [6]. With exercise-induced strain, the pressure may reach 250 cm H_2O.

The container function of the abdominal wall is indispensible. Without the integrity of the abdominal wall, viscera protrude along with whatever coatings of peritoneum, subcutaneous tissue, and skin remains. This protrusion through the otherwise containing influence of the abdominal wall is a hernia. If the coatings fail, the abdominal contents under the influence of even normal intra-abdominal pressure will rush from the body as an evisceration.

Anatomical Boundaries

For the purpose of this chapter, the term *abdominal wall* refers to the anterior abdominal wall, and failings are limited to acquired failure caused by either surgical incision or ventral hernia of natural causes. No discussion of herniation through natural weaknesses and orifices is included. The anterior abdominal wall in terms of structural integrity is composed of muscle and fascia attached to the costal margin, spine, pelvic rim, and pubis (Fig. 3.1). The attachments are firm although elastic. The fascia is cleverly engineered to overlap with decussation in the midline linea alba (Fig. 3.2).

R. Merrell, MD
Department of Surgery,
Virginia Commonwealth University,
Richmond, VA, USA
e-mail: rcmerrel@vcu.edu

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_3,

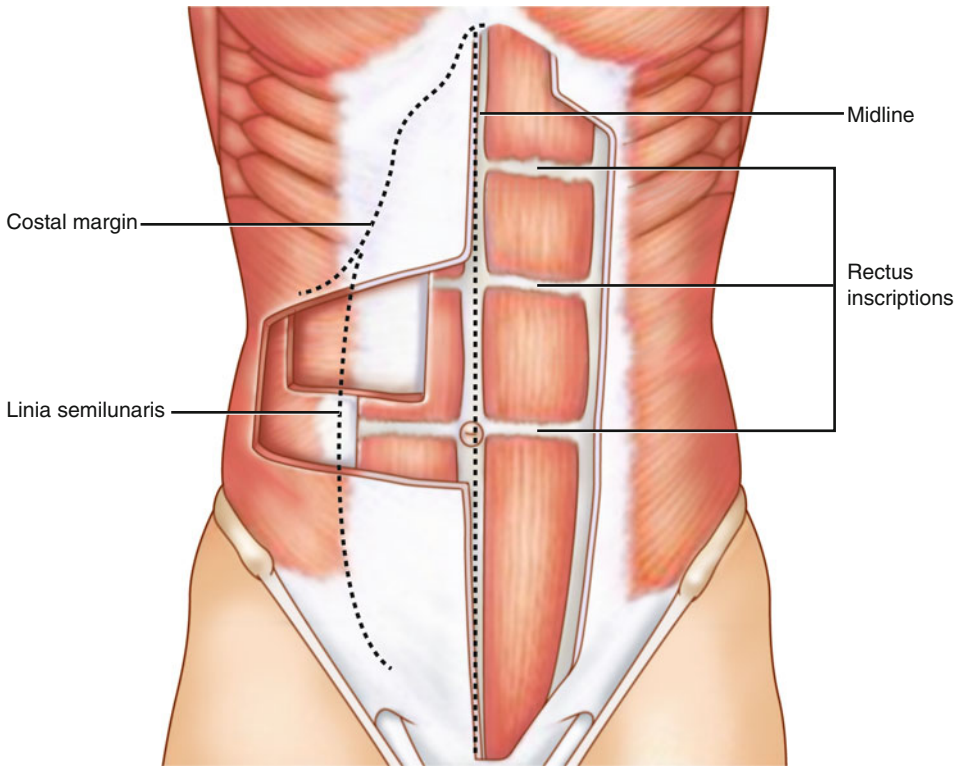


Fig. 3.1 Frontal view of the abdominal wall at the level of the fascia indicating midline, costal margin, linea semilunaris, and rectus inscriptions

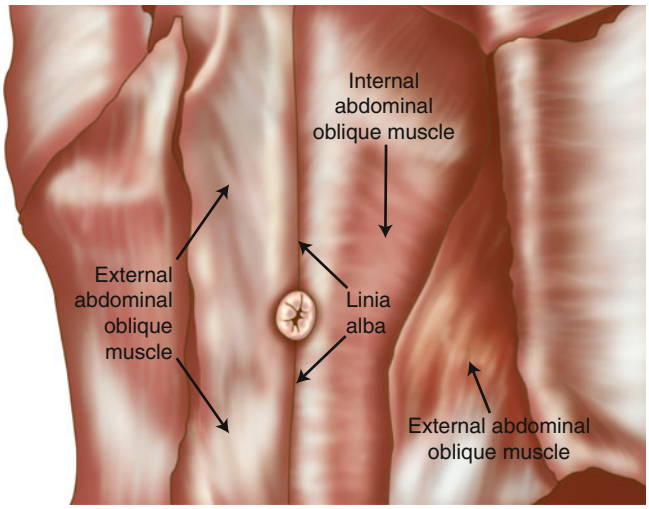


Fig. 3.2 Criss-crossing decussating fibers at the linea alba, with umbilicus

The fascia has a dominance of collagen type 1 and is dynamic rather than static, with a fairly vigorous biological turnover. The fascia in turn invests three layers of abdominal wall musculature, which are oriented not in parallel but at various angles to increase strength (Fig. 3.3a, b). The transversus abdominis is more or less oriented horizontally. The internal oblique is oriented superiorly, and the external oblique is at a right angle to this, directed essentially as hands would be thrust into the pocket. The musculofascial structure is further differentiated toward the midline. The fascia of the internal oblique splits to invest the rectus abdominis above the arcuate line, and the anterior leaflet fuses with the external oblique fascia to form the rectus fascia and the linea alba at the midline. Below the arcuate line, the fascia of the internal oblique sweeps anterior to the rectus entirely to unite with the external oblique to form something of a bulwark in the lower abdomen, where gravity would predict pressures in the wall will be somewhat greater than in the cephalad abdomen. The layers of the abdominal wall are easiest seen in cross section by computed tomographic imaging (Fig. 3.4a, b). The three layers and their relationship to the rectus abdominis are clearly visible.

The abdominal wall has neurovascular bundles coming from the back in a dermatomal distribution from T8 to T10. Crucial blood supply comes from the superior and inferior epigastric vessels along the belly of the rectus abdominis (Fig. 3.5a, b). The merger and fusion of the external and internal oblique muscles form the linea semilunaris to the lateral aspect of the rectus. Just below the umbilicus, the change in the fascia of the internal oblique from separation cephalad to invest the rectus to a unitary sweep behind the rectus forms an arch termed the arcuate line.

Abdominal Wall Distensibility

The anterior abdominal wall is strong but easily distended, which is explained by the structure. The collagen stretches, as does the muscle. The abdominal wall can be stretched

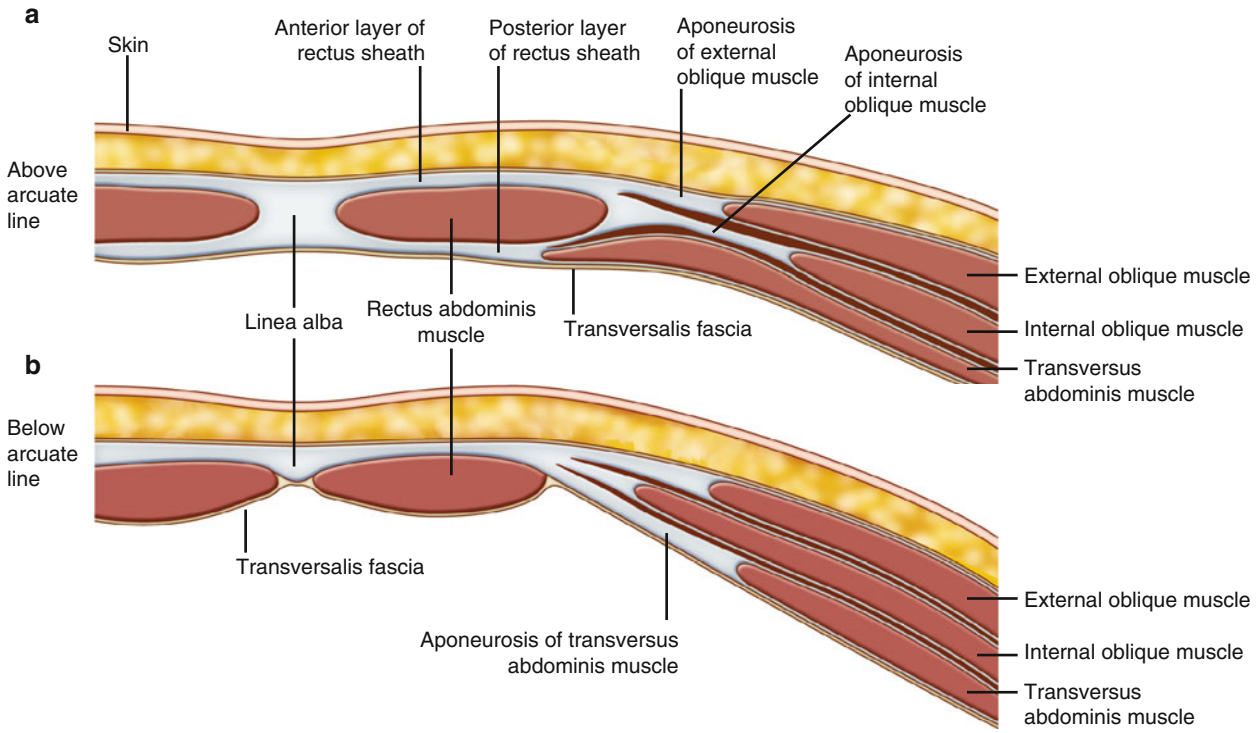


Fig. 3.3 (**a**) Cross section of the anterior abdominal wall indicating the split of the internal oblique fascia to invest the rectus above the arcuate line. (**b**) Internal oblique fascia sweeping behind the rectus below the arcuate line

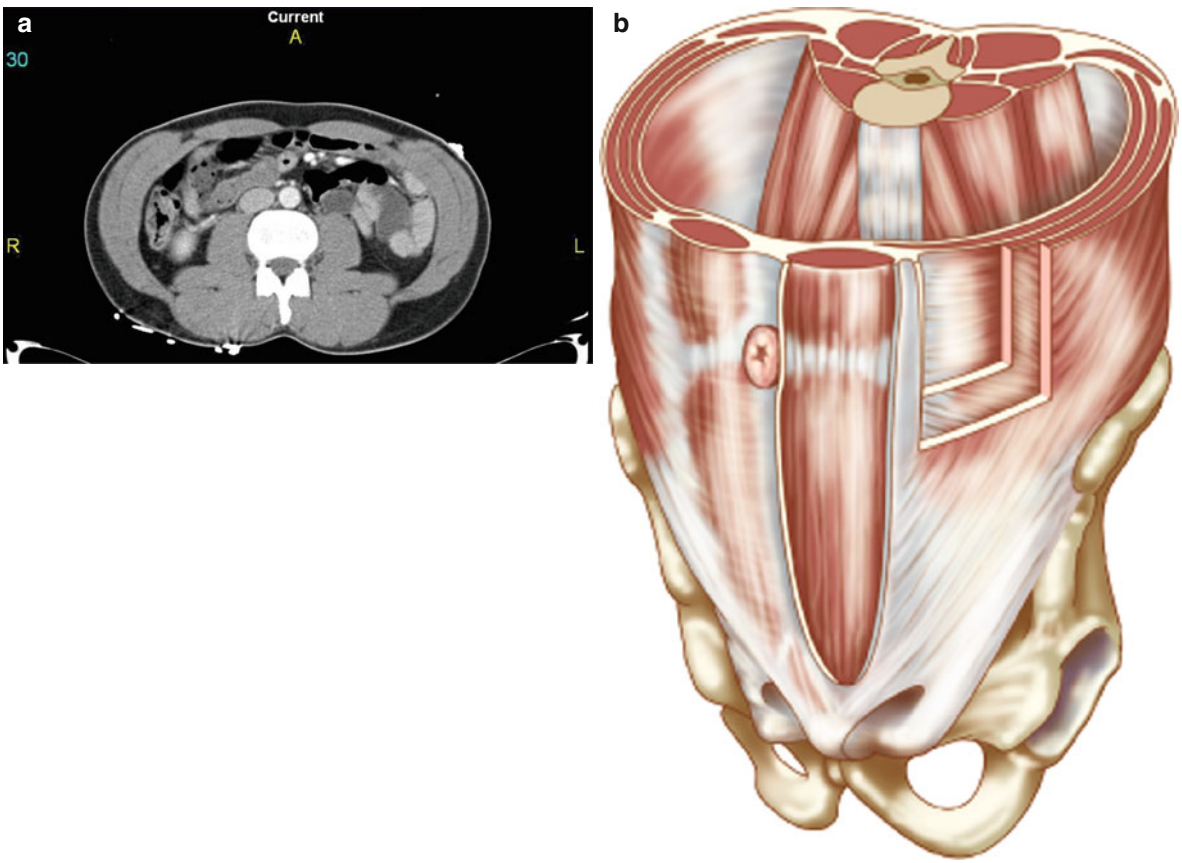


Fig. 3.4 (**a**, **b**) Layers of the abdominal wall as seen in cross-sectional computed tomography (CT) (**a**) and in illustrated form (**b**). The three layers and their relationship to the rectus abdominis are clearly seen

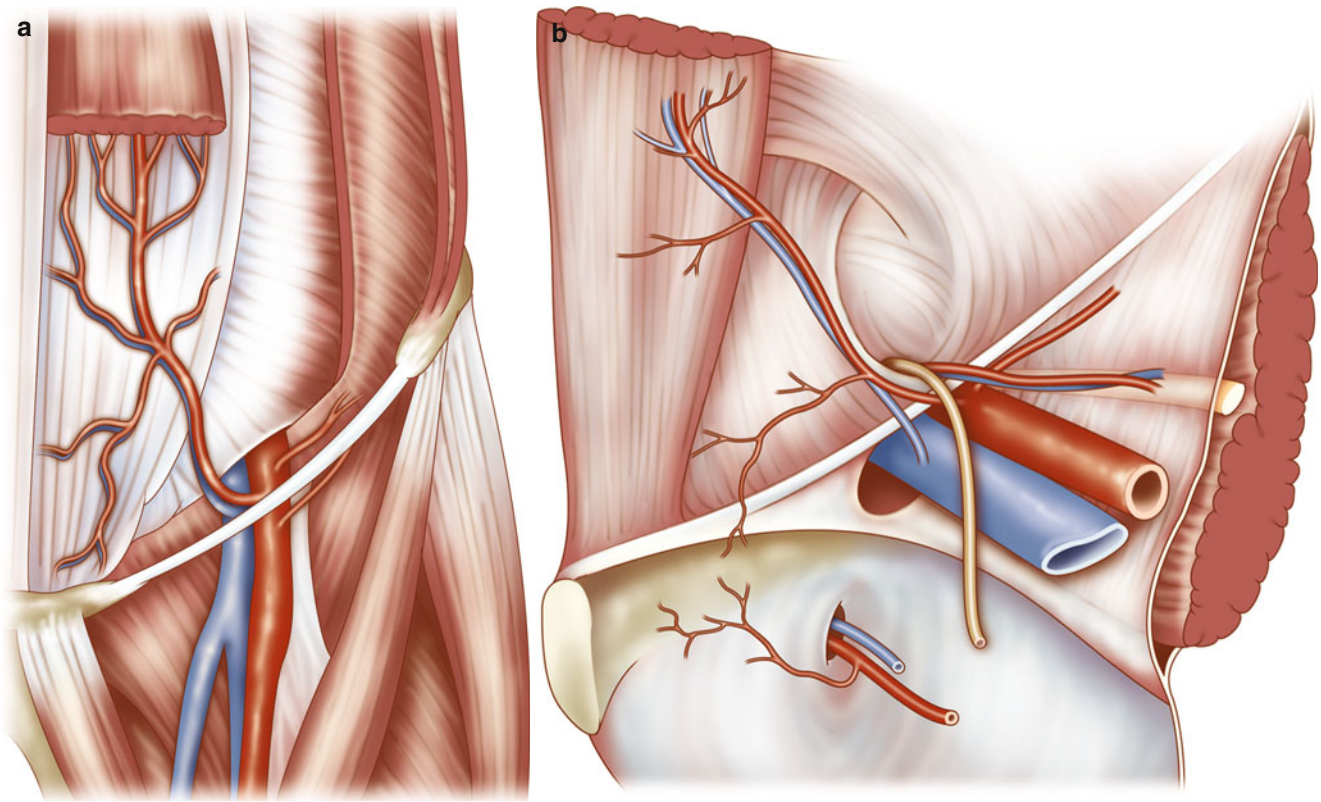


Fig. 3.5 (**a**, **b**) Posterior views of the anterior abdominal wall showing inferior epigastric vessels coming from below at external iliac and suggestion of dermatomal vessels coming from the sides. This is critical in planning a repair to have blood supply

quite thin without losing its integrity, as in ascites. The distensibility is remarkable in that even an excess of gas in a compressible viscera such as the colon or obstructed small bowel will stretch the abdominal wall to fabulous dimensions without threatening structural integrity. Please note the contrast with the pressure and wall tension features of the cecum in obstruction. Past 14-cm distension, the integrity of the cecum is in great peril, but the anterior abdominal wall is durable. The Laplace effect is certainly applicable to the abdominal wall, but the wall tensions are not an issue for the native anatomy. The issue is only of importance when we consider the bursting strength of the altered wall, say after repair from laparotomy. The abdominal wall will fall back to its proper tension and dimension almost immediately after the obstruction is relieved. This resumption of size is also remarkable after delivery of conceptus or drainage of ascites.

The triple layering and the decussation of the linea alba create a restraining structure that is remarkable for its high bursting strength. In fact, there is no force that can breach the abdominal wall integrity except that of the well-intended surgeon, an assailant, or some other penetrating assault. It is aptly named a *wall* because its ability to hold in the natural and hold out the offensive is a great compliment to evolution and biology.

In human biology, natural function is most often studied in the context of pathology. Except for early work on anatomy with cadaver dissection, the majority of our advances in understanding the human state have been prompted by studying its shortcomings in disease. Such is the case for a thorough comprehension of the anterior abdominal wall. The abdominal wall after injury and repair has a higher representation of immature collagen type III that persists. This material lacks many of the better characteristics of collagen type I, and that difference has been used to explain the propensity of the integrity of the wall of the abdomen to fail after incision and repair [7]. Abnormal collagen has been associated with poor abdominal repair in congenital conditions such as Ehlers–Danlos syndrome [8]. Abnormal collagen in aortic aneurysms was proposed by Tilson many years ago, and an association with high hernia rates after aneurysm repair was identified [9]. Indeed, it is well recognized that the repair of the abdominal wall does not lead to restoration of its full glory, and failure of the wall through hernia has been an affliction that continues to the present despite massive efforts to reproduce what nature does so

well: to create a retaining and protective wall over the abdominal cavity that allows massive excursion in strain and pressure during the extremes of human work, reproduction, athleticism, and most external physical assaults, at least those with blunt instruments.

Surgical Implications

The anatomy and physiology of massive abdominal wall hernia deserve mention. As the abdominal contents emerge from the abdomen proper into the large sac of peritoneum, subcutaneous tissue, and skin, the pressure in the abdomen is maintained. However, with cough or strain the contents can leap from the cavity with propulsive and painful consequence. Because the hernia contents are associated with prior operation, they may obstruct because of adhesions in the sac. However, with large hernias, the likelihood of incarceration into the neck of the hernia seems to diminish. The defects may be single or multiple. There is a dictum that says "a hernia never gets smaller with the exception of the congenital umbilical hernia in the first 3 years of life." Indeed, the progress of the exodus from the cavity proper is relentless, and the hernia sac may come to hold more of the abdominal viscera than the contracted abdominal cavity. The abnormal anatomy and physiology of the herniated abdomen seem to demand restoration to normal to the extent possible with surgical intervention. However, with truly massive herniation and insufficient volume remaining in the cavity, repair of a hernia, in fact, may not be feasible. Furthermore, as repairs demand increasingly greater surgical measures, there is a balance between patient interests in the restoration versus the danger and morbidity of the repair itself. With massive hernia well compensated by nature, the obligation to repair must be considered an elective matter and not a surgical certainty.

A further remark should be reserved for prevention. Massive abdominal hernia should not be considered inevitable. With sepsis, massive distension, malnutrition, ascites leak, cancer invasion, cardiac insufficiency, hypoxia, multiple fistulae in inflammatory bowel disease, and major resection of the abdominal wall itself, perhaps some hernias are inevitable. However, as an operating principle, consideration of the hermetic closure of the violated abdomen, even if in stages, should be a large concern to the original operating surgeon. Of the over 100,000 ventral hernia repairs per year in the United States, surely most could be considered preventable [10]. How is prevention ensured?

Conclusion

Detailed knowledge of the native anatomy, physiology, physics, and biology of the abdominal wall should permit a coherent approach to the choice of closing materials and their application technique. The surgeon should strive to replicate as closely as possible normal abdominal wall anatomy.

References

1. Knaebel HP. Abdominal compartment syndrome. In: Bland KI et al., editors. General surgery: principles and international practice, vol. 2. London: Springer; 2008. p. 157–9.
2. Mulier JP, Dillemans BRS, Crombach M, Missant C, Sels A. On the abdominal pressure volume relationship. Int J Anesthesiol. 2009;21:1.
3. Astudillo R, Merrell R, Sanchez J, Olmedo S. Ventral herniorrhaphy aided by pneumoperitoneum. Arch Surg. 1986;121:935–6.
4. Smith JH, Merrell RC, Raffin TA. Reversal of postoperative anuria by decompressive celiotomy. Arch Intern Med. 1985;145:553–4.
5. Saggi BH, Sugerman HJ, Ivatury RR, Bloomfield GL. Abdominal compartment syndrome. J Trauma. 1998;45:597–609.
6. Addington WR, Stephens RE, Phelipa MM, Widdicombe JG, Ockey RR. Intra-abdominal pressures during voluntary and reflex cough. Cough. 2008;4:2.
7. Henriksen NA, Yadete DH, Sorensen LT, Agren MS, Jorgensen LN. Connective tissue alteration in abdominal wall hernia. Br J Surg. 2010;98:210–9.
8. Ehlers-Danlos syndrome. A.D.A.M. medical encyclopedia. Atlanta: NIH; 2011.
9. Chew DK, Knoetgen J, Xia S, Tilson MD. The role of a putative microfibrillar protein (80 kDa) in abdominal aortic aneurysm disease. J Surg Res. 2003;114:25–9.
10. Rutkow IM. Demographic and socioeconomic aspects of hernia repair in the United States in 2003. Surg Clin North Am. 2003;83:1045–51. v-vi.

The Biology of Complex Abdominal Wall Defects: Definitions and Causes

4

Fernando Turégano and Andrés García-Marín

Definition of Complex Abdominal Wall Defects

Although there is no single universally accepted definition, the term *complex abdominal wall defect* (CAWD) generally describes wounds that may anatomically involve several tissues, often develop after severe injuries and their surgical management, and do not heal in a timely manner or fail to heal completely. Comorbidities are common, and often multiple. Prolonged periods of wound management can delay chemo-radiation treatments, represent a significant toll on a patient's quality of life, compound psychological devastation on top of injury and illness, and might lead to cosmetically unacceptable results [1].

The CAWDs usually require a distinct and individualized, frequently interdisciplinary, intervention beyond primary repair or the simple placement of mesh. These CAWDs include recurrent hernias with multiple failed repairs, infection or other local tissue compromise, inadequate soft tissue coverage, or multiple sites of abdominal wall defects. A subset of patients requires concomitant procedures, such as enterostomy or enterocutaneous fistula (ECF) takedown, bowel resection, or specific plastic surgical approaches, including complex wound closure, panniculectomy, and abdominoplasty [2, 3]. Some authors have suggested the following CAWD criteria be used to identify patients who might require special closure techniques for an abdominal wall defect: large size (>40 cm^2), absence of stable skin coverage, recurrence of defect after prior closure attempts, infected or exposed mesh, patient who is systemically compromised (e.g., intercurrent malignancy), compromised local abdominal tissues (e.g., irradiation, corticosteroid dependence), and concomitant visceral complications (e.g., ECF) [3].

The CAWDs are not all alike, and their anatomic complexity varies; the comorbidities and previous surgical histories of different patients vary as well. All of this has a significant impact on the outcome [4]. These defects can be superficial, involving only some layers of the soft tissues of the abdomen (Fig. 4.1), or they can be full thickness, extending into the abdominal cavity.

Causes of Complex Abdominal Wall Defects

Full-thickness open abdominal (OA) wounds primarily are encountered in patients after acute trauma, infectious processes, or abdominal catastrophes. In some instances, such defects represent life-threatening conditions with loss of domain, persistent infections, exposed abdominal viscera, bowel fistulas, and lateral retraction of the abdominal wall (Fig. 4.2). Furthermore, some patients are gravely ill, in poor general health, with several significant medical problems, including sepsis, compromised nutritional status, immunosuppression, and cardiopulmonary problems. Such patients will need to be managed aggressively and in a timely fashion to avoid further complications and deterioration that could affect the outcome of any future reconstructive procedure or endanger their lives.

In other patients, there is no tissue loss but simply a loss of domain with chronic and long-standing recurrent incisional hernias (Fig. 4.3) [5]. Long-standing neglected primary abdominal wall hernias with loss of domain, which can create a complex clinical problem, are less frequent (Fig. 4.4).

In a practical and specific sense, acquired CAWDs are mainly caused by abdominal wall infections complicating surgical procedures, with resulting recurrent incisional

F. Turégano, MD, PhD (✉)
Department of General Surgery and Emergency Surgery,
University General Hospital Gregorio Maranon,
Dr. Esquerdo 46, Madrid 28007, Spain
e-mail: fturegano.hgugm@salud.madrid.org

A. García-Marín, MD
Department of General Surgery and Digestive Surgery,
University Hospital San Juan de Alicante,
Ctra. Alicante-Valencia s/n, Alicante 03550, Spain
e-mail: andres.garciam@umh.es

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_4,

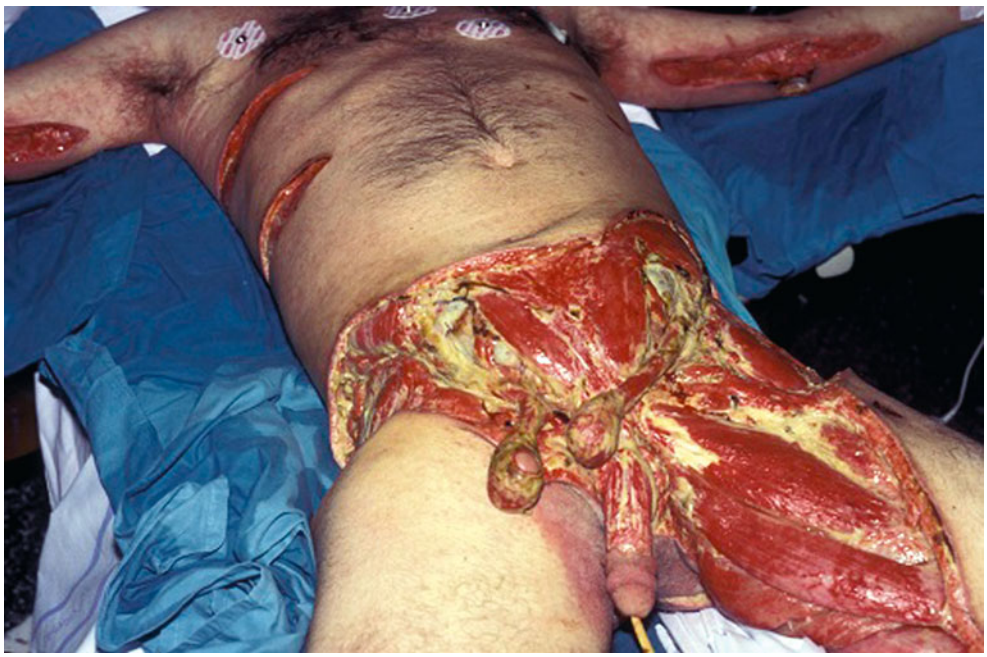


Fig. 4.1 Extensive fasciitis by *Streptococcus pyogenes* of urethral source involving the abdominal and thoracic walls and the extremities and creating a superficial complex abdominal wall defect

hernias, the OA approach after damage control (DC) procedures in acute care surgical problems, or less frequently, ablative resection of primary or recurrent tumors, among other less-common conditions [5].

Abdominal Wall Infections and Recurrent Incisional Hernias

An acute wound infection is the main etiologic factor, although not the only one, behind the development of recurrent incisional hernias. These ventral hernias represent the main etiologic group within most series of CAWDs. Ghazi et al. from Emory University in Atlanta described a series of 165 patients with CAWDs treated over a 7-year period; of these individuals, 101 (61 %) had recurrent ventral hernias [6].

To a lesser degree, severe and extensive abdominal wall necrotizing infections requiring surgical resection can also occasionally result in CAWDs. They occur most frequently after gastrointestinal operations, especially in the immune-compromised host with multiple comorbidities, and might be associated with fistulas of the gastrointestinal tract. Clostridial myonecrosis, although rare, is the most severe form of abdominal wall infection (Figs. 4.5 and 4.6).

Failure of biomaterials represents a significant setback in patient care. Patients might present with an array of problems ranging from wound dehiscence and infection to suture line disruptions with subsequent formation or recurrence of abdominal wall hernias, mesh extrusion, or even intra-abdominal complications such as bowel damage and fistulas. The incidence of fistula formation with various alloplastic materials has been reported to be as high as 33 %. It has also been well recognized that the incidence of fistulas is increased with the use of some type of synthetic prostheses, and that fistula formation can occur even when absorbable meshes are used.

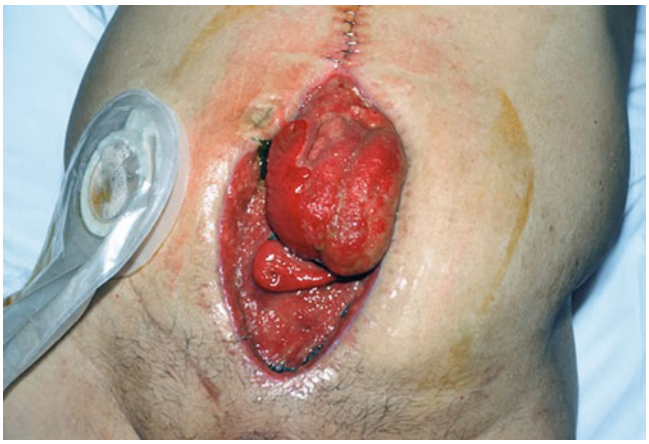


Fig. 4.2 Postoperative full-thickness complex abdominal wall defect with entero-atmospheric fistula

Damage Control, the Open Abdomen and Approach

CAWDs in this setting are the result of emergency laparotomies performed for a number of severe conditions and can pose a formidable challenge to the clinical surgeon. A DC laparotomy in trauma and emergency surgery, with repeated reentries in the abdominal cavity, is a harbinger of a potential CAWD. The DC surgery and the OA approach have led to an increase in survival of the patient with severe trauma,

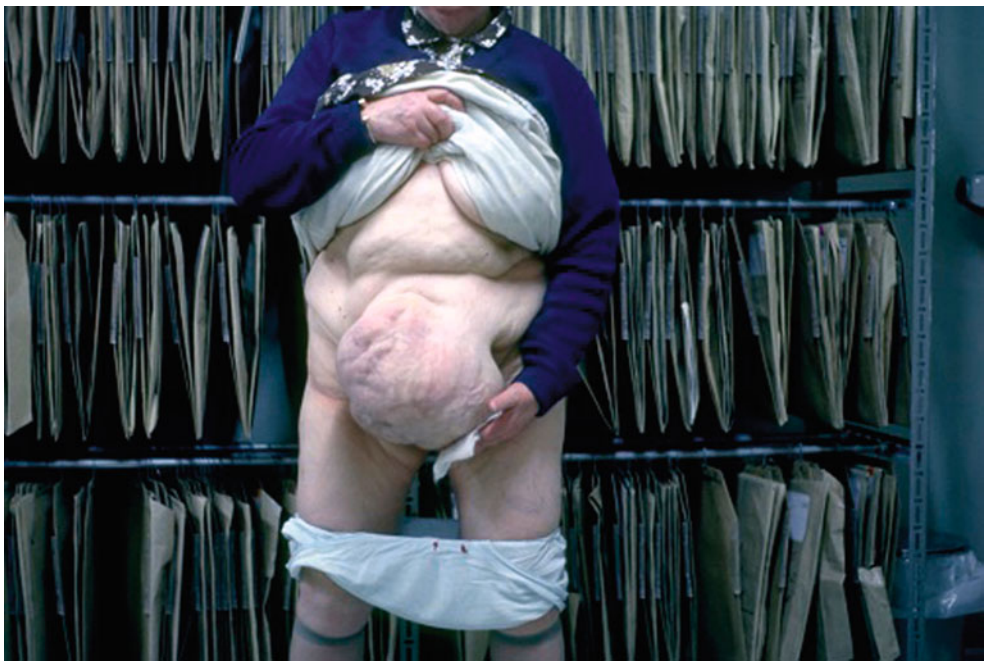


Fig. 4.3 Loss of domain after recurrent incisional hernia

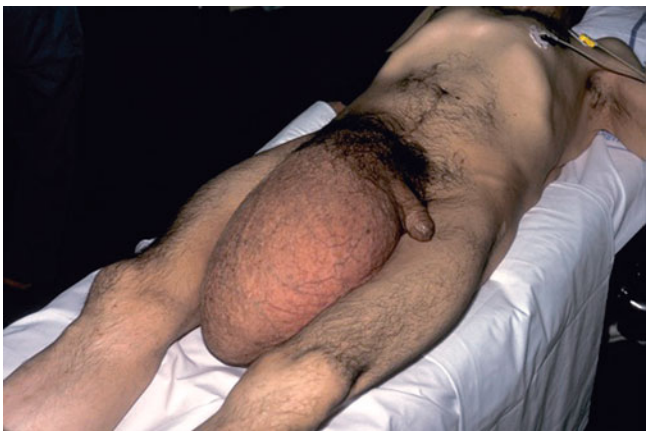


Fig. 4.4 Loss of domain after long-standing neglected right inguinoscrotal hernia

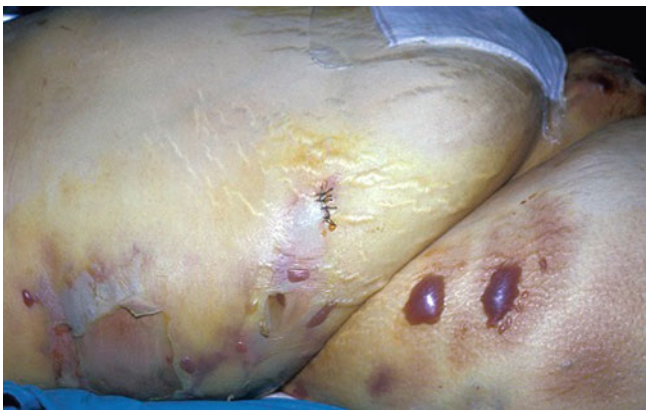


Fig. 4.5 Postoperative fulminant necrotizing fasciitis of the abdominal wall after creation of a colostomy for diverticulitis

and this has created an increased need to reconstruct complex defects thereafter (Fig. 4.7). The incidence of chronic ventral hernias is common in this setting, with a wide range (13–80 %) that depends on patient-specific factors and institutional patterns of practice (i.e., aggressive fascial repair vs. a "planned ventral hernia" approach) [7]. Because of the potentially devastating consequences of prosthetic infections, biologic meshes, both cross-linked and non-cross-linked, are currently being recommended when native tissue component repair is not possible [8–11]. These meshes, together with the vacuum pack technique, might diminish the rate of planned ventral hernia approaches in the future in favor of early primary fascial closure [12], with a likely decrease in the overall morbidity and the percentage of CAWDs resulting from this DC/OA surgery [13]. Nevertheless, the data to date suggest that the majority of patients repaired with biological mesh might develop laxity of the repair, resulting in a hernia 6–12 months later [14].

Surgical site infections and intra-abdominal abscesses associated with DC/OA occur in as many as 83 % of cases and might also contribute to postoperative fascial dehiscence (reported in up to 25 % of DC/OA patients) [15].

Resection of Abdominal Wall Tumors

Primary malignancies of the abdominal wall are uncommon. Desmoid tumors are benign fibrous tumors that arise from the musculoaponeurotic structures of the abdominal wall. They are frequently locally invasive (aggressive fibromatosis)

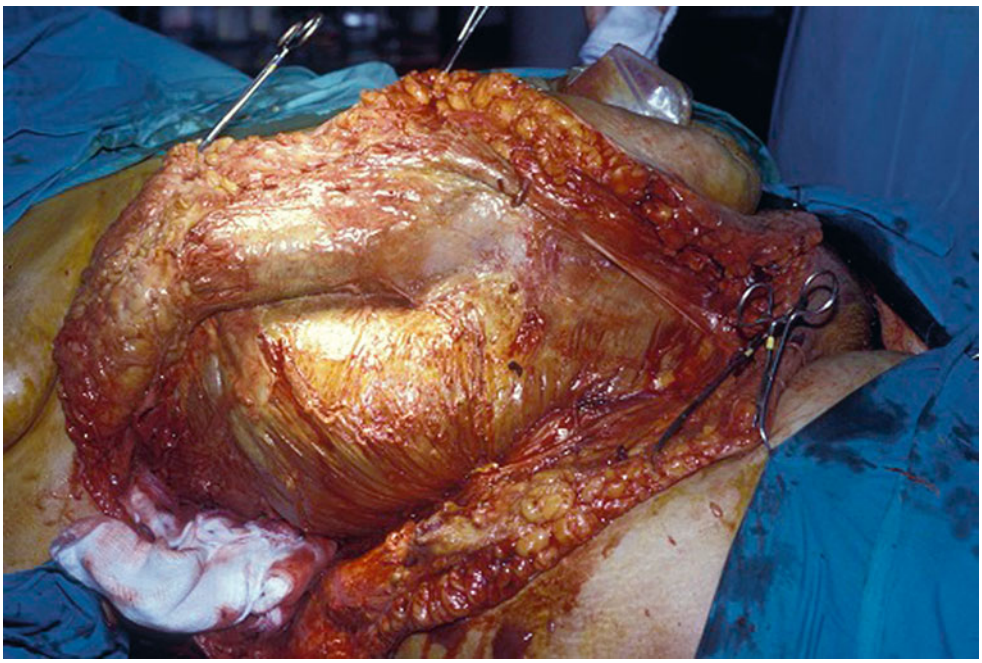


Fig. 4.6 Same patient as in Fig. 4.5 undergoing extensive debridement and resection of the infected abdominal wall tissues

(Figs. 4.8 and 4.9), and local recurrence rates of 25–65 % after local excision have been reported. Treatment requires wide excision followed by complex abdominal wall reconstruction in some cases. This reconstruction is usually performed immediately with synthetic materials (meshes) or myocutaneous flaps when the defect is extensive [16], usually in collaboration with plastic and reconstructive surgeons.

The Biology of Complex Abdominal Wall Defects

Complex Recurrent Incisional Hernias and the Pathophysiology of Wound Healing of the Abdominal Wall

The abdominal cavity represents one of the most active areas of surgical activity, and surgical procedures involving the gastrointestinal (GI) tract are among the most common procedures performed. Full understanding of the pathophysiology of the healing responses after the surgical procedure remains elusive. Nevertheless, progress in this area is of great interest because complications of abdominal healing represent a significant clinical and economic burden as well as a decrease in quality of life [17].

The abdominal wall is a complex region of the body; all of its components are organized in a delicate balance to provide maximal protection while preserving physiologic and locomotive function. It is a laminated cylinder of muscle and fascia with an overlying, well-vascularized skin envelope. It serves as a core unit for musculoskeletal posturing, a protective barrier for the viscera, and a base for respiratory

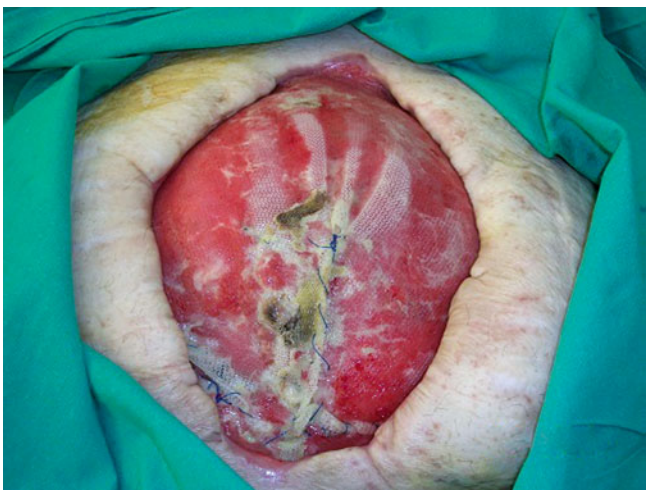


Fig. 4.7 Open abdomen with overlying synthetic mesh and lateral retraction of the abdominal wall

mechanics. The maintenance of constant intra-abdominal pressure allows for support in respiration, locomotion of the trunk, as well as micturition and defecation, among other physiologic functions [17].

When the abdominal wall is in a weakened state, intra-abdominal pressure follows fluid patterns and tends to exert the greatest pressure at the weakest point as opposed to the natural state of diffuse and equal distribution [6]. Although true strangulation of hernia contents is uncommon, many patients with a recurrent incisional hernia have lifestyle-limiting symptoms that necessitate operative intervention. Patients may present with chronic dull abdominal pain. They might have postural alterations, leading to lumbar lordosis

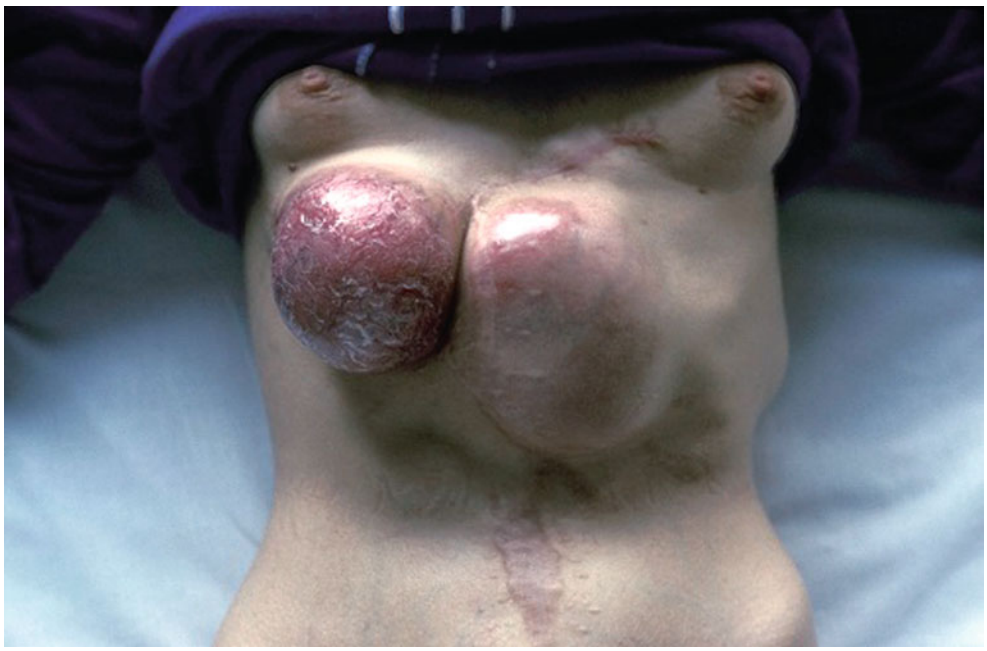


Fig. 4.8 Recurrent fatal aggressive fibromatosis of the abdominal wall in a 19-year-old woman

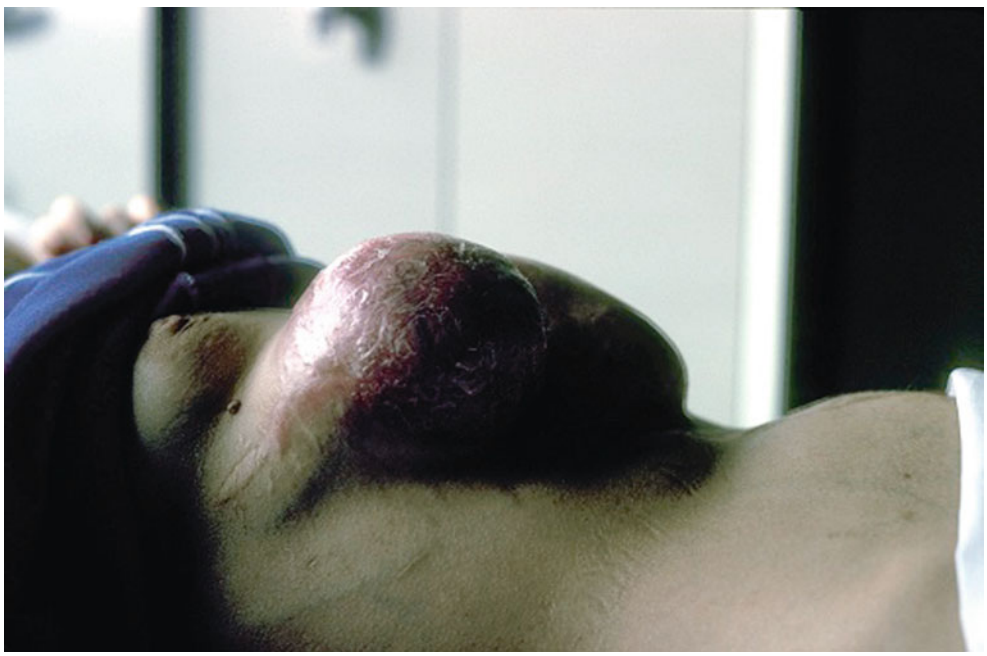


Fig. 4.9 Lateral view of the same patient in Fig. 4.8

and chronic back pain. A massive CAWD can also lead to paradoxical respiratory motions, which inhibit respiratory mechanics.

Biological and Mechanical Factors Involved

Modern surgical practice suggests that biologic and mechanical pathways overlap during normal acute wound healing. The cellular and molecular processes activated to repair tissue from the moment of injury are under the control of biologic and mechanical signals. Successful acute wound healing occurs when a dynamic balance is met between the loads placed across a provisional matrix and the feedback and feed-forward responses of repair cells [18].

When a midline incisional hernia develops, the normal force across the composite myofascial structure is lost, functionally resulting in passive unloading of the lateral abdominal wall. Although the adjacent rectus muscles maintain their origin and insertion, the insertion of lateral oblique muscles is lost following midline laparotomy and incisional hernia formation. The linea alba of the abdominal wall is anatomically a tendon that, when severed, should induce pathologic abdominal wall muscle changes similar to those observed in

the soleus and gastrocnemius muscles when the Achilles tendon is divided [19]. In a rat model of chronic incisional hernia formation, the authors showed that internal oblique muscles in herniated abdominal walls developed pathologic fibrosis, disuse atrophy, and changes in muscle fiber-type composition. Myopathic disuse atrophic changes significantly altered the phenotype of the herniated anterior abdominal wall. Hernia defects do not enlarge simply by repetitive evisceration of peritoneal contents dilating a fascial defect. Rather, the lateral muscular components of the abdominal wall retract away from the midline fascial defect, and this has therapeutic implications.

Laparotomy wound healing is a complex process involving interplay between many different types of cells; failure with progression to hernia formation is multifactorial. Biologic factors that contribute to simple and complex abdominal wall defects are multiple [20, 21]:

1. Inflammation: Following initial insult, first neutrophils and later monocytes-macrophages arrive at the injured tissue, debride it, and secrete growth factors (GFs). Wound strength is low during this phase and depends only on the sutures; a prolonged inflammatory response such as seen with incisional foreign bodies or infections predispose to wound failure; besides, microorganisms can degrade GFs and synthesize proteinases that remove extracellular matrix [20, 22]. Steroids can reduce inflammation but inhibit collagen synthesis.
2. Fibroblasts: Fibroblasts are responsible for collagen synthesis and the recovery of wound-breaking strength; this is the dominant cell type during the proliferative and remodeling phases. Little is known about defective fibroblast function in wound failure. Some authors have suggested that the loss of abdominal wall load forces signaling as a result of fascial healing failure would select an abnormal population of repair fibroblasts (mechano-transduction pathways) similar to those widely described in tendons, ligaments, and bone repair [20, 23, 24]. Recent in vitro studies suggested that early fascial separation and diminished wound tension might lead to loss of a key stimulatory mechanical signal for fibroblast proliferation, alignment, and contraction function, resulting in the inability to heal the initial wound failure with subsequent progression to hernia formation [25].
3. Collagen: Collagen is the main structural protein in abdominal wall fascial layers (at least 80 % of tissue dry weight). Defects are described either in its synthesis, with an increase of type III collagen and decrease of collagen I/III ratio and with thinner and less-resistant fibers, or in its degradation, with an increase of matrix metalloproteinase (MMP) activity [20, 26]. Numerous studies have now associated incisional hernias with impaired collagen and tissue protease metabolism, and there is a strong correlation between MMP-1 and MMP-13 overexpression and recurrent hernia [26–28].
4. Growth Factors: It is not known whether delays in the appearance of GFs contribute to the development of incisional hernias. Experimental models have demonstrated that wound treatment with transforming GF beta 2 or basic fibroblast GF stimulates angiogenesis, fibroblast chemotaxis, and collagen production, increasing wound resistance and reducing the incidence of incisional hernia [22, 29, 30].

Local and General Factors Affecting Wound Healing

Local Factors

Closure Under Tension and Blood Supply

It now appears that, in load-bearing systems such as the abdominal wall, a tension equilibrium point exists that maximizes repair signals to wound repair fibroblasts (mechano-transduction pathways) [27]. Nevertheless, closure under excessive tension is probably the most common reason for several complications, ranging from superficial wound dehiscence, infection, and tissue necrosis and loss to abdominal compartment syndrome (ACS) [5]. The site of an incision might disturb the blood supply to a wound. Vertical parallel incisions on the same side of the midline impair healing of the wound placed more medially and risk necrosis of the intervening skin bridge. Suturing might adversely affect the blood supply of a healing wound, especially if there is infection and edema.

Hematoma

Postoperative seromas and hematomas, if not recognized early on and appropriately managed, also might result in wound dehiscence, infection, and tissue loss [5]. A mass of blood apparently exerts a toxic effect independent of the level of bacterial contamination and of the amount of internal pressure they produce, theoretically obstructing the dermal circulation and causing necrosis [31].

Infection

Infection is the most common complication of wound healing. The principal biochemical abnormality in infected wounds seems to be a disturbance of fibroblast proliferation and subsequent collagen metabolism. In DC surgery, the incidence of dehiscence and abdominal wall infections is approximately 9 and 25 %, respectively, and their development is multifactorial [15]. The intra-abdominal hypertension (IAH) that commonly develops in this population reduces abdominal blood flow even in the face of maintained arterial perfusion pressures, contributing to local edema and ischemia. This impairs wound healing, and the ischemic tissue provides a site for bacterial infection.

Irradiation

There are several hypotheses on the role of circulatory decrease and radiation-induced direct cellular damage. Recent advances highlight that transforming GF beta 1 is the master switch in pathogenesis of radiation fibrosis [32].

Mechanical Stress

A rise in intra-abdominal pressure by coughing or distention of intestine is a factor in abdominal wound failure. Sutures might cut through the abdominal wall or break.

Surgical Technique

Good technique and gentle handling is one of the most important factors affecting healing in surgical practice.

Tissue Type

The surface epithelium of the skin retains its power of regeneration throughout life. The bulk of tissue lost dictates whether the process of repair is primary or secondary.

General Factors

Age

Wound healing complications (e.g., abdominal wound dehiscence) are more common in elderly persons. Age affects epithelialization and maturation of the scar as well as gain of tensile strength.

Anemia

Anemia has been linked with an increased incidence of abdominal wound dehiscence, although it is almost impossible to separate it from other factors, such as the nutritional state and the type of surgery performed.

Diabetes

Failure of wound healing, particularly related to infection, is encountered in up to 10 % of diabetic patients undergoing operations. It has been known for some time that neuropathy, atherosclerosis, and propensity to infection, all frequently encountered in diabetic patients, might contribute to wound-healing failure. A large body of evidence from in vitro and in vivo studies indicates that advanced glycation end products might play a role in the pathogenesis of impaired diabetic wound healing [33].

Nutrition

Undoubtedly, there is a relationship between malnutrition and abdominal wound dehiscence and infection. The exuberant cellular and biochemical events that constitute the wound-healing cascade require energy, amino acids, oxygen, metals, trace minerals, and vitamins for successful completion. Many nutritional deficiencies have an impact on wound healing by impeding fibroblast proliferation, collagen synthesis, and epithelialization. There are also nutrients that can enhance wound-healing responses [34].

Steroids

Experimentally, large doses of steroids depress the healing process and reduce wound strength. Nevertheless, care should be used in assigning wound-healing problems to steroid therapy because many patients receiving steroids are elderly, malnourished, and often suffering from malignant disease. Steroid therapy begun several days postoperatively has little effect on wound healing, and acute stress or single-dose steroids have no effect on healing.

Jaundice

Experimental evidence in abdominal incisions suggests that jaundice delays the appearance of wound fibroblasts and new blood vessels and affects collagen synthesis, although the clinical relevance of these findings is uncertain. The role of jaundice in predisposing to problems of wound healing is probably multifactorial. The baseline synthesis of types I and III collagen in the skin is decreased in jaundiced patients; this is partly restored by the resolution of jaundice [35].

Malignant Disease

It is difficult to conclude from clinical studies what effect malignant tumor cells or their systemic sequelae have on healing because associated local problems, such as infection and obstruction, might also be present.

Obesity

Incisional hernias are significantly associated with obesity, partly through an increased occurrence of wound hematomas and infection [36].

Temperature

Wounds heal more slowly in low temperatures, probably through reflex vasoconstriction.

Trauma, Hypovolemia, and Hypoxia

Postraumatic hypovolemia and low inspired oxygen tension are associated with delayed healing, especially delayed collagen synthesis. There is also an increased susceptibility to infection, probably because of tissue hypoxia.

Uremia

Experimental evidence three decades ago showed that certain aspects of wound healing might be adversely affected by uremia, leading to considerable diminution in the bursting strength of the laparotomy wound [37]. Also, a high wound complication rate was found after abdominal operations in patients undergoing long-term peritoneal dialysis. Poor nutrition together with a high urea level were found to be significant [38]. In vitro studies have shown that uremic solutes decrease endothelial proliferation and wound repair [39].

Complex Abdominal Wall Defects from Damage Control Surgery and the Open Abdomen

The peritoneal environment is instrumental in the response to injury that occurs with DC surgery or trauma. The peritoneum is composed of mesothelial cells that respond to

surgically induced tissue trauma, ischemia, and infection. The local inflammatory response within the abdomen results in a copious fluid and cellular response in the first 48 h, but this will continue at a slower rate while the abdomen remains open [40]. The initial response involves migration and activation of macrophages in the peritoneum. Fibroblasts, platelets, and chemoattractants such as thrombin and plasmin are part of the cascade for healing and functional restoration. Vascular injury and subsequent endothelial cell activation result in fibrinogen accumulation and chemokine release. Mast cells recruited secondary to the peritoneal trauma release histamine and vasoactive kinins, which in turn increase capillary permeability [17].

After 48 h, the formation of fibrin within the exudate results in a gelatinous mass in which intestine and omentum are loosely held. During the next 4 or 5 days, this loose coagulum is replaced by increasingly tough adhesions as polymerization of fibrin and collagen occurs, and, by day 10, the abdomen is sealed by vascular, organizing granulation.

The practical implication of this healing process is that, beyond the first 10 days, any attempt to suture the fascial edges or dissect the bowel away from the posterior aspect of the anterior abdominal wall is likely to result in multiple enterotomies and fistulas [41]. Enterocutaneous fistulas are the second most common type of abdominal complication associated with DC/OA, and they arise as a result of a leaking anastomosis, bowel ischemia, obstruction, exposure of the bowel to the air, or ill-advised dissection. The incidence varies between 5 and 19 %, depending on the presenting diagnosis/indication for DC/OA [7, 15]. If the fistula arises in a mobile portion of the bowel, it might slowly rise to the surface, where mucosa might be seen (enteroatmospheric fistula, which rarely closes spontaneously). The lack of skin surface around the fistula makes for difficult management, aggravating the already-existing abdominal wall defect. The organizing granulation might adhere to the margins of the fistula, and eventually it will be incorporated into the scar, uniting the edges of the abdominal wall. Thus, if a fistula is present after a period of 10 days in an OA, a long period of supportive treatment is inevitable before repair and closure of the CAWD is contemplated [41].

Although the maturing adhesions are laying down increasing amounts of fibrin and collagen from the first week, a strong and sudden increase in intra-abdominal pressure, such as from a strong cough, in the first 3 weeks might rupture the fragile coagulum holding the gut, omentum, and abdominal wall together, spilling intestine onto the surface of the abdomen. Such eviscerations might produce serosal tears and fistulas. Furthermore, the process of adhesion formation and maturation fixes the omentum and bowel to the edges and posterior aspects of the abdominal wall.

Because of the natural elasticity of the abdominal wall structures, wound retraction in the OA will progress during the first week, and this could produce evisceration, with the bowel losing its "right of abode" within the abdominal cavity. The practical implication for the surgeon is that evisceration should be converted as rapidly as possible to an eventration: The abdomen, though open, contains most or all of the intestines. By the end of the first 2 weeks, vascular granulation will occlude the surface of the OA, uniting the edges of the wound. Over the succeeding weeks and months, collagenization of the wound proceeds to convert this granulation into a scar. During this process, a hernia might not be visible because of the density of the scar. Over the succeeding months and up to a year, the collagen is slowly removed, the scar thins and softens, and a hernia will become apparent. If this large, granulating, OA wound is skin grafted, wound contraction and the development of collagen might be impaired, leading to the early development of large hernias with a progressive loss of abdominal abode.

On the basis of limited evidence, functional status in patients with a CAWD resulting from DC/OA seems to be dependent on several factors, including the size of the hernia, the presence of skin and subcutaneous tissue overlying the midline defect, and the presence of a fistula [42]. There are some reports of up to 55–78 % of patients eventually returning to work after abdominal closure or reconstruction [43], although other studies of patients with large chronic ventral hernias showed persistent significant impairment of activity, productivity, and quality of life [44]. The successful repair of these CAWDs might be a challenge [45], and the biology of the healing process in this OA approach must be well understood and respected by the surgeon to achieve a successful final outcome.

Summary

In summary, the definition of what constitutes a CAWD is not universal, but its causes are varied and well recognised by practicing surgeons all over the world. The complex mechanisms and factors intervening in wound repair at the molecular level are not fully understood to this day, but the biology of these defects and the difficulties involved in their management are better known. CAWDs are very often a real challenge to the technical abilities, patience and wisdom of surgeons.

References

1. Park H, Copeland C, Henry S, Barbul A. Complex wounds and their management. Surg Clin North Am. 2010;90:1181–94.
2. Hadeed JG, Walsh MD, Pappas TN, et al. Complex abdominal wall hernias. A new classification system and approach to management based on review of 133 consecutive patients. Ann Plast Surg. 2011;66:497–503.

3. Mathes SS, Steinwald PM, Foster RD, et al. Complex abdominal wall reconstruction: a comparison of flaps and mesh closure. Am J Surg. 2000;232:286–96.
4. Varkarakis G, Daniels J, Coker K, et al. Effects of comorbidities and implant reinforcement on outcomes after component reconstruction of the abdominal wall. Ann Plast Surg. 2010;64:595–7.
5. Cohen M. Management of abdominal wall defects resulting from complications of surgical procedures. Clin Plast Surg. 2006;33:281–94.
6. Ghazi B, Deigni O, Yezhelyev M, Losken A. Current options in the management of complex abdominal wall defects. Ann Plast Surg. 2011;66:488–92.
7. Smith BP, Adams RC, Doraiswamy VA, et al. Review of abdominal damage control and open abdomens: focus on gastrointestinal complications. J Gastrointestin Liver Dis. 2010;19:425–35.
8. Connolly PT, Teubner A, Lees NP, Anderson ID, Scott NA, Carlson GL. Outcome of reconstructive surgery for intestinal fistula in the open abdomen. Ann Surg. 2008;247:440–4.
9. Latifi R, Gustafson M. Abdominal wall reconstruction in patients with enterocutaneous fistulas. Eur J Trauma Emerg Surg. 2011;37:241–50.
10. Shankaran V, Weber DJ, Reed RL, Luchette FA. A review of available prosthetics for ventral hernia repair. Ann Surg. 2011;253:16–26.
11. Díaz JJ, Dutton WD, Ott MM, et al. Eastern Association for the Surgery of Trauma: a review of the management of the open abdomen—part 2 "management of the open abdomen". J Trauma. 2011; 71:502–12.
12. Hatch QM, Osterhout LM, Ashraf A, et al. Current use of damage-control laparotomy, closure rates, and predictors of early fascial closure at the first take-back. J Trauma. 2011;70:1429–36.
13. Miller RS, Morris JA, Diaz JJ, Herring MB, May AK. Complications after 344 damage-control open celiotomies. J Trauma. 2005;59: 1365–74.
14. De Moya MA, Dunham M, Inaba K, et al. Long-term outcome of acellular dermal matrix when used for large traumatic open abdomen. J Trauma. 2008;65:349–53.
15. Shapiro MB, Jenkins DH, Schwab W, Rotondo MF. Damage control: collective review. J Trauma. 2000;49:969–78.
16. Bertani E, Chiappa A, Estori A, et al. Desmoid tumors of the anterior abdominal wall: results from a monocentric surgical experience and review of the literature. Ann Surg Oncol. 2009;16:1642–9.
17. Munireddy S, Kavalukas SL, Barbul A. Intra-abdominal healing: gastrointestinal tract and adhesions. Surg Clin North Am. 2010; 90:1227–36.
18. DuBay DA, Franz MG. Acute wound healing: the biology of acute wound failure. Surg Clin North Am. 2003;83:463–81.
19. DuBay DA, Choi W, Urbanchek MG, et al. Incisional herniation induces decreased abdominal wall compliance via oblique muscle atrophy and fibrosis. Ann Surg. 2007;245:140–6.
20. Franz MG. The biology of hernia formation. Surg Clin North Am. 2008;88:1–15.
21. Bellón JM, Durán HJ. Biological factors involved in the genesis of incisional hernia. Cir Esp. 2008;83:3–7.
22. Payne WG, Wright TE, Ko F, et al. Bacterial degradation of growth factors. J Appl Res. 2003;3:35–40.
23. DuBay DA, Wang X, Adamson B, et al. Progressive fascial wound failure impairs subsequent abdominal wall repairs: a new animal model of incisional hernia formation. Surgery. 2005;137:463–71.
24. Benjamin M, Hillen B. Mechanical influences on cells, tissues and organs—"mechanical morphogenesis". Eur J Morphol. 2003;41:3–7.
25. Culbertson EJ, Xing L, Wen Y, Franz MG. Loss of mechanical strain impairs abdominal wall fibroblast proliferation, orientation, and collagen contraction function. Surgery. 2011;150:410–7.
26. Henriksen NA, Yadete DH, Sorensen LT, et al. Connective tissue alteration in abdominal wall hernia. Br J Surg. 2011;98:210–9.
27. Franz MG. The biology of hernias and the abdominal wall. Hernia. 2006;10:462–71.
28. Antoniou SA, Antoniou GA, Granderath FA, et al. The role of matrix metalloproteinases in the pathogenesis of abdominal wall hernias. Eur J Clin Invest. 2009;39:953–9.
29. Franz MG, Kuhn MA, Nguyen K, et al. Transforming growth factor beta2 lowers the incidence of incisional hernias. J Surg Res. 2001; 97:109–16.
30. DuBay DA, Wang X, Kuhn MA, et al. The prevention of incisional hernia formation using a delayed-release polymer of basic fibroblast growth factor. Ann Surg. 2004;240:179–86.
31. Bucknall TE. Factors affecting wound healing. Probl Gen Surg. 1989;6:194–219.
32. Devalia HL, Mansfield L. Radiotherapy and wound healing. Int Wound J. 2008;5:40–4.
33. Peppa M, Stavroulakis P, Raptis SA. Advanced glycoxidation products and impaired diabetic wound healing. Wound Repair Regen. 2009;17:461–72.
34. Kavalukas SL, Barbul A. Nutrition and wound healing: an update. Plast Reconstr Surg. 2011;127 Suppl 1Suppl 1:38S–43S.
35. Koivukangas V, Oikarinen A, Risteli J, Haukipuro K. Effect of jaundice and its resolution on wound re-epithelization, skin collagen synthesis, and serum collagen propeptide levels in patients with neoplastic pancreaticobiliary obstruction. J Surg Res. 2005;124: 237–43.
36. Yahchouchy-Chouillard E, Aura T, Picone O, Etienne JC, Fingerhut A. Incisional hernias. I. Related risk factors. Dig Surg. 2003; 20:3–9.
37. Colin JF, Elliot P, Ellis H. The effect of uremia upon wound healing. An experimental study. Br J Surg. 1979;66:793–7.
38. Moffat FL, Deitel M, Thompson DA. Abdominal surgery in patients undergoing long-term peritoneal dialysis. Surgery. 1982;92: 598–604.
39. Dou L, Bertrand E, Cerini C, et al. The uremic solutes p-cresol and indoxyl sulfate inhibit endothelial proliferation and wound repair. Kidney Int. 2004;65:442–51.
40. Faull RJ. Peritoneal defences against infection: winning the battle but losing the war? Semin Dial. 2000;13:47–53.
41. De Costa A. Making a virtue of necessity: managing the open abdomen. ANZ J Surg. 2006;76:356–63.
42. Fabian TC. Damage control in trauma: laparotomy wound management acute to chronic. Surg Clin North Am. 2007;87:73–93.
43. Cheatham ML, Safcsak K. Longterm impact of abdominal decompression: a prospective comparative analysis. J Am Coll Surg. 2008; 207:573–9.
44. Uranues S, Salehi B, Bergamaschi R. Adverse events, quality of life, and recurrence rates after laparoscopic adhesiolysis and recurrent incisional hernia mesh repair in patients with previous failed repairs. J Am Coll Surg. 2008;207:663–9.
45. Howdieshell TR, Proctor CD, Sternberg E, Cue JI, Mondy JS, Hawkins ML. Temporary abdominal closure followed by definitive abdominal wall reconstruction of the open abdomen. Am J Surg. 2004;188:301–6.

Preoperative Patient Optimization

5

Ruben Peralta and Rifat Latifi

Introduction

In the quest to provide the best care for patients undergoing major abdominal wall reconstruction, a multidisciplinary and systematic approach should be undertaken by all members involved in the operative management of the patients. Since the 1980s, we have seen significant advances in the management of medical and surgical conditions of critically injured patients; these advances have led to improved survival. As a result, however, the practicing surgeon is faced with the management of the open abdomen and large abdominal wall defects that require major abdominal wall reconstruction [1–7]. The reconstruction of a large abdominal wall defect might present a major insult to patients who are already unconditioned physiologically and psychologically [8]. This chapter focuses on the multidisciplinary approach and measures to take into consideration in the preoperative optimization of complex cases, which are frequently associated with complications, high rate of hernia recurrence requiring multiple procedures, and mortality [1–13].

R. Peralta, MD, FACS
Trauma Section, Department of Surgery,
Hamad General Hospital,
Doha, Qatar
e-mail: rperaltamd@gmail.com

R. Latifi, MD, FACS (✉)
Department of Surgery,
The University of Arizona,
1501 N. Campbell Ave,
Tucson, AZ 85724, USA

Trauma Section, Department of Surgery,
Weill Cornell Medical College,
Hamad General Hospital, Doha, Qatar
e-mail: latifi@surgery.arizona.edu

Preoperative Optimization

In complex cases, preoperative optimization encompasses a thorough preoperative evaluation and takes into consideration the timing of the surgical repair.

Preoperative Evaluation

All patients undergoing a major abdominal wall reconstruction procedure should receive a systematic preoperative evaluation and objective assessment of their risk through established methods such as those of the American Society of Anesthesiologists (ASA) Physical Status Classification System, the Goldman Cardiac Risk Index, and the like [14, 15]. Obtaining preoperative information can lead to better preparation of the patient for a major surgical procedure and modification of intraoperative strategy, management, and postoperative care, all of which will result in better outcomes and patient satisfaction. The guidelines for the preoperative evaluation of complex cases will facilitate the entire process. We should take into consideration, however, that "one size does not fit all"; frequently, such complicated patients might require deviations from the already-established guidelines. Other considerations to be addressed during this period include the following: patient and family expectations and the expertise of the operating team (i.e., attending surgeon and anesthesiologist, intensive care unit nurses, and other healthcare providers involved in the medical care from admission to discharge, including rehabilitation) [16].

Timing of the Surgical Repair

Getting ready for a long and complex reconstructive procedure requires timing and preparation to achieve optimization of the patient. A recent published review article by the working group of the Eastern Association for the Surgery of

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_5, © Springer Science+Business Media New York 2013

Trauma (EAST) defined the timing and type of procedures commonly used in the closure of the abdominal wall defect and planned ventral hernia; the authors used a common language with a proposed clinical flow diagram [17].

The main goal of the preoperative evaluation is to achieve the best possible optimization of the patient, and this might require the postponement of the definitive closure or repair and the establishment of a temporary closure in the acute setting or during the resuscitative phase (damage control concept) [1–3].

Preoperative Evaluation Clinic

The preoperative evaluation process should be undertaken in a location where all healthcare providers are active participants in the process [18]. Our experience and the literature confirm that a multidisciplinary team is efficient, reduces chance of errors and cost, and decreases operating room cancellations [19–21].

The evaluation process starts with a thorough history and physical examination and should be performed by a multidisciplinary team. The anesthesiologist, intensivist, and surgeon involved in the procedure should be part of the evaluating team and ideally should communicate the indications and risk of the procedure to the patient and close relatives in the same setting.

Assessing the Perioperative Risk

Assessing the risk in a systematic fashion is imperative and should focus on evaluating the capacity of the patient to withstand the acute physiological stress resulting from prolonged operative procedures and general anesthesia that extends well into the recovery and rehabilitation phases. Furthermore, we should treat major life-threatening common conditions, such as hypoxia, hypoglycemia, major fluid and electrolyte imbalances, sepsis, coagulopathy and other major organ impairment, and estimate if the patient can meet the increased oxygen demand caused by the stress response to surgery and anesthesia. By zeroing in on the neurological, cardiovascular, respiratory and renal systems, we will have a better grasp of the long-term functional outcome of those patients.

Neurological System Evaluation

A significant number of our patients have a history of major injuries, including traumatic brain injury, major abdominal vascular injury, and devastating surgical catastrophic conditions requiring prolonged hospitalization. Furthermore, delirium is a common condition in these patients and is associated with increased length of stay, morbidity, and mortality [22–30].

Patients with recent history of traumatic brain injury, spinal cord injury, and cerebrovascular accident or patients with high index of clinical suspicion or recent neurological deterioration might require neuroimaging studies and monitoring prior to the procedure. If the major abdominal wall reconstruction will be performed during the acute traumatic brain injury phase, an intracranial pressure (ICP) monitoring device might be indicated as per *The Brain Trauma Foundation: Guidelines for the Management of Severe Traumatic Brain Injury* [31].

Patients with a spinal cord injury may present unique challenges in the management of the intraoperative, acute postoperative, and rehabilitation phases depending on the level of the cord injury. Patients with high spinal cord injuries might require a secure airway, prolonged ventilator support, and prolonged rehabilitation care in specialized centers.

Cardiovascular System Evaluation

Patients undergoing major abdominal wall reconstruction are at risk of major perioperative cardiac events. There are numerous published guidelines to be used in the evaluation of the cardiovascular risk [32–37]. Some patients, however, arrive with devastating neurological and orthopedic injuries or are elderly, which makes the process of obtaining an accurate functional status almost impossible, partly because of their limited mobility or altered mental status. It is also pertinent to mention that there are important limitations of some of the cardiovascular risk indexes used today. For example, the Lee index is a practical way to assess the cardiac risk, but it does not take into consideration emergency surgery and the increasing number of elderly patients undergoing surgical procedures today [33]. In addition, it is important to delineate that currently many of the multiple procedures included in such risk indexes are performed in a minimally invasive fashion.

For patients undergoing noncardiac surgery, another algorithm used for stratification according to the patients' perioperative cardiac risk was published by the American College of Cardiology/American Heart Association (ACC/AHA). Strong evidence-based practice is lacking for this guideline [34–37]. The routine use of noninvasive testing or stress testing is not recommended for most of these patients [38]. We recommend the revised Lee cardiac risk index, which quantitates cardiac risk, and its basis is the number of risk factors : high-risk surgery, ischemic heart disease, congestive heart failure, cerebrovascular disease, insulin-dependent diabetes mellitus, renal failure, hypertension, and age greater than 75 [33, 39]. Stress testing is not predictive of myocardial ischemia/infarction (MI) or death and is only recommended in patients with unstable angina or an

active arrhythmia. Cardiology consultation, when clinically indicated, is necessary for optimal care of the patient [38]. In a recent study of patients for vascular surgery, the use of beta-blocker decreases cardiac risk, and the timing and dosage (titration) influence outcomes, but improper usage might increase stroke and the death rate; the authors recommended that not all patients for vascular surgery should take these drugs [40–42].

Respiratory System Evaluation

Evaluating respiratory function may be necessary in patients undergoing major abdominal wall reconstruction. The history of a respiratory condition should be considered, including asthma and chronic obstructive pulmonary diseases. Any respiratory infection should be eradicated prior to a surgical procedure. Physiological capacity has been compared with the ASA Physical Status Classification System, and has been associated with postoperative morbidity [43]. Studies of preoperative cardiopulmonary exercise testing (CPET) have shown that a reduced oxygen uptake at anaerobic threshold (AT) and elevated ventilatory equivalent for carbon dioxide (VE/VCO) were associated with reduced short- and medium-term survival after major surgery. The authors determined that using CPET for patients undergoing high-risk surgery can accurately identify the majority of high-risk patients [44].

Renal System Evaluation

Acute and chronic kidney derangements are frequent in this population of patients because, in the majority of cases, the etiology of the large abdominal wall defects is from major trauma or catastrophic general surgery and abdominal vascular conditions. In severely injured patients, despite advances in resuscitation, acute kidney injury (AKI) is still a frequent occurrence and remains an important predictor of multiorgan failure and mortality [45].

There are few perioperative measures to take into consideration in the management of such complex patients: prevention of contrast-induced nephropathy with acetylcysteine and fluid management, control of diabetes mellitus and hypertension, optimization of the fluid status of the patient, and close monitoring of aminoglycoside administration.

In the acute setting, AKI can be associated in severely burned and polytrauma patients as a result of increased intra-abdominal pressure and development of the abdominal compartment syndrome (ACS), which should be recognized in a timely manner and the abdomen promptly decompressed to reverse the renal dysfunction [46]. The use of nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided in the setting of hypoperfusion and renal dysfunction.

Another condition that is associated with AKI is rhabdomyolysis. The management of rhadomyolysis is to focus on the correction of the underlying cause (i.e., compartment syndrome, etc.) and undertake prompt and vigorous volume replacement. Compartment syndrome in the extremities is a clinical diagnosis, and fasciotomy of the affected limb should be performed as soon as it is recognized [5, 47]. The most common method used in evaluation and monitoring of renal function deterioration is measurement of the serum creatinine and blood urea nitrogen levels. Measures of glomerular filtration rate and creatinine clearance are also commonly employed. Control of urea levels can prevent platelet dysfunction and mental status changes. Optimizations of renal function in patients with AKI and chronic kidney conditions might require renal replacement therapies to obtain a good control of uremia, electrolyte disturbance such as hyperkalemia and acidosis, and fluid status [47].

Gastrointestinal System Evaluation

Evaluation and optimization of the entire gastrointestinal (GI) system is of major importance because derangement of GI tract continuity is a frequent complication in patients requiring abdominal wall reconstruction because of major abdominal wall defect [48–50].

Disruption in the continuity of the intestine will affect the course of management in the acute and elective reconstruction settings. Enterocutaneous fistulas remain among the most challenging complications associated with patients with open abdomen and major abdominal trauma requiring abdominal wall reconstruction. In patients undergoing major surgery, goal-directed hemodynamic therapy (GDT), by maintaining adequate systemic oxygenation, can protect organs particularly at risk of perioperative hypoperfusion and is effective in reducing GI complications as described by a recent metanalysis [51].

The effort of the multidisciplinary team is to reestablish continuity of the GI tract, enabling prompt use of oral or enteral feeding to optimize the patient's nutritional status. The authors recommended a nine-step treatment strategy in abdominal wall reconstruction in patients with an open abdomen and enterocutaneous fistulas; and this is further described in Chap. 16 in this book.

Endocrine System Evaluation

Endocrine disorders are common in critically ill patients and have a global effect on the patient's well-being. A systematic approach to the evaluation and management of common endocrinological conditions should be undertaken during the preoperative period. Examples of common endocrinological derangements observed in critically ill patients

include sodium-level abnormalities, thyroid dysfunction, relative adrenal insufficiency, and abnormal glucose level, among others.

Hematologic and Coagulation Evaluation

Patients might have a history of hematological disorder or have become coagulopathic during the course of management of the severe clinical condition or injury, partly because of the acute major trauma insult, sepsis, acidosis, hypothermia, or iatrogenic effects caused by heparin-induced thrombocytopenia or chronic use of antiplatelet medications. Patient also might have a history of a hypercoagulable state, and the condition could be exacerbated during the hospitalization.

Infections

Infections are frequent in patients with an open abdomen, and source control should be obtained before embarking on abdominal reconstruction. Goal-directed therapies have improved outcomes in patients with severe sepsis and septic shock and are part of our standard of care [52–55].

Nutritional Evaluation and Optimization

The nutritional status of the patient should be considered early in the course of the management of complex conditions. Evaluation and optimization of the nutritional status should be performed prior to major surgical procedures. Methods include evaluation of serum albumin level, prealbumin level, and indirect calorimeter measurements, depending on the availability of the measure at your institution. We strongly recommend early aggressive nutritional support through the initiation of enteral feeding unless a patient's condition dictates otherwise. Our second option is the optimization of nutritional status through initiation and maintenance of parenteral nutrition. Elective abdominal wall reconstruction should be postponed in patients with a history of recent weight loss of 15 % or more, along with an albumin level less than 3 g/dL. There is a strong association reported between postoperative albumin level and morbidity and mortality [56, 57]. Consideration should be given to addressing chronic conditions, such as chronic malnutrition; chronic alcoholism, which is associated with multivitamin deficiencies (thiamine and folate deficiency); and electrolyte abnormalities in sodium, magnesium, phosphorus, potassium, and calcium. In our practice, we prefer to give patients extra supplements of vitamin C, vitamin E, micronutrients such as zinc and selenium, and if clinically indicated, vitamin A. Glycemic control is important in nutritional management and optimization of these patients. Sepsis control and eradication of infectious foci are main components of the management armamentarium and are crucial for obtaining nutritional optimization.

Control of Premorbid Conditions

As previously detailed in this chapter, all chronic conditions should be addressed and optimized per current published clinical practice guidelines. These conditions include diabetes, hypertension, heart problems, thyroid disease, obesity, and those involving the kidney and pulmonary system.

Social and Addiction Issues

Patients who have suffered and survived major injuries and undergoing emergency general surgery and vascular procedures requiring damage control have an associated decreased quality of life [8]. A significant number of patients have a history of chronic complications of alcohol or drug abuse, such as financial instability, homelessness, abusive behaviors, chronic and acute legal problems, and prescription drug abuse, which might require addiction and psychiatry evaluation and management before undergoing major abdominal wall reconstruction. Patients should be enrolled in a smoking cessation program prior to the surgical reconstruction.

Prevention Strategies

For all patients undergoing major abdominal surgery, some conditions can be prevented with a systematic approach: (1) thromboembolic complications by implementing deep venous thrombosis prophylaxis (mechanical and pharmacological treatment if not contraindicated); (2) prevention of surgical site infections by timely administration of perioperative antibiotics; (3) prevention of GI bleeding in high-risk patients, and implementation of various published critical care bundles practiced in your institution [58].

Summary

Preoperative evaluation and optimization are parts of a multidisciplinary process associated with improved outcomes in patients undergoing major abdominal reconstruction procedures. The most frequent method of optimization in the acute setting is fluid management. Surgery of complex abdominal wall defects could be a major undertaking for any surgeon and is associated with frequent complications. Planned, systematic evaluation; perioperative risk assessment; and appropriate timing are essential for providing the best functional outcome.

References

1. Rotondo MF, Schwab CW, McGonigal MD, et al. "Damage control": an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma. 1993;35:375–83.
2. Shapiro MB, Jenkins DH, Schwab CW, et al. Damage control: collective review. J Trauma. 2000;49:969–78.
3. Moore EE, Burch JM, Franciose RJ, et al. Staged physiologic restoration and damage control surgery. World J Surg. 1998;22:1184–91.
4. Rasmussen TE, Hallet Jr JW, Noel AA, et al. Early abdominal closure with mesh reduces multiple organ failure after ruptured abdominal aortic aneurysm repair: guidelines from a 10-year case–control study. J Vasc Surg. 2002;35:246–53.
5. Mcnelis J, Soffer S, Marini CP, et al. Abdominal compartment syndrome in the surgical intensive care unit. Am Surg. 2002;68:18–23.
6. Dutton WD, Diaz JJ, Miller RS. Critical care issues in managing complex open abdominal wound. J Intensive Care Med. 2012;27(3):161–71.
7. Diaz JJ, Cullinane DC, Dutton WD, et al. The management of the open abdomen in trauma and emergency general surgery: part 1—damage control. J Trauma. 2010;68(6):1425–38.
8. Zarzaur BL, DiCocco JM, Shahan CP, et al. Quality of life after abdominal wall reconstruction following open abdomen. J Trauma. 2011;70:285–91.
9. Scott BG, Feanny MA, Hirshberg A. Early definitive closure of the open abdomen: a quiet revolution. Scand J Surg. 2005;94:9–14.
10. Jernigan TW, Fabian TC, Croce MA, et al. Staged management of giant abdominal wall defects. Acute and long-term results. Ann Surg. 2003;238:349–57.
11. Mudge M, Hughes LE. Incisional hernia: a 10-year prospective study of incidence and attitudes. Br J Surg. 1985;72:70–1.
12. Cassar K, Munro A. Surgical treatment of incisional hernia. Br J Surg. 2002;89:534–45.
13. Peralta R, Latifi R. Long-term outcomes of abdominal wall reconstruction. What are the real numbers? World J Surg. 2012;36:534–8.
14. American Society of Anesthesiology (ASA) Physical Status Classification System: http://www.asahq.org/Home/For-Members/Clinical-Information/ASA-Physical-Status-Classification-System Accessed 30 Sep. 2012.
15. Goldman L, Caldera DL, Mussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med. 1977;297:845–50.
16. Garcia-Miguel FJ, Serrano-Aquilar PG, et al. Preoperative assessment. Lancet. 2003;362:1749–57.
17. Diaz JJ, Dutton WD, Ott MM, et al. Eastern Association for the Surgery of Trauma: a review of the management of the open abdomen—part2 "Management of the open abdomen". J Trauma. 2011;71:502–12.
18. Holt NF, Silverman DG, Prasad R, et al. Preanesthesia clinics, information management, and operating room delays: results of a survey of practicing anesthesiologists. Anesth Analg. 2007;104:615–8.
19. Fischer SP. Development and effectiveness of an anesthesia preoperative evaluation clinic in a teaching hospital. Anesthesiology. 1996;85:196–206.
20. Fischer SP. Cost-effective preoperative evaluation and testing. Chest. 1999;115:96S–100S.
21. Correll DJ, Bader AM, Hull MW, et al. Value of preoperative clinic visits in identifying issues with potential impact on operating room efficiency. Anesthesiology. 2006;105:1254–9; discussion 6A.
22. Trzepacz PT. Delirium. Advances in diagnosis, pathophysiology, and treatment. Psychiatr Clin North Am. 1996;19:429–48.
23. American Psychiatric Association. Practice guideline for the treatment of patients with delirium. Am J Psychiatry. 1999;156:1–20.
24. Marcantonio ER, Flacker JM, Michaels M, Resnick NM. Delirium is independently associated with poor functional recovery after hip fracture. J Am Geriatr Soc. 2000;48:618–24.
25. Sasajima Y, Sasajima T, Uchida H, Kawai S, Haga M, Akasaka N, et al. Postoperative delirium in patients with chronic lower limb ischaemia: what are the specific markers? Eur J Vasc Endovasc Surg. 2000;20:132–7.
26. Mullen JO, Mullen NL. Hip fracture mortality. A prospective, multifactorial study to predict and minimize death risk. Clin Orthop Relat Res. 1992;280:214–22.
27. Nightingale S, Holmes J, Mason J, House A. Psychiatric illness and mortality after hip fracture. Lancet. 2001;357:1264–5.
28. Inouye SK. The dilemma of delirium: clinical and research controversies regarding diagnosis and evaluation of delirium in hospitalized elderly medical patients. Am J Med. 1994;97:278–88.
29. Blacker DJ, Flemming KD, Link MJ, Brown Jr RD. The preoperative cerebrovascular consultation: common cerebrovascular questions before general or cardiac surgery. Mayo Clin Proc. 2004;79:223–9.
30. Wong GTC, Sun NCH. Providing perioperative care for patients with hip fractures. Osteoporos Int. 2010;21 Suppl 4:S547–53.
31. The brain trauma foundation: guidelines for the management of severe traumatic brain injury. 3rd edition: https://www.braintrauma.org/pdf/protected/Guidelines_Management_2007wbookmarks.pdf.
32. Poldermans D, Bax JJ, Boersma E, De Hert S, Eeckhout E, Fowkes G, et al. Task Force for Preoperative Cardiac Risk Assessment and Perioperative Cardiac Management in Non-cardiac Surgery of the ESC and endorsed by the ESA. Guidelines for pre-operative cardiac risk assessment and perioperative cardiac management in non-cardiac surgery: the Task Force for Preoperative Cardiac Risk Assessment and Perioperative Cardiac Management in Non-cardiac Surgery of the European Society of Cardiology (ESC) and endorsed by the European Society of Anaesthesiology (ESA). Eur Heart J. 2009;30:2769–812.
33. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook EF, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation. 1999;100:1043–9.
34. Halliburton B, Bell D, Preston J. AANA journal course: update for nurse anesthetists. Part 4: preoperative cardiac evaluation. AANA J. 2004;72:365–71.
35. Almanaseer Y, Mukherjee D, Kline-Rogers EM, Kesterson SK, Sonnad SS, Rogers B, et al. Implementation of the ACC/AHA guidelines for preoperative cardiac risk assessment in a general medicine preoperative clinic: improving efficiency and preserving outcomes. Cardiology. 2005;103:24–9.
36. Eagle KA, Berger PB, Calkins H, Chaitman BR, Ewy GA, Fleischmann KE, et al. ACC/AHA guideline update for perioperative cardiovascular evaluation for noncardiac surgery—executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery). J Am Coll Cardiol. 2002;39:542–53.
37. Devereaux PJ, Goldman L, Cook DJ, et al. Perioperative cardiac events in patients undergoing noncardiac surgery: a review of the magnitude of the problem, the pathophysiology of the events and methods to estimate and communicate risk. CMAJ. 2005;173(6):627–34.
38. Kertai MD, Boersma E, Bax JJ, Heijenbrok-Kal MH, Hunink MG, L'Talien GJ, et al. A meta-analysis comparing the prognostic accuracy of six diagnostic tests for predicting perioperative cardiac risk in patients undergoing major vascular surgery. Heart. 2003;89:1327–34.
39. Chobanian AV, Bakris GL, Black HR, et al. National Heart Lung, Blood Institute Joint National Committee on Prevention DE, Treatment of High Blood Pressure, National High Blood Pressure Education Program Coordinating Committee. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA. 2003;289:2560–72.
40. Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof EL, Fleischmann KE, et al. 2009 ACCF/AHA focused update on

perioperative beta blockade incorporated into the ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2009;120:e169–276.
41. Fleisher LA, Beckman JA, Brown KA, Calkins H, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary. Circulation. 2007; 116:1971–96.
42. Baue SM, Cayne NS, Veith FJ. New developments in the preoperative evaluation and perioperative management of coronary artery disease in patients undergoing vascular surgery. J Vasc Surg. 2010; 51(1):242–51.
43. Devereaux PJ, Xavier D, Pogue J, POISE (PeriOperative Ischemic Evaluation) Investigators, et al. Characteristics and short-term prognosis of perioperative myocardial infarction in patients undergoing noncardiac surgery: a cohort study. Ann Intern Med. 2011;154:523–8.
44. Hightower CE, Riedel BJ, Feig BW, Morris GS, Ensor Jr JE, Woodruff VD, et al. A pilot study evaluating predictors of postoperative outcomes after major abdominal surgery: physiological capacity compared with the ASA Physical Status Classification System. Br J Anaesth. 2010;104(4):465–71.
45. Wilson RJ, Davies S, Yates D, Redman J, et al. Impaired functional capacity is associated with all-cause mortality after major elective intra-abdominal surgery. Br J Anaesth. 2010;105(3):297–303.
46. Wohlauer MV, Sauaia A, Moore EE, et al. Acute kidney injury and posttrauma multiple organ failure: the canary in the coal mine. J Trauma. 2012;72(2):373–80.
47. Peralta R, Iribarne A, Manrique OJ, et al. Unique critical care issues related to trauma. In: Sheridan RL, editor. The trauma handbook of the Massachusetts General Hospital. Philadelphia: Lippincott Williams & Wilkins; 2004. p. 120–7.
48. Peralta R, Hojman H. Abdominal compartment syndrome. Int Anesthesiol Clin. 2001;39(1):75–94.
49. Latifi R, Gustafson M. Abdominal wall reconstruction in patients with enterocutaneous fistulas. J Trauma Emerg Surg. 2011;37:241–50.
50. Latifi R, Joseph B, Kulvatunyou N, Wynne JL, O'Keeffe T, Tang A, et al. Enterocutaneous fistulas and a hostile abdomen: reoperative surgical approaches. World J Surg. 2011;36(3):516–23.
51. Becker HP, Willms A, Schwab R. Small bowel fistulas and the open abdomen. Scand J Surg. 2007;96(4):263–71.
52. Giglio MT, Marucci M, Testini M, et al. Goal-directed haemodynamic therapy and gastrointestinal complications in major surgery: a meta-analysis of trandomized controlled trials. Br J Anaesth. 2009;103(5):637–46.
53. Marshall C, Maier RV, Jimenez M, Dellinger EP. Source control in the management of severe sepsis and septic shock: an evidence-based review. Crit Care Med. 2004;32(11):S513–26.
54. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345:1368–77.
55. Dellinger RP, Levy MM, Carlet JM, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med. 2008;36:1394–6.
56. Visschers RG, Olde Damink SW, Winkens B, et al. Treatment strategies in 135 consecutive patients with enterocutaneous fistulas. World J Surg. 2008;32(3):445–53.
57. Latifi R. Nutritional therapy in critically ill and injured patients. Surg Clin North Am. 2011;91(3):579–93.
58. Peralta R, Iribarne A, Manrique OJ, et al. Prevention strategies: thromboembolic complications, alcohol withdrawal, infection and gastrointestinal bleeding. In: Sheridan RL, editor. The trauma handbook of the Massachusetts General Hospital. Philadelphia: Lippincott Williams & Wilkins; 2004. p. 195–209.

6 Perioperative Radiologic Evaluation of Patients with Difficult Abdominal Wall Defects

Fahim Habib, Antonio C. Marttos Jr., and Bruno Monteiro T. Pereira

Introduction

The number of patients undergoing complex operative interventions for the surgical repair of abdominal wall defects has increased greatly over the last several years [1]. The basis for this near-exponential increase is the result of two key factors.

First, as a result of advancements in medical science, patients who were previously denied operative intervention because of comorbidities or severity of disease are now undergoing laparotomy. In a good portion of these patients, a damage control approach is often adopted [2, 3], with the creation of an open abdomen; many cannot be closed during the initial operation, resulting in a planned ventral hernia [4]. Even when primary closure is achieved, the proinflammatory milieu created by the severity of the underlying disease and the resultant malnutrition frequently lead to acute wound failure and the development of enterocutaneous fistulae. In addition, the use of stomas is frequent in this group.

Second, since the early 1990's, the use of more complex surgical techniques in reconstructing the abdominal wall have become increasingly popular [5–7]. Adoption of component separation techniques has allowed large defects to be closed primarily. This, coupled with the availability of a plethora of synthetic and biologic materials as adjuncts to support such complex repairs, has broadened the surgical options available for the repair of these defects [8].

F. Habib, MD, FACS
Division of Trauma Surgery, Broward Health Medical Center,
1600 South Andrews Ave, Ft Lauderdale, FL 33316, USA
e-mail: fahimhabib@icloud.com

A.C. Marttos Jr., MD (✉)
Department of Surgery, University of Miami,
1800 NW 10th Ave, Miami, FL 33133, USA
e-mail: amarttos@med.miami.edu

B.M.T. Pereira, MD, MSC
Division of Trauma Surgery, Department of Surgery,
University of Campinas, MMOC,
47-SI, Campinas 13025130, Brazil
e-mail: drbrunomonteiro@hotmail.com

The use of the appropriate radiologic imaging modality assists the surgeon in planning the surgical management of the patient with complex abdominal wall defects. Radiologic imaging can be used to establish a diagnosis, define the defect when this defect is not clinically apparent, characterize the condition of the various components of the abdominal wall, determine the presence and location of interloop intestinal fistulae, provide intraoperative guidance, detect postoperative complications, and identify recurrences.

Diagnosis

In the vast majority of patients, the diagnosis can be made with physical examination alone. Careful examination can reveal the defect, its margins, likely contents, reducibility, presence of associated fistulae or stoma, and the condition of the overlying skin. On occasion, the diagnosis might not be as readily apparent. This is most likely to be witnessed in patients with a large body habitus (Fig. 6.1a, b) and in patients with associated tenderness that precludes a thorough examination. Physical examination similarly might be inadequate in certain anatomical locations, such as the subxiphoid region, where divarication of the recti is difficult to distinguish from true herniation (Fig. 6.2). In such conditions, additional imaging modalities are warranted and include ultrasonography (US), computed tomography (CT), and, rarely, magnetic resonance imaging (MRI).

Ultrasonography

Ultrasonography is a noninvasive, easily performed, readily available, and relatively inexpensive modality. Its use in the diagnosis of abdominal wall hernias was first described by Spangen [9], and it has since been well validated [10–12]. Images are acquired using grayscale imaging and a high-frequency 5- or 7-MHz transducer. Imaging is performed in the supine and standing positions, both with and without the performance of a

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_6,

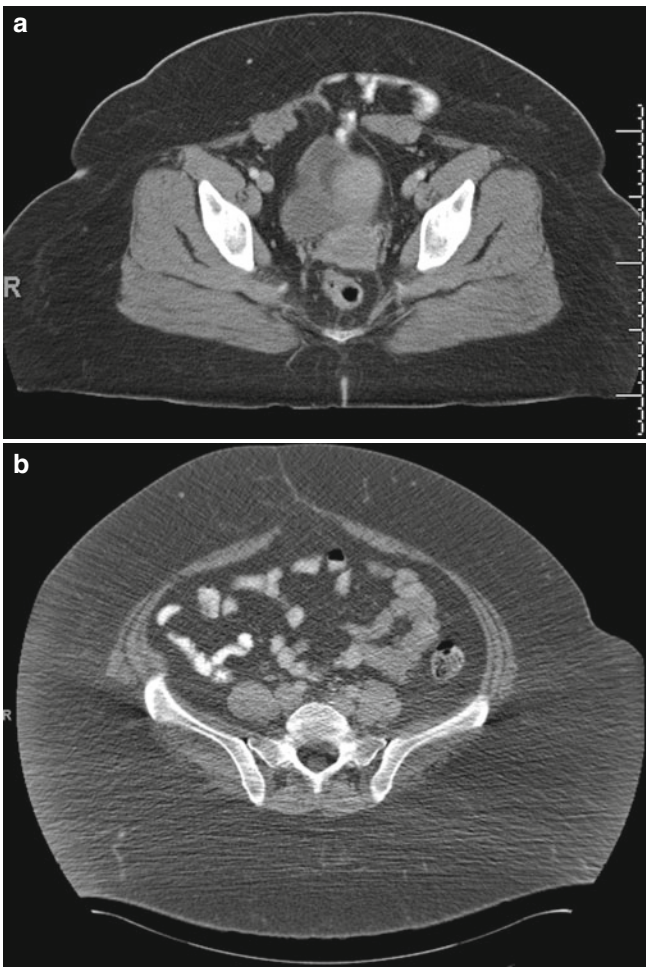


Fig. 6.1 (**a**, **b**) A small incisional hernia in patients with a large body habitus is difficult to diagnose on physical examination, but it is clearly seen on CT

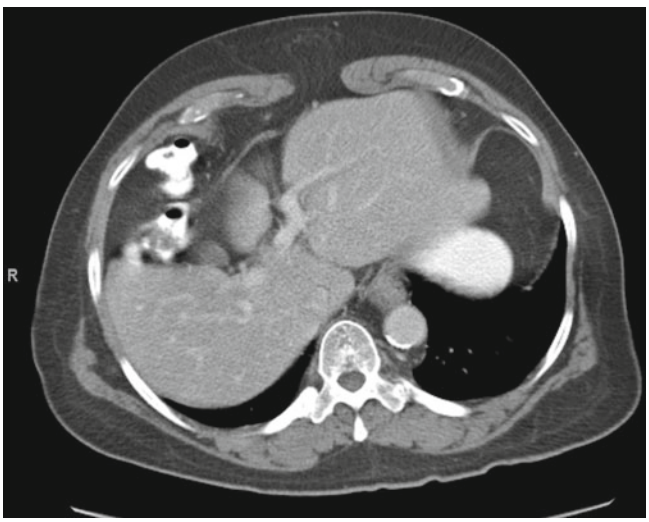


Fig. 6.2 Subxiphoid defect with herniation of omentum. A posterior component separation with retrorectus placement of the mesh will allow adequate superior overlap of at least 5 cm to reduce recurrence

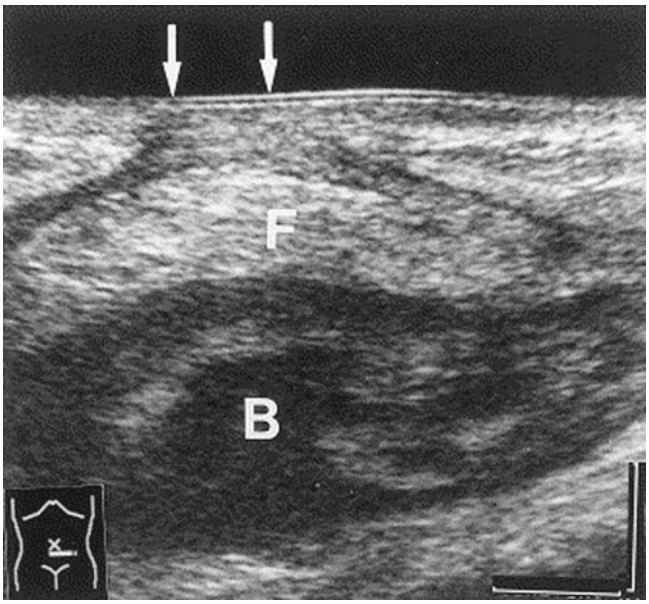


Fig. 6.3 Sonogram of an abdominal wall hernia in a postoperative patient. Transverse scanning of the lower abdomen identified a fascial defect (*arrow*), herniated bowel loop (*B*) and omental fat (*F*) along the linea alba. ———: 1 cm (Reprinted with permission of Elsevier from Ishida et al. [15])

Valsalva maneuver [13, 14]. Recent improvements in technology have resulted in notably improved images, with the dull gray abdominal wall muscles and "hyperechoic" bright fascia more easily visualized. The hernia defect can be appreciated as a discontinuity in the structures of the abdominal wall, potentially with abdominal contents herniating through the defect (Fig. 6.3). Use of real-time imaging allows the dynamic visualization of the abdominal muscles with the hernia contents seen traversing through the defect. The use of the Valsalva maneuver can further accentuate the herniation of contents, and it is especially useful when static imaging is equivocal and in certain anatomic locations, such as with a spigelian hernia [16]. Imaging can assist in the detection of additional defects, the presence of which might alter the operative plan or constitute a potential cause of recurrence. US can furthermore distinguish between hernias and other abdominal wall masses, such as tumors, seromas, hematomas, and abscesses. As US is operator dependent, close communication between the surgeon and sonographer is critical.

Computerized Scan

Multidetector row CT with reformatting is currently the ideal modality for establishing the diagnosis [17–20]. Axial imaging is performed in the supine position with thin (5-mm) slices. Intravenous contrast is administered if there is need to assess the vascular supply of the hernia contents. Oral contrast helps visualize bowel loops and is routinely

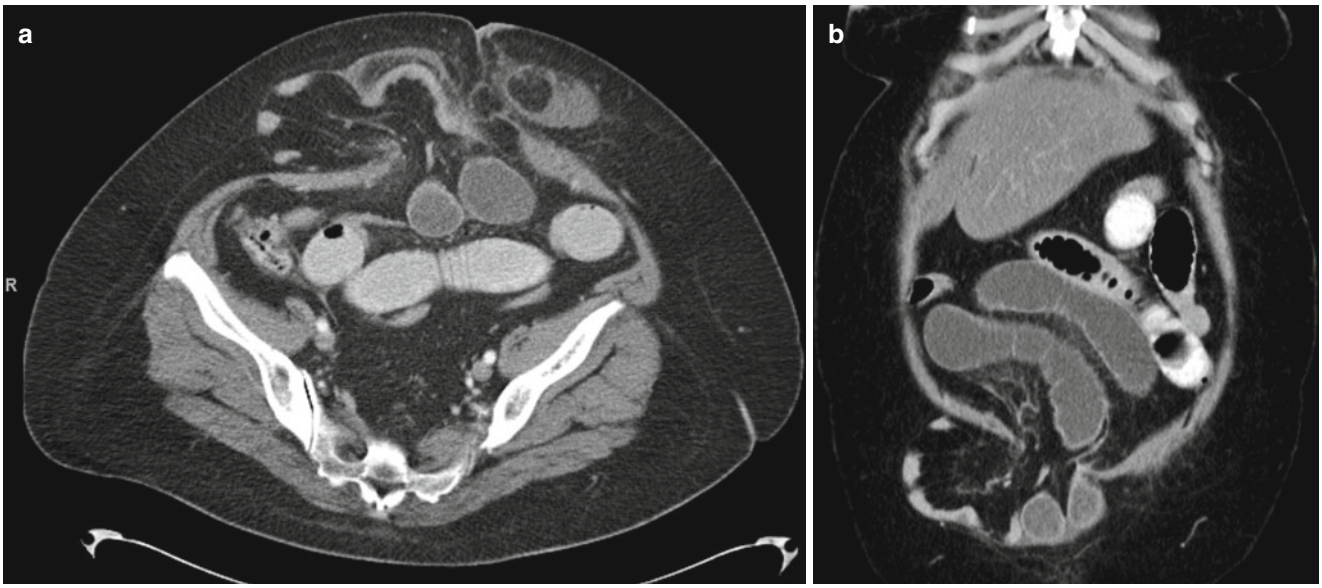


Fig. 6.4 (**a**, **b**) Differential caliber of bowel loops, which, in conjunction with inability to reduce the hernia on physical examination, indicates the presence of incarceration. Emergent operative intervention is indicated

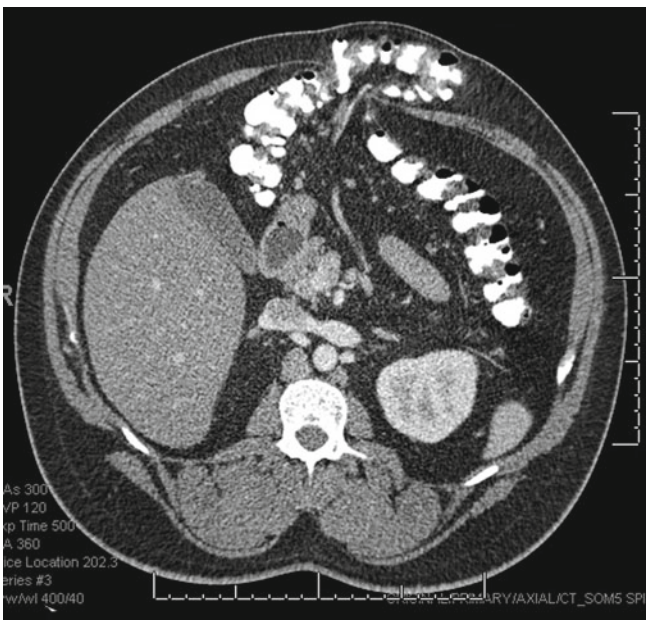


Fig. 6.5 Use of oral contrast allows determination of the caliber and quality of the bowel. There is no wall thickening or lack of contrast in the distal bowel, and the vascular supply to the segment of bowel appears intact. In conjunction with physical examination, these findings are comforting in that the bowel is not at risk, and an elective operation can be planned

administered in all cases. In subtle hernias, image acquisition is performed using the Valsalva maneuver. Multiplanar reformatting allows better appreciation of the anatomy in a manner more familiar to the surgeon. CT is especially useful in identifying hernias in unusual locations, such as with lumbar [21, 22], obturator [23], sciatic [24], and perineal hernias [25]; these are challenging to detect either on physical examination or with US. CT not only identifies the presence of a hernia but also allows for the detection of complications, including bowel obstruction, incarceration, and strangulation. Bowel obstruction is identified when the transition point is located at the level of the hernia, and the bowel proximal and distal to the hernia is dilated and decompressed, respectively. Although incarceration is a clinical diagnosis, the hernia contents have bearing on the timing of the operation. Presence of bowel in the incarcerated hernia mandates immediate operative intervention to prevent strangulation of the contents, especially if there is fluid within the hernia sac, thickening of the bowel wall, or luminal dilation. Strangulation is suggested by the presence of fluid-filled loops of bowel with proximal dilation, abnormal attenuation of the thickened abdominal wall, engorgement of the mesenteric vessels, mesenteric haziness, and ascites (Fig. 6.4a, b). In contrast, the absence of these findings on imaging and clinical examination indicates a low risk for incarceration and strangulation, allowing an elective approach to the hernia repair after optimization of the patient's general medical condition if necessary (Fig. 6.5).

Barium Studies with Small-Bowel Follow-Through

Barium studies with small-bowel follow-through study and barium enemas have been described as a useful diagnostic modality [26]. Diagnosis of a hernia is made when contrast-filled bowel loops are seen extending beyond the fascial planes of the anterior abdominal wall (Fig. 6.6). Reducibility is determined by manual compression of the

loops under fluoroscopy. Presence of obstruction can be identified by a difference in bowel caliber proximal and distal to the hernia and a failure to return the bowel loops to their normal position with manual reduction. Use of barium studies has largely been replaced by CT with oral or rectal contrast. Barium studies, however, might have utility in regions of the world with limited resources where CT might not be available.

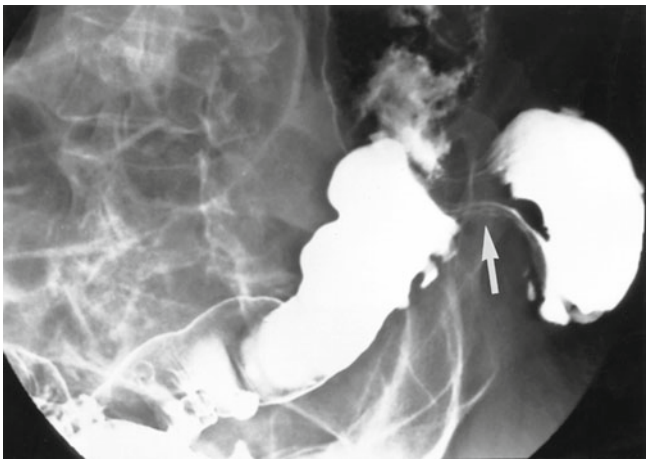


Fig. 6.6 Single-contrast barium enema demonstrating a short segment of herniated descending colon lying lateral to the iliac crest (Reprinted with permission of BMJ Publishing Group from Hide et al. [27])

Magnetic Resonance Imaging

MRI, similar to CT, allows delineation of the layers of the abdominal wall, highlighting the presence of the hernia and its contents (Fig. 6.7). However, MRI offers no particular advantage over CT and is not routinely obtained in making the diagnosis. Theoretically, MRI might be the preferred modality in the pregnant woman because of its favorable safety profile for the fetus.

Operative Planning Guided by Imaging Techniques

No imaging modality in isolation can guide selection of the operative intervention best suited for the individual patient. Imaging must be used in conjunction with a clinical assessment of the patient to select the operation that has the greatest likelihood of success. Of the various imaging modalities, CT has the greatest impact on decision making. The use of multiplanar reconstruction allows the anatomy of the defect and abdominal wall musculature to be better understood. It also allows for better conceptualization of the defect in three dimensions, giving the surgeon a mental image of the operative intervention required (Figs. 6.8a, b and 6.9). CT also visualizes the entire abdominal wall, allowing multiple

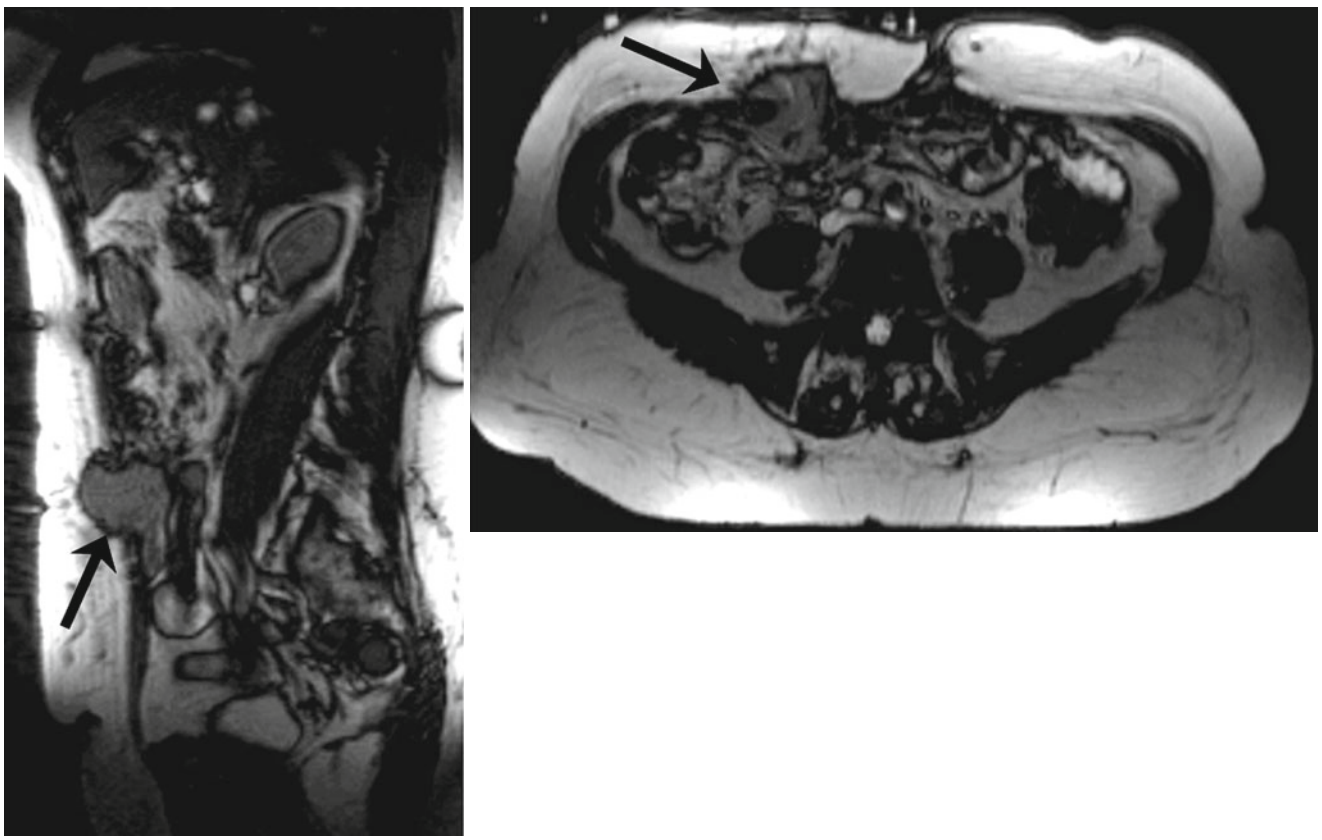


Fig. 6.7 Recurrence of a laparoscopically treated incisional hernia in the right abdominal wall (*arrows*) (Reprinted with kind permission of Springer Science + Business Media from Kirchhoff et al. [28])

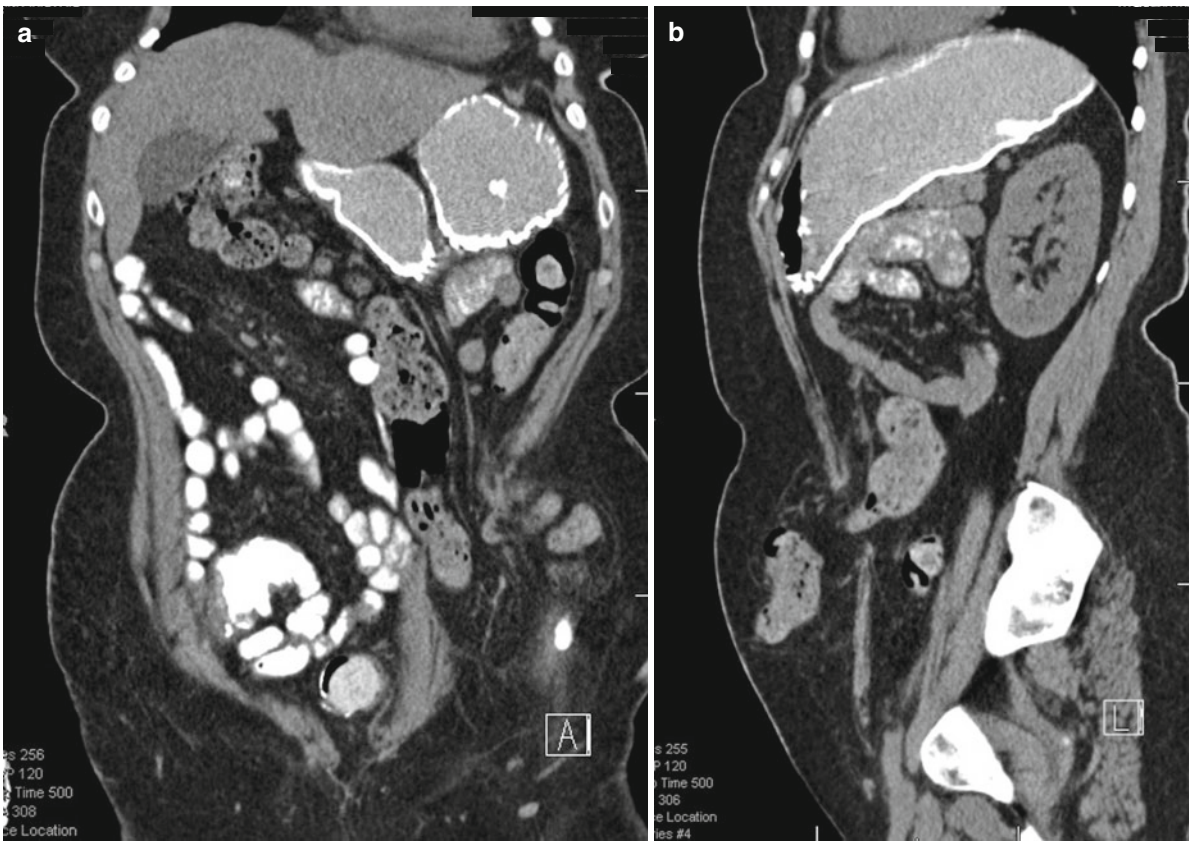


Fig. 6.8 (**a**, **b**) Multiplanar reconstruction allows the defect to be better understood in terms of surgeon familiarity

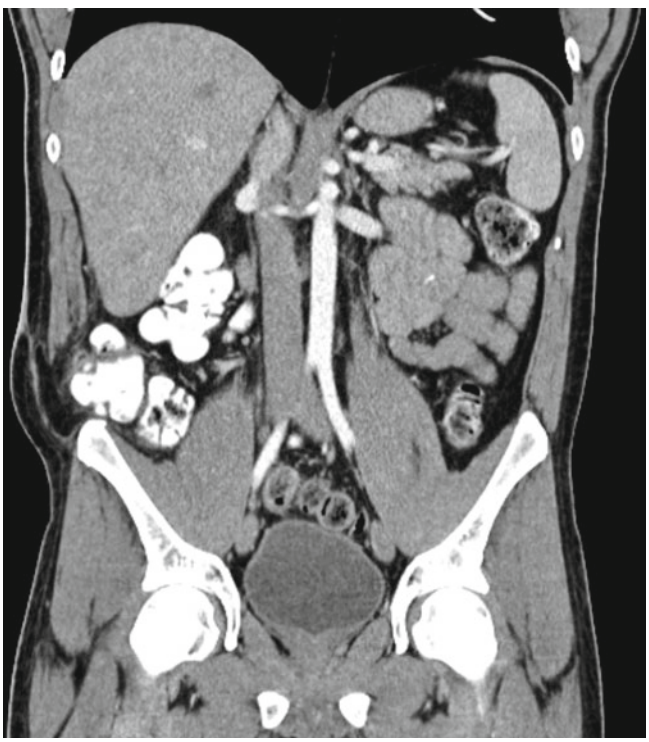


Fig. 6.9 Traumatic lumbar hernia following a motorcycle accident. The lateral musculature has been avulsed from the iliac crest. Repair requires access to the space between the transversalis fascia and the peritoneum. The mesh is allowed to drape well down into the pelvis and is secured to the iliac crest using tacks that will penetrate bone. No tacks are placed below the iliac crest for fear of injuring neurovascular structures

hernia defects to be identified (Fig. 6.10a–d). A failure to identify all defects present results in the selection of operative procedures that are less than ideal for the patient and increases the risk of hernia recurrence.

In giant ventral hernias, a large proportion of abdominal contents is contained in the hernia (Fig. 6.11a–c). Consequently, there is a reduction in the volume of the peritoneal cavity, resulting in a loss of domain. Returning the abdominal contents into the peritoneal cavity during hernia repair has significant physiologic consequences because of the development of an abdominal compartment syndrome with respiratory consequences, renal dysfunction, intestinal ischemia, and hemodynamic compromise. Although some studies described complex calculations to help target patients at risk [29] and others relied on a defect size greater than 10 cm in width as an indicator for recurrence [30], neither approach is clinically useful. The best current approach likely relies on using axial CT scan images to compare the contents of the native abdominal cavity with that in the hernia or "second abdomen." In giant hernias with over 50% of the contents located within the hernia sac, a progressive preoperative pneumoperitoneum is recommended [31].

A second important factor in decision making is the need for reapproximation of the musculature to create a dynamic functional abdominal wall. In the elderly, who typically lead a sedentary lifestyle with significant comorbidities, the use

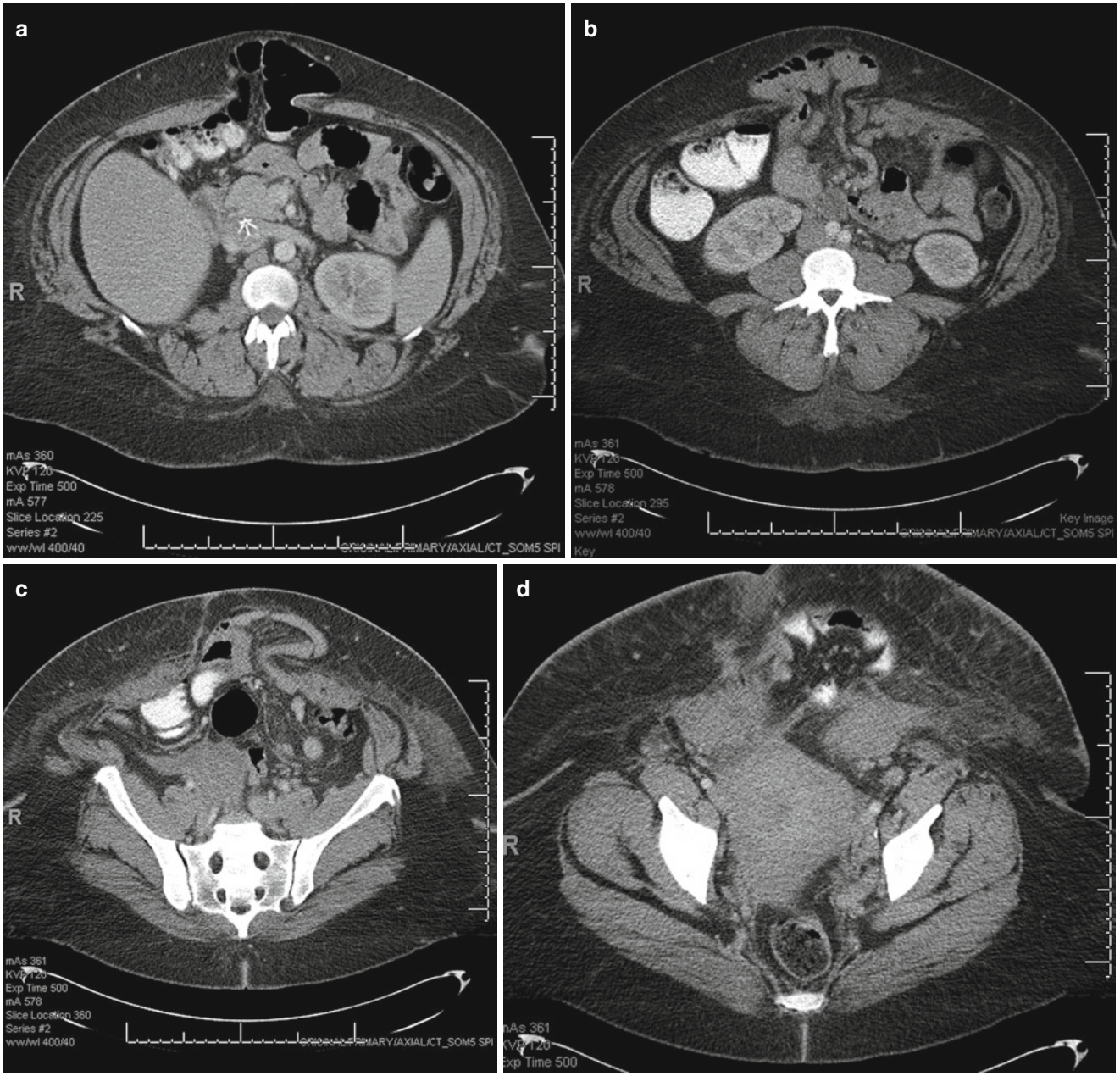


Fig. 6.10 (**a**–**d**) Multiple hernia defects along the entire length of the midline of the abdominal wall. There is an adequately sized rectus muscle and good lateral wall musculature. An endoscopic component separation of the external oblique aponeurosis with a retrorectus placement of the mesh is likely to have a high chance of success

of a mesh to cover the defect with adequate overlap via open or laparoscopic techniques is sufficient. Here, no additional analysis of the CT is necessary. In contrast, for patients in whom a dynamic abdominal wall is desirable, a critical assessment of the CT is essential. It is important to measure the size of the defect, the size and mass of the rectus, and the quality of the lateral abdominal wall musculature.

CT images allow the dimensions of the hernia defect to be accurately measured. We use the size of the hernia defect in its largest dimension as a guide to subsequent operative intervention when a dynamic abdominal wall with medicalization of the rectus muscles is desired. The decision regarding need for approximation of the musculature is made after considering the patient's general health status, functioning, and the need for a functional abdominal wall. In patients with significant underlying disease who would not tolerate an extensive reconstructive procedure and whose level of function and daily activities do not involve significant physical

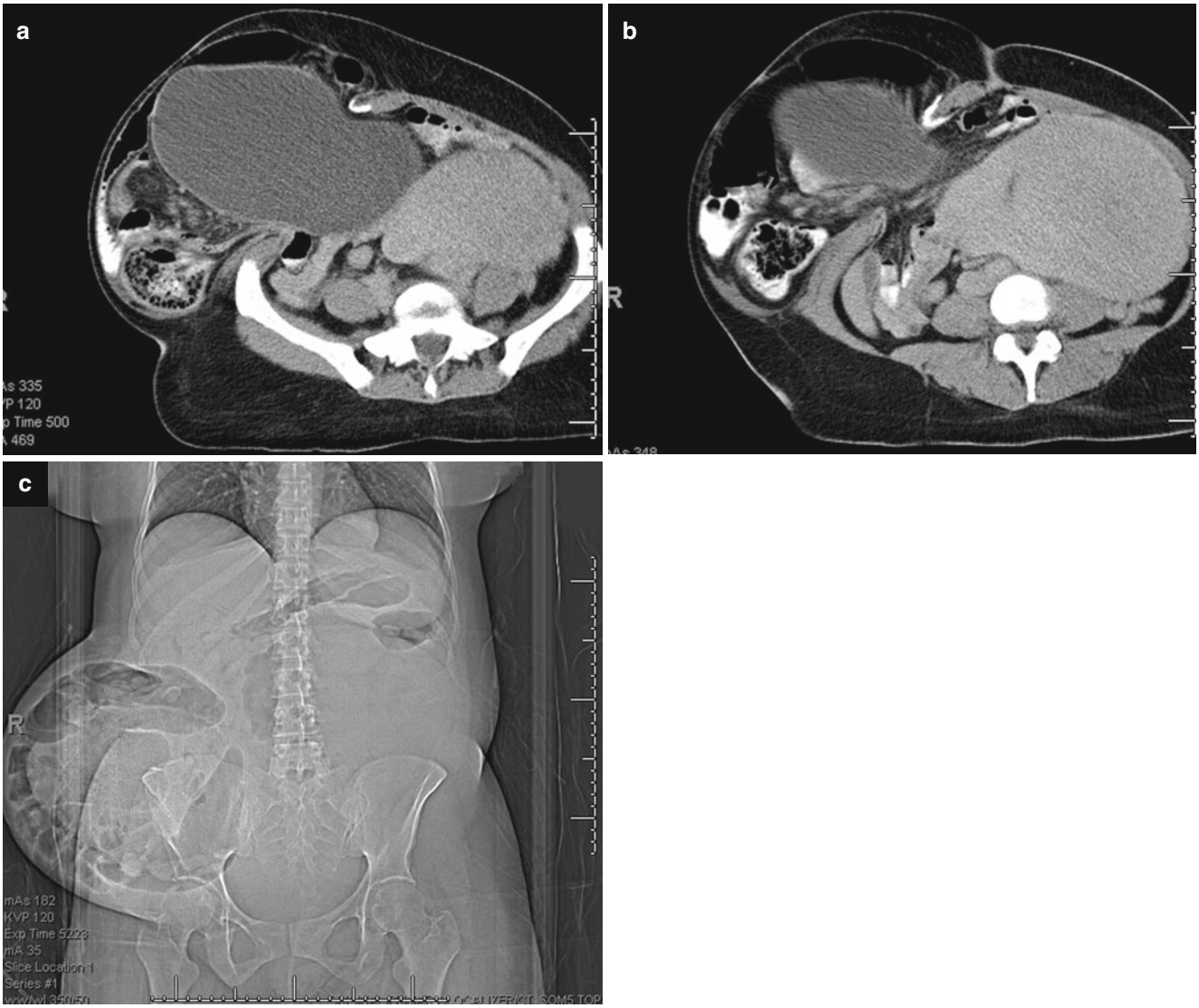


Fig. 6.11 (**a**–**c**) Location of over half the intra-abdominal contents in the hernia sac is highly suggestive of the need for a preoperative pneumoperitoneum

exertion, placement of a mesh in the intra-abdominal position with at least a 5-cm overlap beyond the edges of the hernia defect is generally adequate. However, there might be tension at the interface between the static mesh and the dynamic abdominal wall. Increased tension prior to incorporation of the mesh will result in disruption at the point of maximal stress, with resultant recurrence of the hernia (Fig. 6.12).

For defects with a size less than or equal to 6 cm, the hernia defect can almost always be closed primarily with reinforcement using a synthetic mesh [32] (Fig. 6.13). For hernia defects greater than 6 cm, release of myocutaneous flaps is performed to allow the muscles to come together in the midline. The nature of the myocutaneous flap procedure selected depends on the size and status of the abdominal wall musculature. If the rectus abdominus muscle is of adequate size, approximately 8 cm for an average size adult, component separation involving the external oblique muscles can be performed using open, minimally invasive, or endoscopic techniques (Fig. 6.14). If, despite adequate release of the external oblique, the defect cannot be closed, a posterior component separation is added. In contrast, if the rectus muscles are inadequate as a result of either previous operative intervention or fibrosis, the lateral musculature is evaluated. If adequate lateral musculature is present, a transversus abdominus release will allow for all but the largest of defects to be closed in the midline, supported in almost all cases by a synthetic or biologic prosthesis to potentially reduce recurrence rates. Large defects with a relatively inadequate rectus abdominis and lateral wall musculature suggest that the defect cannot be closed primarily. A bridging type of repair will most likely

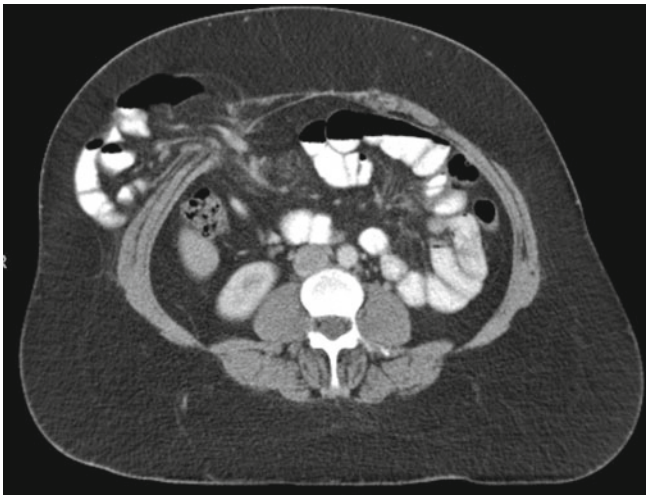


Fig. 6.12 Small hernia defect that can be repaired laparoscopically with primary closure of the defect using the "shoelacing" technique and subsequent reinforcing of the defect with a synthetic mesh

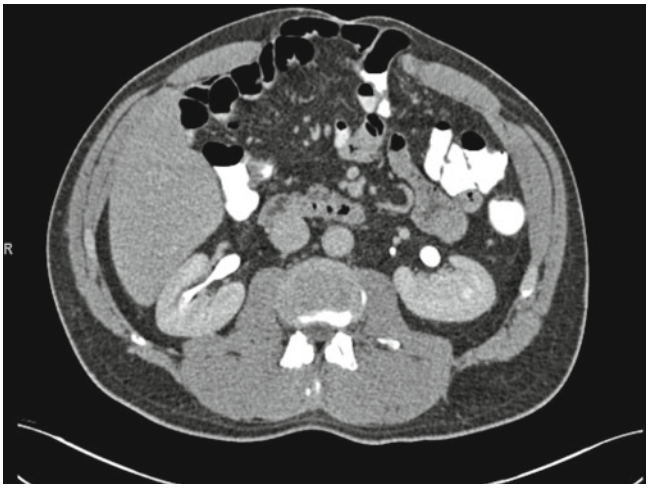


Fig. 6.14 A moderate midline defect with adequate residual abdominal wall musculature. CT findings suggest success with an endoscopic component separation of the external oblique aponeurosis with a retrorectus placement of the mesh

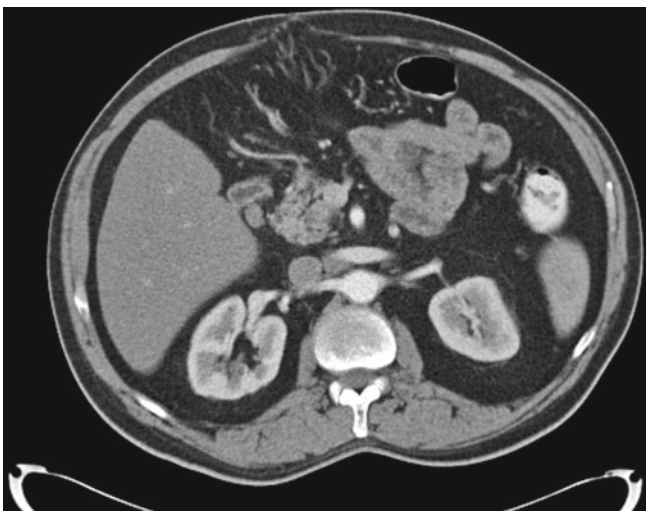


Fig. 6.13 The interaction of the adynamic mesh with the dynamic abdominal wall results in separation at the edge. The use of component separation with reapproximation of the musculature avoids this complication

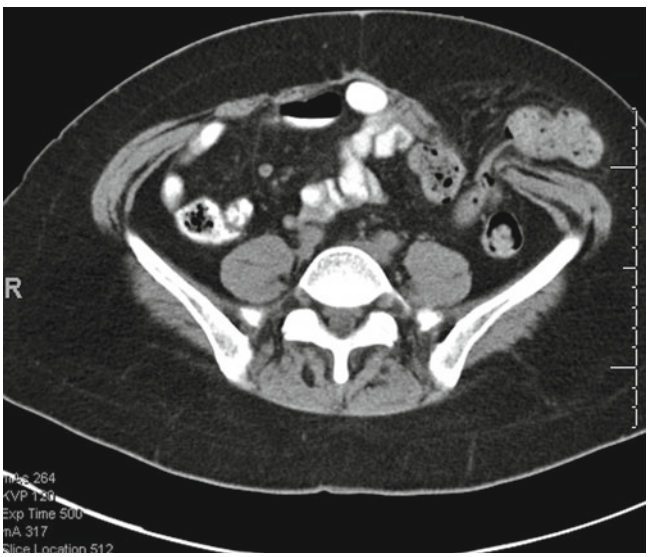


Fig. 6.15 Midline defect with associated parastomal hernia. The rectus muscles are relatively small with a disrupted left lateral wall musculature. Despite component separation, a bridging repair is likely and must be anticipated in setting patients'/surgeon expectations

be necessary, requiring the surgeon's and patient's expectations for the repair to be adjusted accordingly (Fig. 6.15). In patients who have undergone damage control laparotomy because of severity of the injury or surgical process, bowel edema coupled with loss of domain caused by fascial retraction precludes closure in a large proportion of patients. Here, the exposed bowel is covered by a split-thickness skin graft with a planned ventral hernia accepted in lieu of almost certain death. Repair of the resultant defect requires a careful analysis of the relative size of the defect and the available abdominal wall musculature. In cases of large defects with limited lateral wall musculature, the Fabian modification of the component separation is preferred (Fig. 6.16). In certain circumstances, the hernia defect might involve the lateral aspect of the abdominal wall. This might be seen following the creation of a stoma, as with parastomal herniations; laterally placed incisions, as with incisional herniations; and with injury as occurs following penetrating trauma or blunt rupture of the abdominal wall (Figs. 6.17 and 6.18).

Even in the presence of relatively large defects, the presence of redundancy of the lateral wall musculature indicates

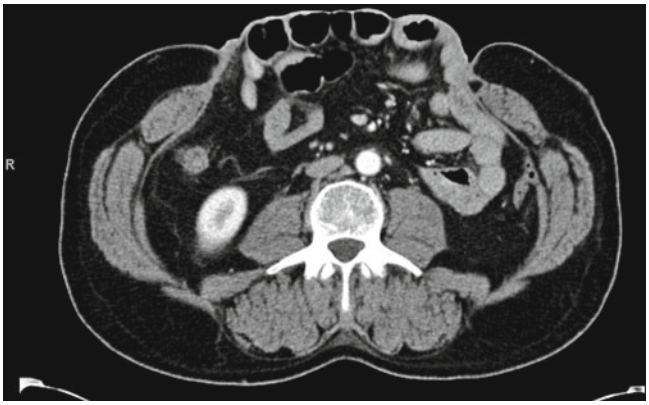


Fig. 6.16 A large defect with bowel covered by a skin graft. The Memphis modification of the component separation technique would be appropriate in this circumstance

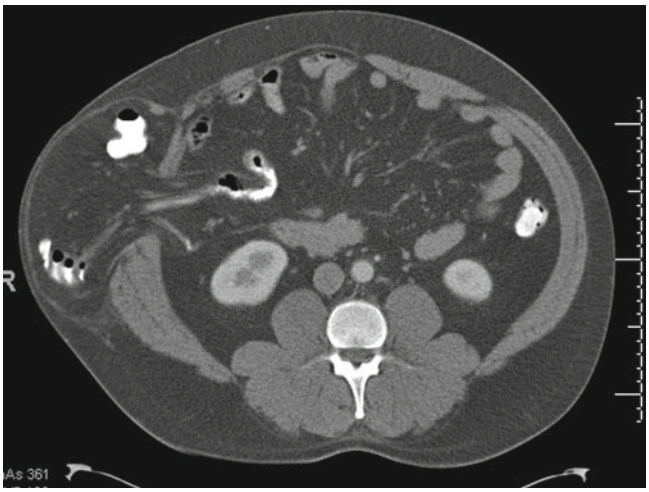


Fig. 6.17 Herniation at the site of a previous stab wound. A unilateral component separation on the affected site allows the defect to be closed with physiologic tension. Support with an intra-abdominal prosthesis further reduces the risk of recurrence

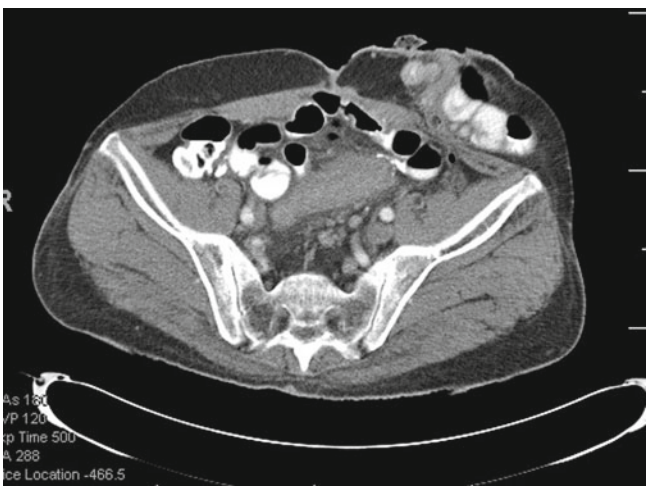


Fig. 6.18 A large parastoma hernia, the stoma, and resultant attenuation of the musculature on the affected side make repair challenging

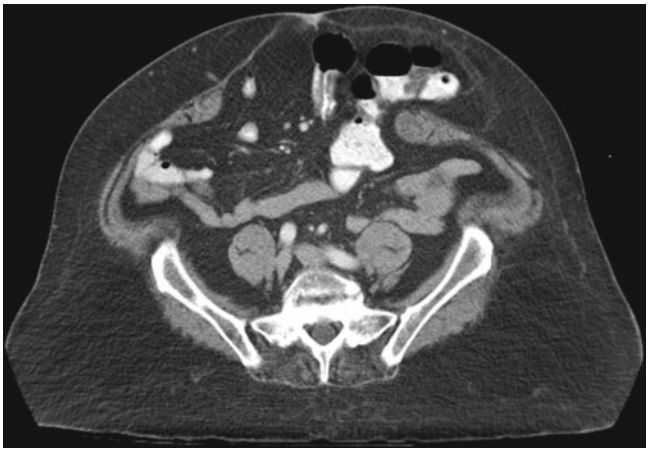


Fig. 6.19 Large abdominal wall defect with significant redundancy of the lateral abdominal wall musculature and moderate size rectus muscles. A transversus abdominus release will bring the musculature back in the midline

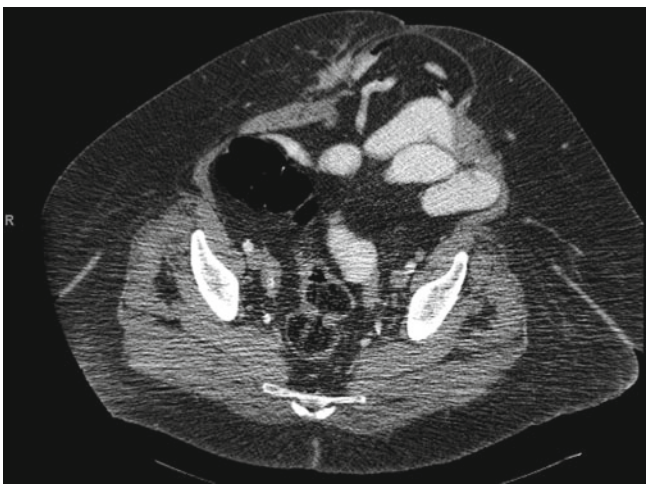


Fig. 6.20 Despite the large size of the hernia, buckling of the left lateral abdominal wall musculature suggests that a component separation will allow the defect to be closed primarily with additional reinforcement using a biologic scaffold

that, subsequent to a component separation procedure, the muscles can be stretched adequately, resulting in an ability to cover the defect (Figs. 6.19 and 6.20).

The CT scan also allows for the identification of the location of enterocutaneous fistulae, stoma, and the quantity and caliber of the bowel. In addition, the presence of an overt or occult parastomal hernia can be identified. These factors might significantly alter the operative plan. Specifically, component separation techniques will have to be altered when stoma or fistulae are present, and a significantly decreased degree of advancement is to be expected on the side of the ostomy (Fig. 6.21). CT accurately identifies the presence of undrained foci of

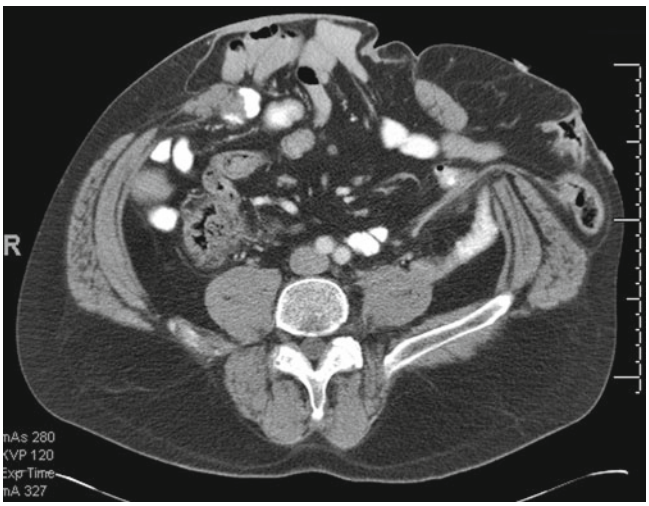


Fig. 6.21 Large incisional hernia with parastoma component indicating need for complex reconstruction with an inability to perform an adequate component separation of the left side; a transverse abdominus release with a bridging repair using a biologic scaffold is likely to yield the best results

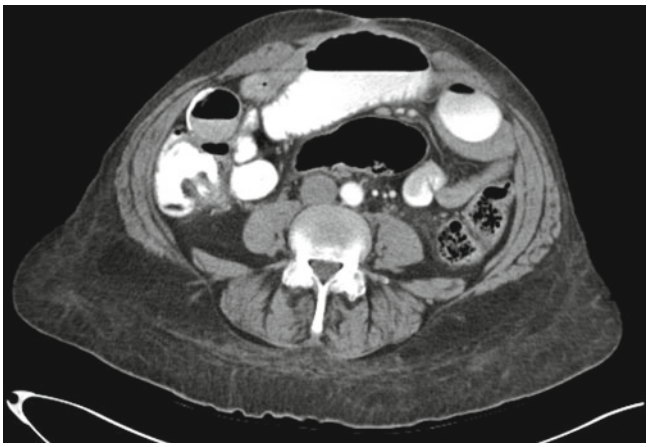


Fig. 6.22 Large abdominal wall defect with significant intra-abdominal contents. Abdominal wall reconstruction has a high risk of postoperative abdominal compartment syndrome. Mechanical bowel preparation reduces intraluminal contents and increases the space in the abdominal cavity

intra-abdominal collections, which must be addressed before definitive operative intervention is undertaken to avoid failure of the repair.

On CT, the relative size of the abdominal cavity and the hernia sac can be easily determined. If there is no indication for a preoperative pneumoperitoneum, the presence of a large amount of stool suggests the need to perform mechanical bowel prep prior to operative intervention. This will reduce the intra-abdominal pressure following reconstruction of the abdominal wall (Fig. 6.22).

Intraoperative Guidance

Complex abdominal wall defects result in significant distortion of the abdominal architecture. As a consequence, the linea semilunaris is often displaced laterally. This is even more challenging when this occurs in obese patients. When performing an endoscopic component separation, the initial incision is made at the tip of the 11th rib, with the intention of entering the space between the external and internal oblique muscles. The lateral displacement of the linea semilunaris might lead to accidental entry medial to the rectus sheath and balloon dissection of the incorrect plane. This might result in injury to the epigastric vessels with potentially significant hemorrhage. This complication can be bypassed by measuring the width of the rectus muscle on the preoperative CT scan and incising beyond the measured location of the linea semilunaris.

Alternatively, intraoperative ultrasonography can be utilized [33]. A 7.5-MHz transducer is used to image the abdominal wall, starting at the lateral edge of the hernia defect at about the level of the 11th rib. Scanning is performed medially to laterally and identifies the echogenic linea semilunaris and the subsequent decussating of the lateral abdominal wall musculature. Scanning can then be repeated at several points along the abdominal wall to trace the outline of the linea semilunaris. The initial incision for the endoscopic component can then be placed in the appropriate location.

Postoperative Radiologic Assessment

Postoperative complications are common following abdominal wall repair, especially when the hernia is large and the operative approach is complex. A large majority of these complications will require some intervention; hence, imaging is a crucial component of the assessment and management of postoperative problems.

Ultrasonography is useful in both the diagnosis and the management of postoperative complications. In addition, the examination can often be performed at the patient's bedside and repeated as often as necessary. On US, seromas appear as well-defined anechoic fluid collections. Although most seromas resolve spontaneously, those that persist beyond 6 weeks cause discomfort or are suspected to be infected are aspirated for therapeutic or diagnostic reasons.

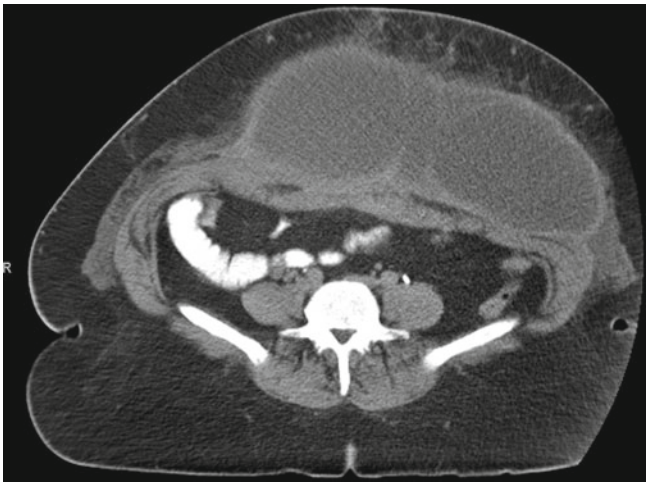


Fig. 6.23 Large seroma anterior to the reconstructed abdominal wall, causing pain. In addition, if left alone, this might result in pressure necrosis of the tissue. Percutaneous drainage with placement of a catheter is easily performed using ultrasound guidance

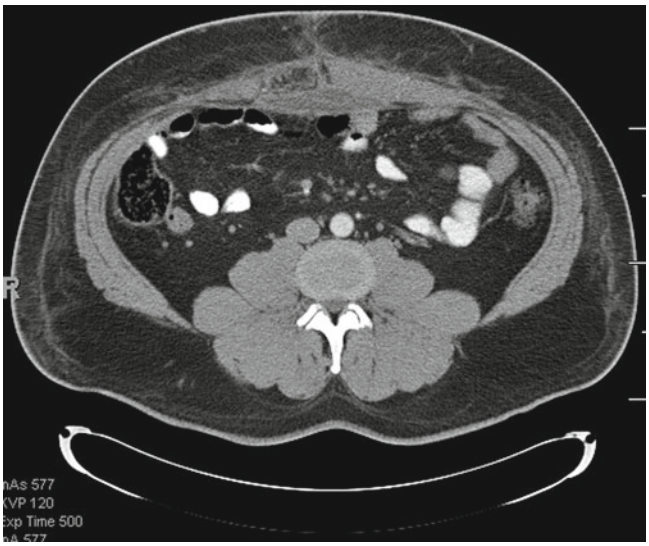


Fig. 6.24 Fluid collection between the reapproximated anterior abdominal wall and the biologic prosthesis placed in the retrorectus position. CT-guided drainage can be performed to evacuate the collection if symptomatic

The aspiration might be performed using US guidance (Fig. 6.23). Imaging might be challenging when the fluid collection is located beneath the mesh, where CT might be preferable (Figs. 6.24 and 6.25a). US can also potentially distinguish seromas from hematomas (Fig. 6.25b, c) from recurrence.

CT scanning is more expensive than US and exposes the patient to ionizing radiation, but it has the distinct advantage of demonstrating greater anatomic detail. It can easily distinguish between seromas, hematomas, and recurrence. The location of the collection can be defined even if it lies deep into the mesh. In addition, CT guidance can be used to evacuate the collections accurately. This becomes especially useful when the collection is located deep to the muscles or adjacent to critical structures and must be approached from unusual angles to avoid inadvertent injury. Inflammatory response to the implanted mesh can also be detected using CT. Irregular enhancement of the tissue surrounding the mesh is seen following the administration of intravenous contrast (Fig. 6.26). Localized fluid collections or air in the soft tissue, however, indicates mesh infection and the need for its removal. The presence of air-fluid levels indicates the likely presence of an abscess (Fig. 6.27a, b). In the past, this mandated removal of the mesh and the acceptance of a recurrence with plans for later reoperation. Current management varies with the type of mesh used. Biologic scaffolds are likely to disintegrate owing to the enzymatic activity of the bacteria and the resultant host inflammatory response. Continued drainage is usually adequate in these cases. Imaging must be performed again prior to drain removal to prevent recurrence of abscess because of incompletely drained collection. Among synthetic meshes, those composed of lightweight polypropylene can often be salvaged with drainage and long-term antibiotic therapy. Polyester mesh, on the other hand, poorly resists infection and often results in multiple draining sinuses. The presence of air in the tissue might suggest the diagnosis of a necrotizing infection requiring emergent intervention. Use of oral contrast will allow for detection of the dreaded complication of enterocutaneous fistula.

CT also remains a key imaging technique to distinguish these complications from that of a rectus sheath hematoma, which might be a consequence of intraoperative injury or from inadvertent injections into the inferior epigastric vessels. The hematoma appears on unenhanced images as a well-defined mass with high attenuation, and there is lack of enhancement with intravenous contrast. Further, the hematoma resolves over time with no specific treatment. Correction of any coagulopathy, analgesics, and warm compresses for comfort are all that is required.

Recurrence

Currently, recurrence remains the benchmark by which the success of complex abdominal wall reconstruction is measured. Both US and CT scan have uses. US can be used to follow patients serially on their postoperative visits to screen

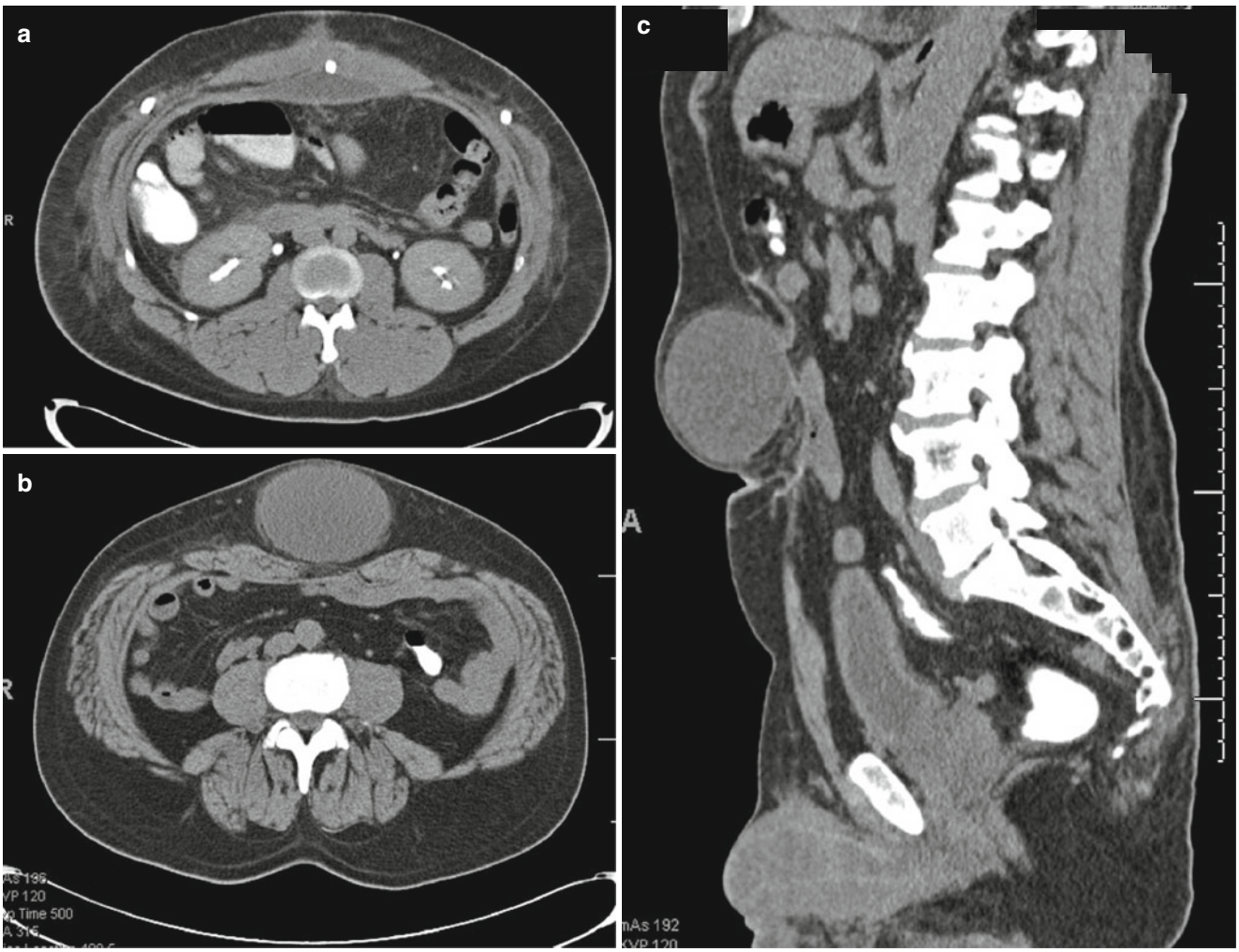


Fig. 6.25 (**a**–**c**) Retrorectus fluid collections are common despite prolonged drainage of the space using closed suction drains. If asymptomatic, they are best left alone, and they resorb over time

for asymptomatic recurrences. In questionable cases, real-time US with performance of the Valsalva maneuver can identify recurrences that might not be otherwise detected. In the majority of cases, CT scan remains the mainstay for the diagnosis of a recurrence of postoperative defects (Fig. 6.28a, b). The incidence of recurrence is influenced by the imaging modality used and the rigor with which its presence is sought. Mesh bulge, seromas (Fig. 6.29), hematomas, and retained hernia contents might result in pseudorecurrences [34]. The characteristics of the recurrent hernia are then used to determine the optimal approach to its repair. Following complex abdominal wall reconstruction, the majority of recurrent hernias are small defects found most often at the edges of the original repair where the static mesh interfaces with the dynamic abdominal wall (Fig. 6.30). Reconstructing the abdominal wall with approximation of the abdominal wall musculature at the original operation can prevent this. When a bridging technique is used, herniation can occur through the central portion of mesh if a synthetic mesh has been employed [35]. This is increasingly more common when lightweight meshes are used. When biologic scaffolds, either human acellular dermis or porcine dermis, are used, there might be progressive bulging of the scaffold [36, 37] (Fig. 6.31). While truly not a recurrent hernia, intra-abdominal contents migrate into the new bioprosthesis and bulge, producing discomfort and impairing the patient's ability to generate adequate intra-abdominal pressure for physiologic activities such as defecation and micturition. The resultant bulge is also cosmetically displeasing. The presence of these features must be considered when deciding to proceed with re-repair.

When the ideal operation is selected as indicated by patient factors such as underlying disease and comorbidities, radiologic imaging can guide selection of the ideal procedure, resulting in optimal outcome with long-term success rates (Fig. 6.32).

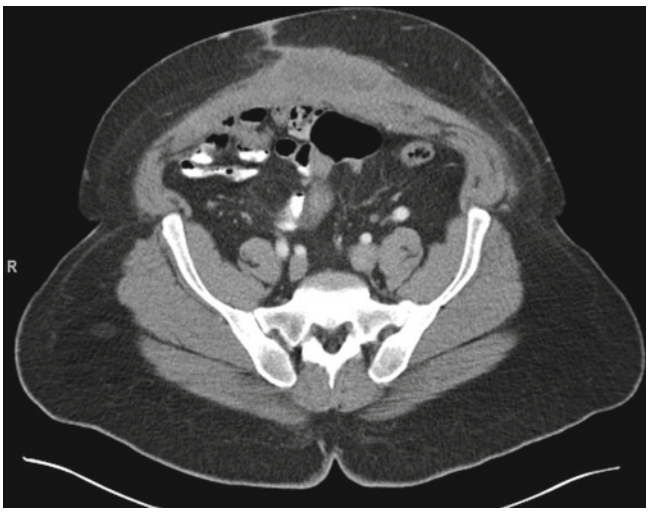


Fig. 6.26 Inflammatory changes without localized collections representing postsurgical changes and reaction to the prosthesis used

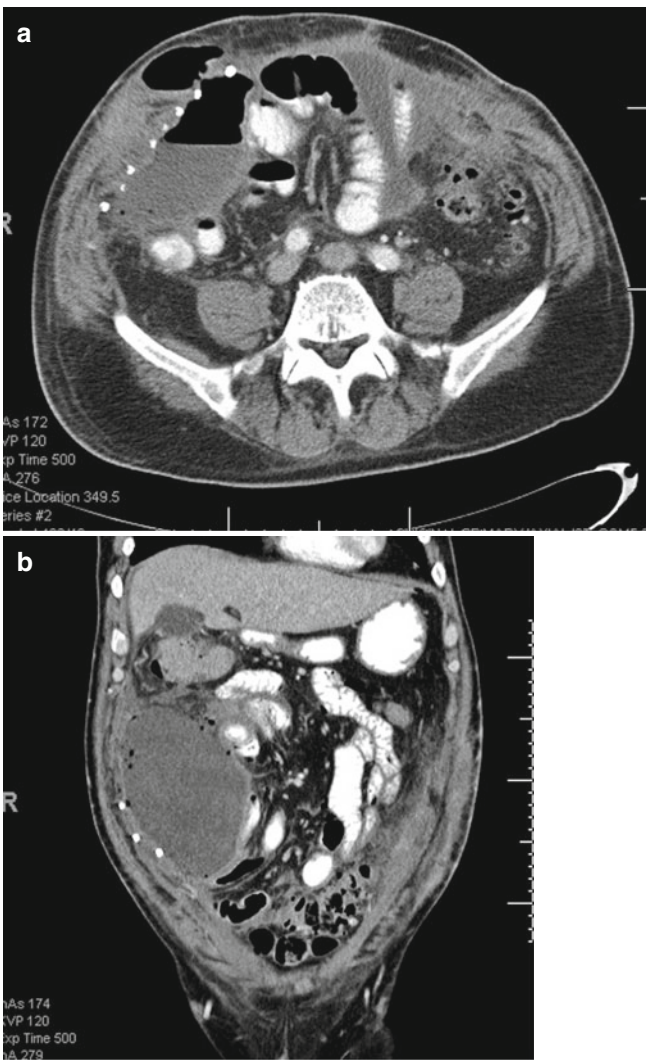


Fig. 6.27 (**a**, **b**) Large fluid collection on either side of the mesh with radiopaque tacks indicating the location of the mesh. Air within the collection suggests that the collection is likely an abscess. CT-guided drainage of the collection will be necessary to drain the abscess and obtain fluid for microbiologic evaluation. Lightweight polypropylene mesh can often be salvaged with drainage and long-term antibiotics

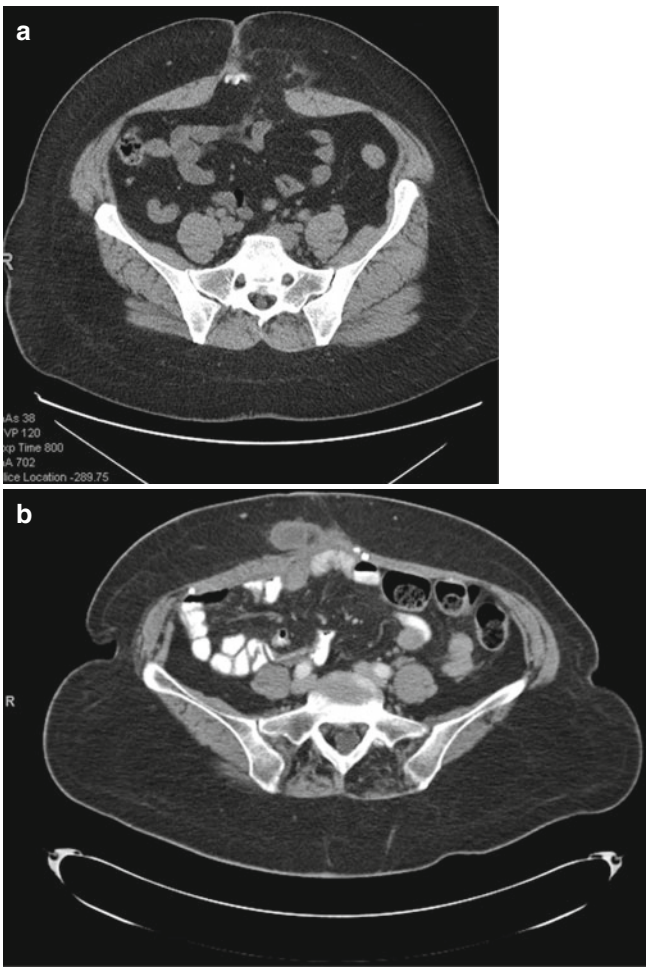


Fig. 6.28 (**a**, **b**) Recurrence at the edge of the previous repair. The small size of the defect lends itself to primary laparoscopic closure of the defect with intra-abdominal placement of synthetic mesh

Summary

The incidence of complex abdominal wall defects is only expected to increase as patients who are more debilitated and surgically complex undergo laparotomy, survive their primary abdominal catastrophes, and necessitate repair of the resultant defects. Radiologic imaging plays an instrumental role in nearly every aspect of the assessment and surgical management of these patients. Key among the imaging modalities are US and CT. Either of these can be used to make the diagnosis, monitor and treat postoperative complications, and detect the presence of recurrence. US also can be used to locate the displaced linea semilunaris while performing an endoscopic component separation procedure. Although ultrasonography avoids exposure to ionizing radiation, CT offers greater anatomic detail, allows the vascularity to be assessed, and reveals the state of the bowel, including the presence and location of the bowel, fistulae, and stomas. Radiological imaging using CT or US is hence of paramount importance in the evaluation and management of patients with complex abdominal wall defects.

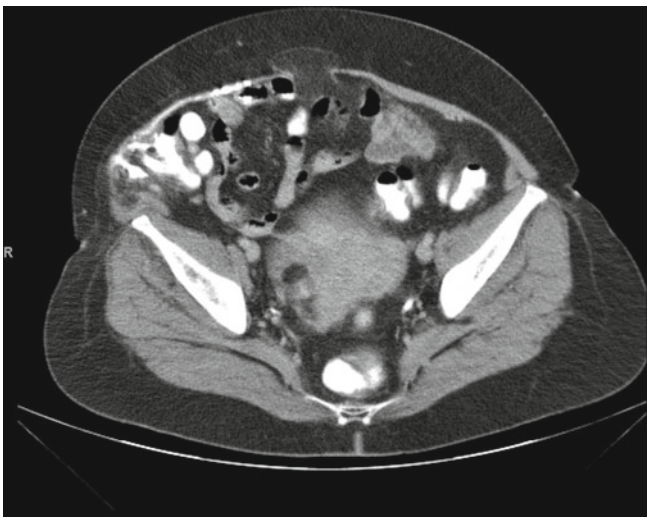


Fig. 6.29 Development of a second hernia at the medial edge of the mesh placed for the repair of the initial hernia. The defect is small and can be approached laparoscopically

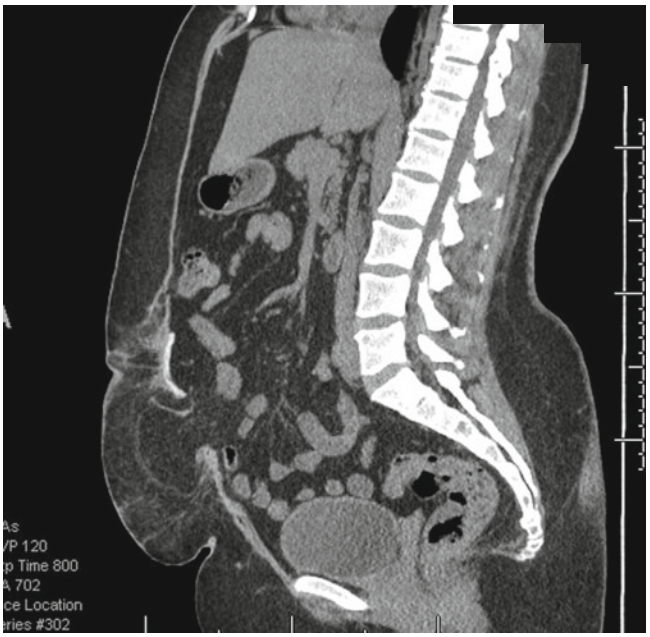


Fig. 6.30 Small seroma anterior to an intact repair. CT helps differentiate this not-uncommon postoperative occurrence from recurrence

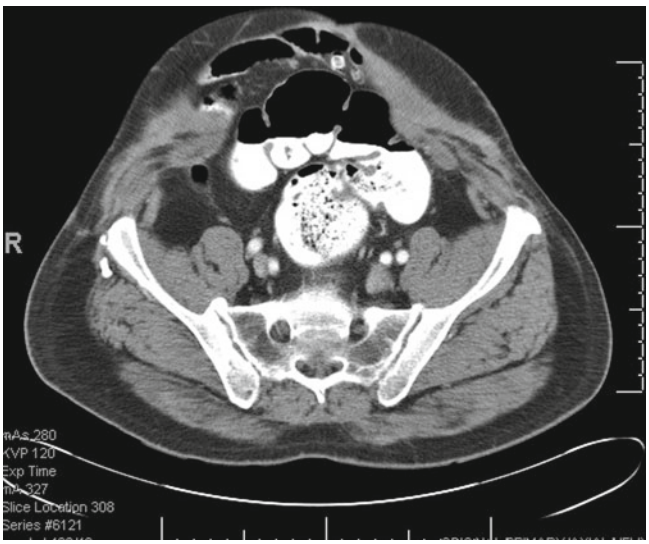


Fig. 6.31 Bulge of the biologic scaffold without actual herniation of abdominal contents in a bridging repair. Although it might cause discomfort and give the appearance of a recurrence, there is no risk of incarceration or strangulation, so this might be an acceptable outcome in most patients, considering the size and nature of the initial defect

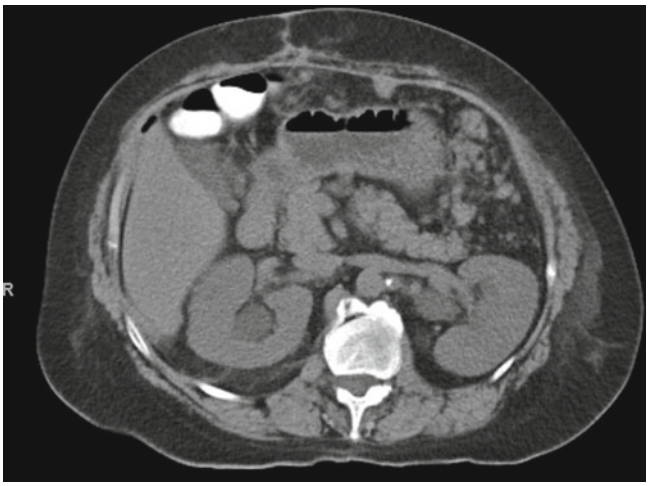


Fig. 6.32 Excellent results with component separation and use of a biologic scaffold at scheduled postoperative imaging of two tears following repair

References

1. Clarke TM, Goldberg RF, Lloyd JM, Rosales-Velderrain A, Bowers SP. The increasing utilization of component separation technique during ventral hernia repair: association of patient, payor, and community demographics. SAGES 2012 Annual Meeting, San Diego, CA. 2012.
2. Hatch QM, Osterhout LM, Ashraf A, Podbielski J, Kozar RA, Wade CE, et al. Current useo of damage-control laparotomy, closure rates, and predicotrs of early fascial closure at the Forst takeback. J Trauma. 2011;70(6):1429–36.
3. Subramanian A, Balentine C, Palacio CH, Sansgiry S, Berger DH, Awad SS. Outcomes of damage-control celiotomy in elderly trauma patients with intra-abdominal catastrophes. Am J Surg. 2010;200(6):783–8.
4. Leppaniemi A, Tukiainen E. Planned hernia repair and late abdominal wall reconstruction. World J Surg. 2012;36(3):511–5.

5. Ramirez OM, Ruas E, Dellon AL. Components separation method of closure of abdominal-wall defects: and anatomic and clinical study. Plast Reconstr Surg. 1990;86(3):519–26.
6. van Geffen HJ, Simmermacher RK, Bosscha K, van der Werken C, Hillen B. Anatomical considerations for the surgery of the anterolateral abdominal wall. Hernia. 2004;8(2):93–7.
7. DiCocco JM, Fabian TC, Emmett KP, Magnotti LG, Goldberg SP, Croce MA. Components separation for abdominal wall reconstruction: the Memphis modification. Surgery. 2012;151(1):118–25.
8. Bachman S, Ramshaw B. Prosthetic material in ventral hernia repair: how do I choose? Surg Clin North Am. 2008;88(1):101–12.
9. Spangen L. Ultrasound as a diagnostic aid in ventral abdominal hernia. J Clin Ultrasound. 1975;3(3):211–3.
10. Young J, Gilbert AI, Graham MF. The use of ultrasound in the diagnosis of abdominal wall hernias. Hernia. 2007;11:347–51.
11. Corsale I, Palladino E. Diagnosis and treatment of epigastric hernia. Minerva Chir. 2000;55(9):607–10.
12. Arregui ME. The value of ultrasound in the diagnosis of hernia. In: Arregui ME, Nagan NF, editors. Inguinal hernia: advances or controversies? New York: Radcliffe Medical Press; 1994.
13. Emby DJ, Aoun G. Valsalva's maneuver in abdominal wall hernia imaging. Am J Roentgenol. 2005;185(4):1081–2.
14. Stavros AT, Rapp C. Dynamic ultrasound of hernias of the groin and anterior abdominal wall. Ultrasound Q. 2010;26(3):135–69.
15. Ishida H, Konno K, Hamashima Y, Naganuma H, Komatsuda T, Sato M, et al. Anterior abdominal wall pathologies detected by high-frequency annular array. Eur J Ultrasound. 1998;7(3):167–74.
16. Losanoff J, Kjossev K, Handijev S, Karamfilova R. The diagnosis of spigelian hernia (SH) by high-resolution real-time sonography. J Ultrasound Med. 1998;17(9):599–600.
17. Aguirre DA, Casola G, Sirlin C. Abdominal wall hernias: MDCT findings. AJR Am J Roentgenol. 2004;161:681–90.
18. Lee GH, Cohen AJ. CT imaging of abdominal hernias. AJR Am J Roentgenol. 1993;148:1209–13.
19. Ghahremani GG, Jiminez MA, Rosenfield M, Rochester D. CT diagnosis of occult incisional hernias. AJR Am J Roentgenol. 1987;148:139–42.
20. Aguirre DA, Santosa AC, Casola G, Sirlin CB. Abdominal wall hernias: imaging, features, complications, and diagnostic pitfall at multi-detector row CT. Radiographics. 2005;2:1501–20.
21. Baker ME, Weinerth JL, Andriani RT, Cohan RH, Dunnick NR. Lumbar hernia: diagnosis by CT. AJR Am J Roentgenol. 1987;148(3):565–7.
22. Farso SH, Racette CD, Lally JF, Willis JS, Mansoory A. Traumatic lumbar hernia: CT diagnosis. AJR Am J Roentgenol. 1990; 154(4):757–9.
23. Stamatiou D, Skandalakis LJ, Zoras O, Mirilas P. Obturator hernia revisited: surgical anatomy, embryology, diagnosis, and technique of repair. Am Surg. 2011;77(9):1147–57.
24. Ghahremani GG, Michael AS. Sciatic hernia with incarcerated ileum: CT and radiographic diagnosis. Gastrointest Radiol. 1991; 16(2):120–2.
25. Lubat E, Gordon RB, Birnbaum BA, Megibow AJ. CT diagnosis of posterior perineal hernia. AJR Am J Roentgenol. 1990;154(4): 761–2.
26. Zafar HM, Levine MS, Rubesin SE, Laufer I. Anterior abdominal wall hernias: findings in barium studies. Radiographics. 2006; 26:691–9.
27. Hide IG, Pike EE, Uberoi R. Lumbar hernia: a rare cause of large bowel obstruction. Postgrad Med J. 1999;75(882):231–2.
28. Kirchhoff S, Ladurner R, Kirchhoff C, Mussack T, Reiser MF, Lienemann A. Detection of recurrent hernia and intraabdominal adhesions following incisional hernia repair: a functional cine MRI-study. Abdom Imaging. 2010;35(2):224–31.
29. Tanaks EY, Yoo JH, Rodriguez AJ, Utiyama EM, Birolini D, Rasslan S. A computerized tomography scan method for calculating the hernia sac and abdominal cavity volume in complex large incisional hernia with loss of domain. Hernia. 2010; 14:63–9.
30. Sabbagh C, Dumont F, Fuks D, Yzet T, Verhaeghe P, Regimbeau JM. Progressive preoperative pneumoperitoneum preparation (the Goni Moreno protocol) prior to large incisional hernia surgery: volumetric, respiratory and clinical impacts. A prospective study. Hernia. 2012;16:33–40.
31. Mcadory RS, Cobb WS, Carbonell AM. Progressive preoperative pneumoperitoneum for hernias with loss of domain. Am Surg. 2009;75(6):504–9.
32. Orenstein SB, Dumeer JL, Monteagudo J, Novitsy MJ, Poi YW. Outcomes of laparoscopic ventral hernia repair with routine defect closure using "shoelacing" technique. Surg Endosc. 2011;25(5): 1452–7.
33. Vu T, Habib F. Utility of ultrasounds in locating the linea semilunaris in the endoscopic component separation technique in ventral hernia repair. Ann R Coll Surg Engl. 2011;93(7):553.
34. Tse GH, Duckworth BM, Stulchfield AD, de Beaux AC, Tulloh B. Pseudo-recurrence following laparoscopic ventral and incisional hernia repair. Hernia. 2010;14(6):583–7.
35. Langer C, Neufang T, Liersch C, Kley T, Becker H. Central mesh recurrence after hernia repair with Marlex—are the meshes strong enough? Hernia. 2001;5(3):164–7.
36. Nahabedian MY. Does AlloDerm stretch? Plast Reconstr Surg. 2007;120(5):1276–80.
37. Blatnik J, Jin J, Rosen M. Abdominal hernia repair with bridging acellular dermal matrix—an expensive hernia sac. Am J Surg. 2008;196(1):47–50.

A Difficult Abdomen: Clinical Course-Based Management

7

Guillermo Higa and Rifat Latifi

Anatomy

A key aspect of repairing complex defects is understanding the anatomy of the abdominal wall. The lateral abdominal wall fasciae and musculature derive their blood supply primarily from the intercostal arteries, lumbar arteries, and deep epigastric arteries (Fig. 7.1). The innervations come from the seventh to the twelfth intercostals and the first lumbar nerves (Fig. 7.2). Those intercostals and the lumbar vessels and nerves travel from the posterior midline to the anterior midline in an oblique, anterior pathway between the internal oblique and transversalis muscles (Fig. 7.3). The vasculature and innervations to the rectus abdominis muscle follow this same pathway. Vertical incisions in the abdominal wall musculature can disrupt both the vasculature and the innervations to the external oblique, internal oblique, transversalis, and rectus abdominis muscles. A transverse incision at the costovertebral margin through the external oblique fascia avoids the major vessels and nerves to the abdominal wall and allows for blunt dissection between

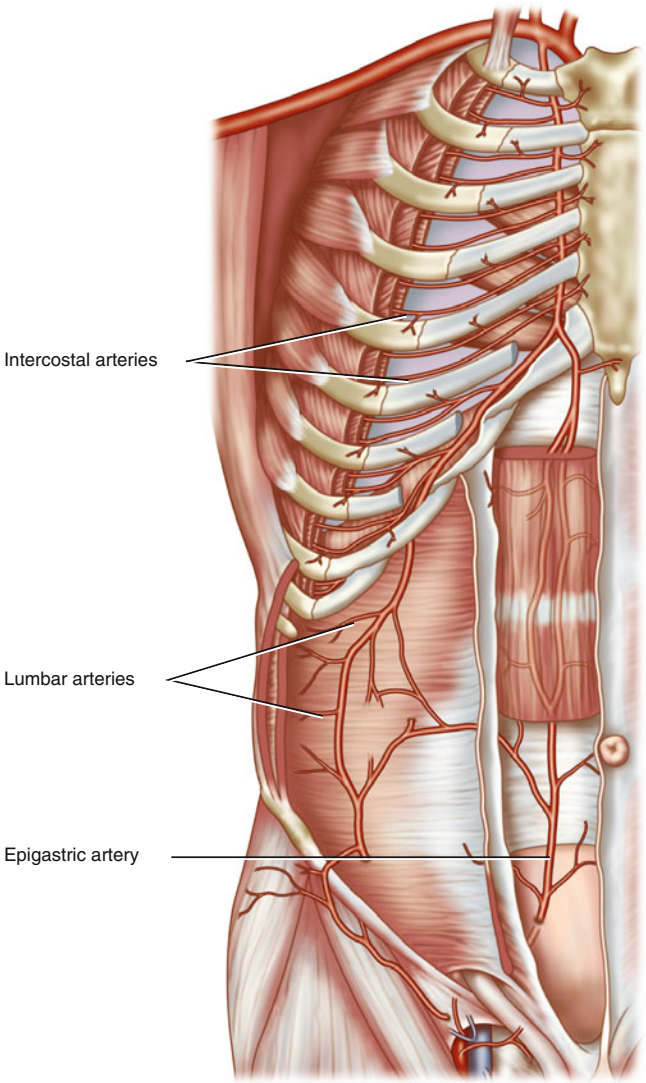


Fig. 7.1 Anatomy of the abdominal wall. The lateral abdominal wall fasciae and musculature derive their blood supply primarily from the intercostal arteries, lumbar artieries, and deep epigastric arteries

G. Higa, MD
Department of Surgery,
Willamette Valley Medical Center,
1663 NW Medinan Drive, McMinnville,
OR 97128, USA
e-mail: guillermo.higa@capellahealth.org

R. Latifi, MD, FACS (✉)
Department of Surgery,
The University of Arizona,
1501 N. Campbell Ave, Tucson,
AZ 85724, USA

Trauma Section, Department of Surgery,
Weill Cornell Medical College,
Hamad General Hospital, Doha, Qatar
e-mail: latifi@surgery.arizona.edu

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_7, © Springer Science+Business Media New York 2013

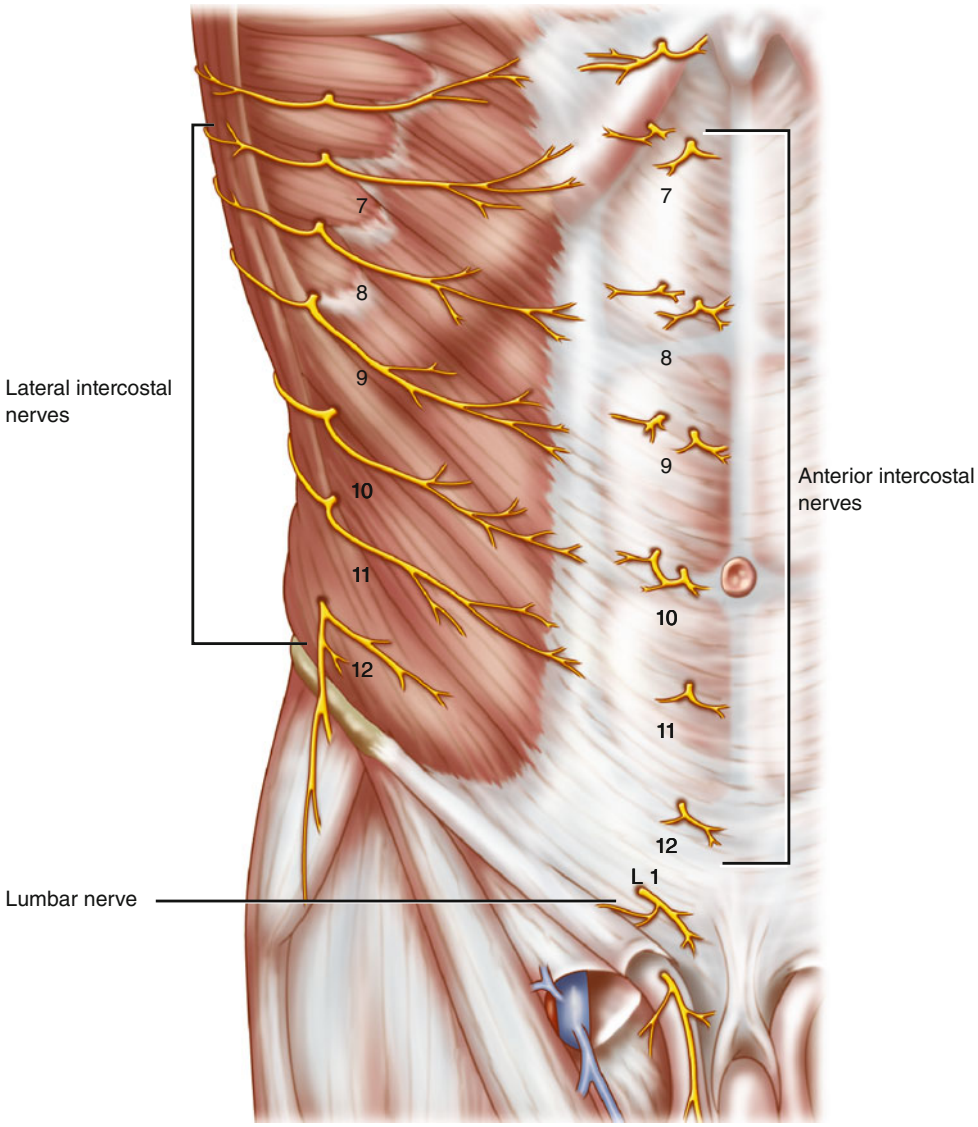


Fig. 7.2 Anatomy of the abdominal wall. The innervations come from the seventh to the twelfth intercostals and the first lumbar nerves

the external and internal oblique muscles. Given the relative avascularity and absence of nerves between the external and internal oblique fasciae from the anterolateral abdominal wall to the lateral border of the rectus sheath, this space is an ideal plane for blunt dissection and subsequent expander placement. It is bordered superiorly by the costovertebral margin, medially by the lateral border of the rectus sheath, laterally by the midaxillary line, and inferiorly by the inguinal ligament [1–4].

Most patients who have previously undergone major abdominal surgery have had an abdominal incision, so their lateral abdominal wall is usually free of scars and defects, thereby providing a well-vascularized soft tissue donor site. The abdominal wall can be anatomically restored with minimal tension and without compromising the integrity of the abdominal muscles, vessels, and nerves. Understanding the pathophysiology and the distorted anatomy of a difficult abdomen is paramount.

Acute Setting

In the acute setting, when patients are undergoing lifesaving procedures, the decision to close or not close the abdomen is not easy.

Leaving the Abdomen Open

The surgeon should recognize the clinical picture of patients who may have abdominal compartment syndrome (ACS) or be at risk for developing it. In that scenario, the surgeon

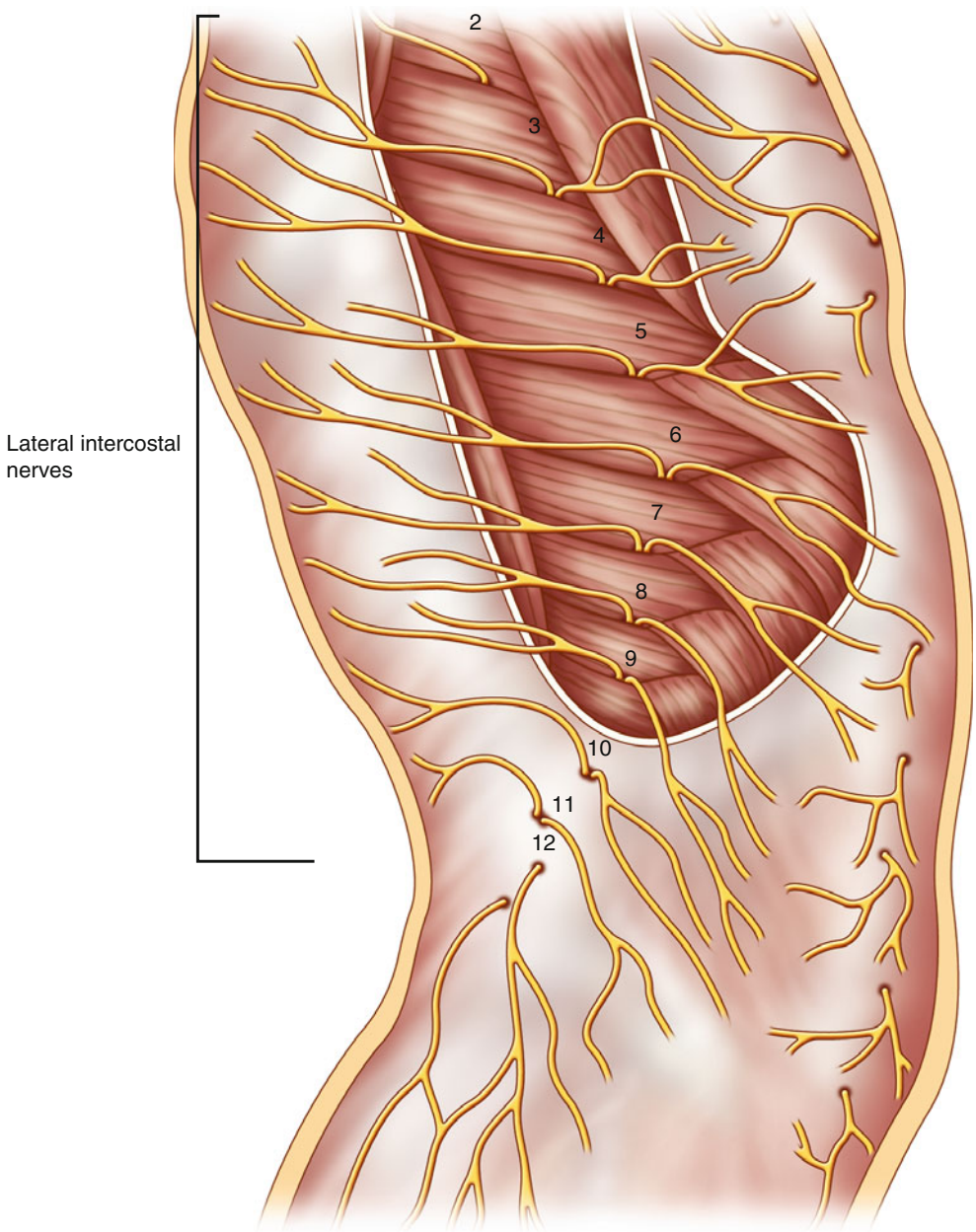


Fig. 7.3 Anatomy of the abdominal wall. The intercostals referenced in Fig. 7.2 and the lumbar vessels and nerves travel from the posterior midline to the anterior midline in an oblique, anterior pathway between the internal oblique and transversalis muscles

should consider leaving the abdomen open temporarily; in patients with more severe cases of ACS or with complicated situations, the abdomen should be left open for an extended time (Figs. 7.4 and 7.5). Leaving the abdomen open is not without major complications, however, including fistulas, hernias, and loss of abdominal wall domain. Leaving the abdomen open commits patients to additional major surgery (Fig. 7.6). Clearly, whatever procedure is selected in the acute setting will affect future decisions and outcomes [5].

Damage control in patients whose abdomen cannot or should not be closed is lifesaving; the most effective procedure was popularized by Rotondo et al. in the 1990s [6]. Patients who have sustained a major abdominal injury, with hemorrhagic shock or peritonitis caused by intra-abdominal sepsis, require extensive resuscitation. The resulting edema of the bowel, retroperitoneum, and abdominal wall causes loss of compliance of the abdominal wall. Primary closure under tension leads to ACS, further tissue necrosis, necrotizing fasciitis, and fascial dehiscence. Damage control must be performed early, before patients become coagulopathic and severely acidotic. Damage control is increasingly used in nontrauma patients; it has potential applications in almost every cavity.

"Closing" the Abdomen

Numerous techniques have been described for handling the acute inability to close the abdomen. For detailed descriptions of how to temporarily "close" the abdomen, see extensive descriptions in various chapters throughout this book. However, the techniques discussed next merit special mention [7–9].

Towel Clip Closure

Although we rarely use towel clip closure, it is the simplest and most rapidly performed technique for temporarily closing an abdominal wound in clinically unstable patients.

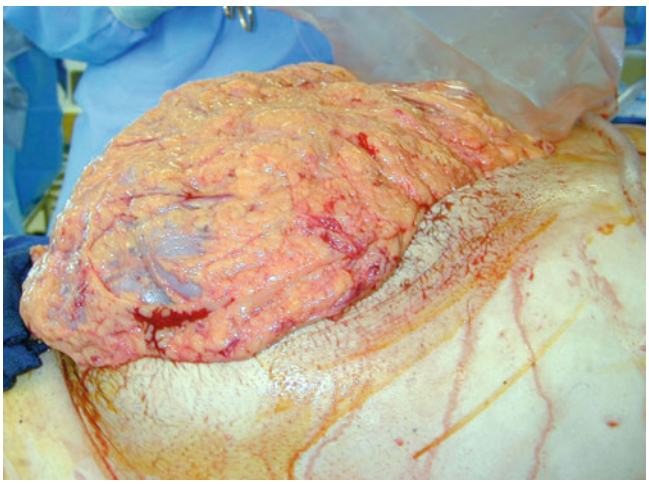


Fig. 7.4 Abdominal compartment syndrome (ACS) developed intraoperatively in a blunt retroperitoneal and extremity injury, requiring immediate decompressive laparatomy

Depending on the length of the incision, up to 25–30 standard towel clips might be necessary to complete closure of the wound during a 2-min period (Fig. 7.7).

Suture Closure

All attempts should be made to preserve the ability to close the skin over the fascia or over the viscera. If possible, skin should be closed in patients who will eventually need a second-look operation, such as those with an ischemic bowel, catastrophic trauma, or other intra-abdominal disasters. The suture closure technique can be used with or without intra-abdominal packing. This technique has serious limitations and might not be applicable in patients with extensive edema of the retroperitoneum or of the viscera itself [7–9].

Retention Sutures

Retention sutures incorporating large portions of tissue tied under tension can forcibly contain the abdominal contents. Unfortunately, retention sutures exacerbate ACS and have been implicated in the development of enterocutaneous fistulas (ECFs), even when the sutures are placed extraperitoneally, so this technique should not be used for temporary closure. Instead, simple closure of the skin, if possible, should be performed. Best, however, is not to use any sutures, but rather to employ other techniques, such as vacuum-assisted closure (VAC), that protect the skin from injuries induced by large sutures.

Temporary Silos

With extensive edema and distention of intra-abdominal organs, an abdominal silo can be inserted to cover the

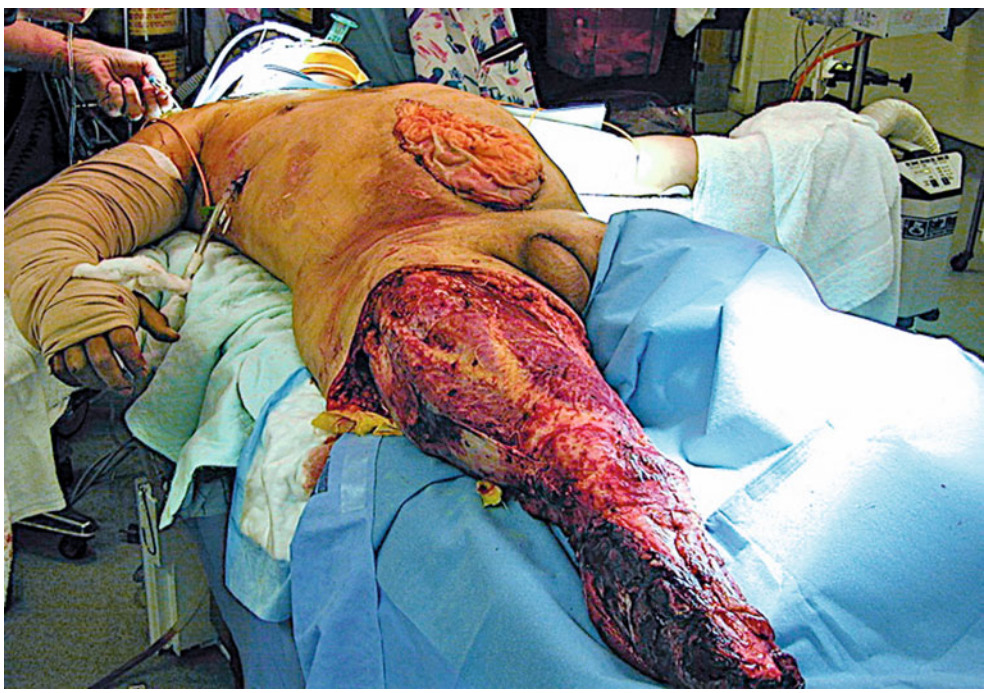


Fig. 7.5 Abdominal compartment syndrome (ACS) resolved in 36 h. Patient was taken back for colostomy (to prevent contamination of perineum) and abdominal wall closure

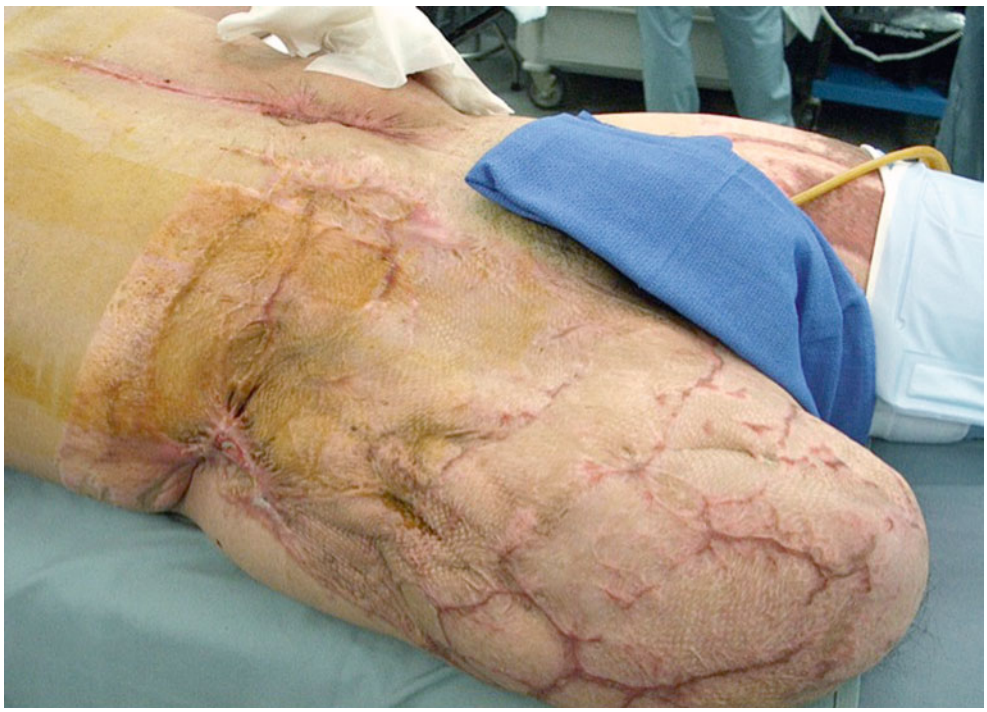


Fig. 7.6 Final look at patient in Fig. 7.5 after multiple operations

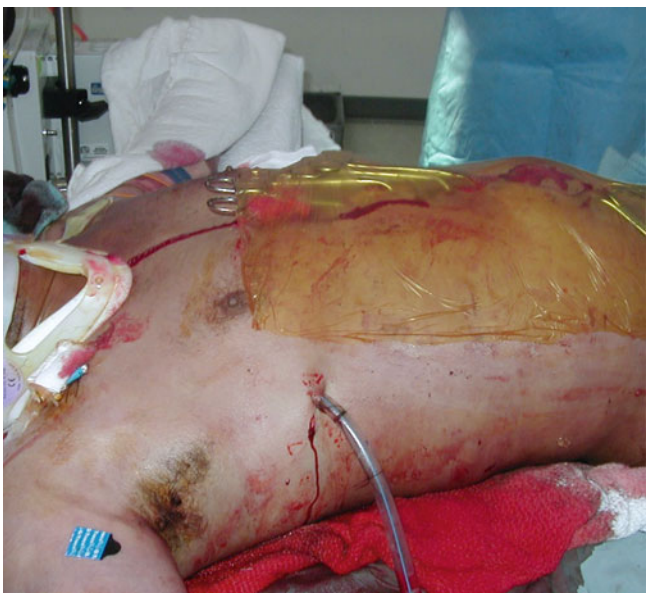


Fig. 7.7 Temporary closure of the abdomen with towel clips. We do not rely on this technique any longer

exposed viscera. Some authors use plastic bags or silos sutured to the skin to allow the viscera to extrude from the peritoneal cavity. We do not prefer this technique because it involves suturing into the skin or fascia; doing so may cause recurrence of ACS. Instead, we cover the intestines with an "intestinal bag" and dressing. The surgeon must be aggressive about returning patients with temporary silos to the operating room as soon as possible, either to close the abdomen permanently or at least to cover the intestines with skin and subcutaneous tissue.

Combination Closure

In patients with significant liver injuries requiring perihepatic packs, a combination of closures might be appropriate. In such patients, tight closure of the upper abdomen is sometimes desirable to maintain tamponade of the injured liver. Partial fascial closure (limited to the upper abdomen) or partial towel clip closure (also limited to the upper abdomen) might be used in conjunction with a silo placed over the lower abdomen.

Vacuum-Assisted Wound Closure

The fundamental reasons for applying suction (via VAC) to an open wound over the midgut are to allow for the rapid removal of peritoneal fluid and to collapse spaces between the viscera. Both steps will make the contents of the abdominal cavity smaller, resulting in a greater chance of subsequently performing a formal aponeurotic closure of the midline incision [10].

Open Packing

Frustrations with the previous closure techniques led to the development of the abdominal wall pack or open-packing technique. It maintains the viscera within the peritoneal cavity, allows for egress of fluid, and can be rapidly performed. When their intestinal edema resolves, patients can be returned to the operating room for removal of the packing and for gradual tightening of the retention sutures until the linea alba can be closed.

Skin Graft

Occasionally with loss of abdominal wall, wound closure cannot be attempted for several weeks. In such patients, the wound is covered with absorbable Vicryl® (Ethicon, Somerville, NJ) mesh (Fig. 7.8), which eventually is allowed to granulate (Fig. 7.9), and a split-thickness skin graft technique is applied (Fig. 7.10). Then, at a later date, the abdominal incisional hernia is addressed [11, 12].

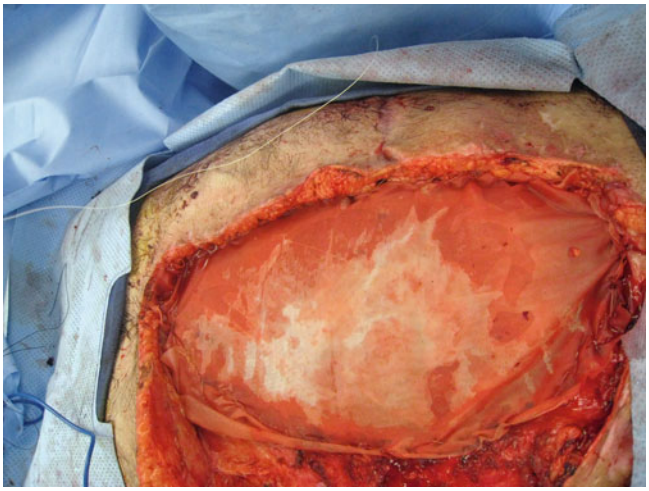


Fig. 7.8 Temporary closure with of the abdomen with Vicryl. This is a useful technique when return to the operating room is expected in 24–36 h and when the abdomen is left to granulate

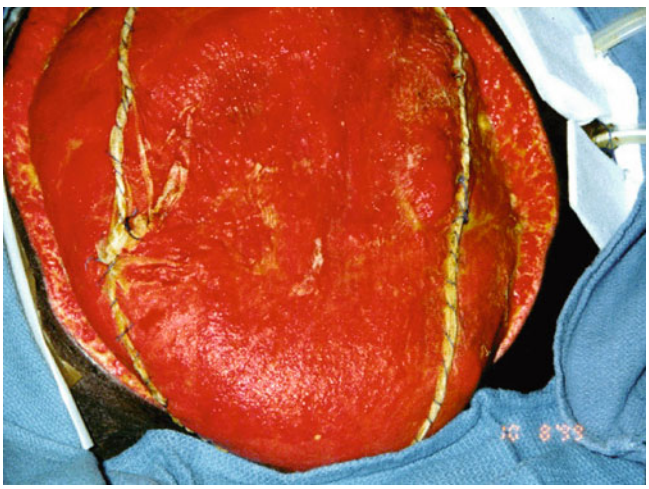


Fig. 7.9 Granulation of the abdomen wall managed with open abdomen and Vicryl "closure"

Chronic Conditions

Indications for Surgical Repair

Once patients have survived the acute stage—which may last for weeks or, worse, for months—deciding whether to reconstruct the abdominal wall defect is necessary. The main indication for reconstruction is a large hernia or the development of multiple fistulas with or without a stoma, ECF, or enteroatmospheric fistulas (EAFs) (Fig. 7.11). Reconstruction may also be mandated after failed attempts to close a celiotomy wound or when components of the abdominal wall, for whatever reason, are either injured or absent.

Specific criteria have been suggested to identify patients who may require special closure techniques, including one or more of the following: large defect size (>40 cm^2); absence of stable skin coverage; hernia recurrence after prior closure attempts; infected or exposed mesh; systemic compromise (concurrent malignancy); local abdominal tissue compromise (irradiation, corticosteroid dependence); and concomitant ECFs [13–15]. Other indications for reconstruction are lack of quality of life, inability to work or to exercise, pain, and recurrent obstructions requiring hospitalizations and frequent surgeries.

Identifying a bona fide indication for reconstruction might seem simple, but it is not an easy task in patients with massive hernias or complex abdominal wall defects. Many surgeons do not consider the mere presence of a hernia to be a sufficient indication for major surgery. But, we believe that large defects should be repaired unless a serious contraindication exists or unless surgery would put the patient at major risk. So, the decision will be between the patient and the surgeon on how they will proceed.

Comorbidities

Analysis of patients with a complex abdominal wall hernia must include an assessment of its components and location. Adequate tissue for direct closure is generally not available. When skin coverage is stable, intraperitoneal mesh placement is recommended. But, when skin coverage is absent or compromised, abdominal wall reconstruction generally requires use of some sort of flap. Regional and distant flaps suitable for reconstruction have been identified; criteria include a reliable vascular pedicle and a safe arc of rotation to the specific zone on the abdominal wall [16–18].

The repair of complex incisional hernias is a common surgical procedure. Every year, in the United States alone, an estimated 250,000 ventral hernia repairs are performed [19, 20]. Despite significant advances in hernia repair techniques and technologies, recurrence rates after a standard ventral herniorrhaphy remain unacceptably high. Wound dehiscence, infections, pain, and suture sinus formation can contribute to postoperative complications. Luijendijk et al. found that nearly a quarter of ventral hernias repaired with synthetic mesh recur within 3 years; the recurrence rate approaches 50 % after primary repair alone and exceeds 60 % by 10 years postoperatively [21].

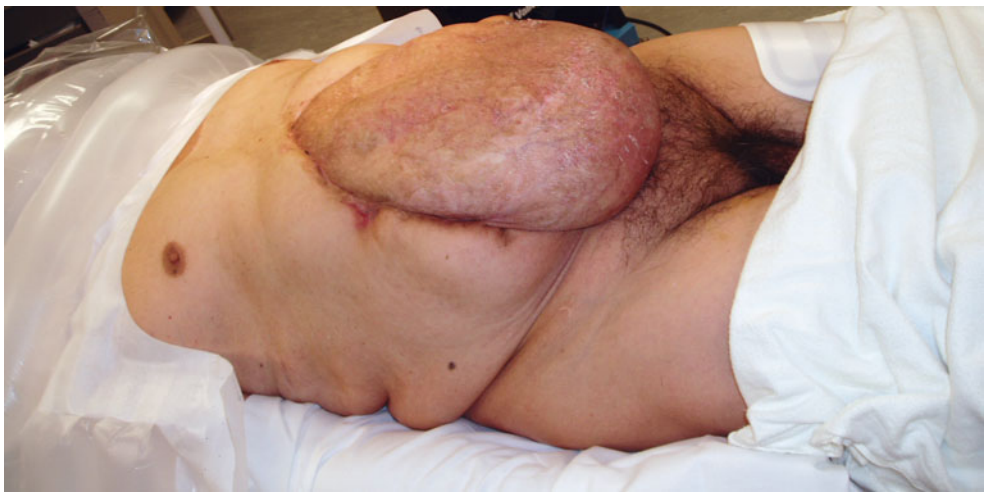


Fig. 7.10 Patient with skin graft matured. At one time, a wait of at least 9–12 months was necessary before embarking on definitive closure and abdominal wall reconstruction in such cases

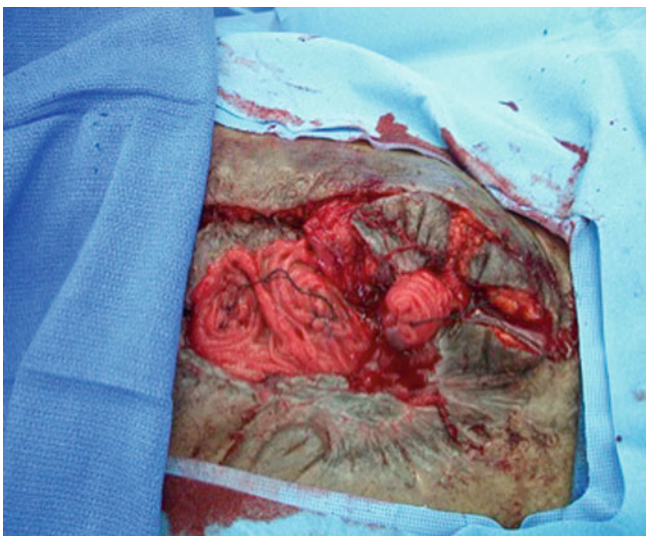


Fig. 7.11 "Fistula city"

After ventral hernia repair, surgical site infections (SSIs) and recurrence are the main issues. Common SSIs include seromas, wound dehiscence, and ECFs. Each of these complications is associated with morbidity and the risk of additional sequelae. Wound dehiscence, for example, may lead to exposure of the repair material; if the material is a permanent synthetic mesh, then it will likely require removal because of the continued risk of infections. The infection rates after ventral hernia repair range from 4 to 16 %, compared with only 2 % after other clean surgical procedures. Luijendijk et al. observed a recurrence rate of 80 % in patients with postoperative infections, compared with 34 % in those without infections [21]. Awad et al. estimated that more than 75 % of all recurrences are caused by infections and inadequate repair material fixation or overlap [22]. Comorbidities and the infection risk are best analyzed using the National Surgical Quality Improvement Program (NSQIP) database. According to its data, significant independent predictors of wound infections include corticosteroid use, smoking, coronary artery disease, chronic obstructive pulmonary disease, low preoperative serum albumin levels, prolonged operative time, and the use of absorbable synthetic mesh [23, 24]. Individual comorbidities may increase the risk of postoperative infections as much as fourfold.

Materials

With any incisional ventral hernia repair, the overriding recommendation is to reinforce the primary fascial closure with a prosthetic repair material. But, deciding on what kind of materials to use in hernia repair is difficult. The surgeon has to consider the individual patient's biology, physiology, infection status, and religion, as well as the cost.

In the late 1990s, biologic materials were introduced as a possible ventral hernia solution [25, 26]. Currently, along with synthetic materials, multiple biologic products are available for use. Still, no consensus exists regarding which patient populations are best served by which materials, how products should be implanted, and what their overall risks of complication and recurrence are.

Synthetic Mesh

Synthetic mesh is currently the most common material used for reinforcement of ventral hernias. It is associated with lower recurrence rates, ease of use, and low cost. Its disadvantages include the risks of visceral adhesions, of erosion into bowel leading to formation of ECFs or bowel obstructions, of extrusion of the repair material, and of infections. Permanent synthetic mesh often requires later surgical removal, necessitating a reoperation. After mesh removal for an infection, the surgeon is left with a contaminated field and a hernia defect larger than the original that still requires a

repair material, leading to a high reinfection rate. Patients may have acute postoperative mesh infections or wound dehiscence that may expose the mesh. Reoperations through synthetic mesh may also lead to infections. A seroma may become infected, leading to subsequent contamination and necessitating mesh removal [27–31].

Biologic Mesh

When the wound infection risk is high, the surgeon may consider the use of biologic mesh in place of permanent synthetic mesh. Some biologic repair materials remain intact even in patients with active infections; such materials are more resistant to infections and do not require removal when exposed or infected. Some biologic repair materials have also demonstrated antimicrobial activity, both in vitro and in animal models [32–34]. The ability of certain biologic materials to support revascularization may contribute to clearance of bacteria. We have previously reported good outcomes with AlloDerm® (LifeCell, Branchburg, NJ) and Strattice™ (LifeCell) repair for incisional hernia repair in high-risk patient groups. These patients could be treated nonsurgically, even when their wounds become frankly infected [35–37].

Grading System

For many surgeons, the choice between synthetic and biologic repair mesh is based on several considerations, including the cost, the operative technique (open vs. laparoscopic), technical expertise, the risk of SSIs, and the individual patient's religion. For patients at low risk for SSIs, the choice of reinforcement should be based on the surgeon's preference and patient factors. The Ventral Hernia Working Group (VHWG) created a system that consists of the following four grades:

Grade 1 (low risk) describes hernias in patients who have no comorbidities; typically, they are younger, healthy individuals.

Grade 2 (comorbid) describes hernias in patients who have comorbidities (e.g., smoking, diabetes, or malnutrition) that increase the risk of SSIs but who do not have evidence of wound contamination or active infections. Thresholds at which the infection risk increases include a blood glucose level equal to or greater than 110 mg/dL (hemoglobin $A_{1C} > 7.0$) and patient age equal to or greater than 75 years. Patients in grade 2 have a wound infection rate four-fold greater than that predicted solely by VHWG wound classification score. The increased risk associated with grade 2 hernias suggests a potential advantage for the use of appropriate biologic repair materials to reinforce open repairs.

Grade 3 (potentially contaminated) is considered when there is evidence of wound contamination. Factors that suggest contamination include the presence of a nearby seroma, violation of the gastrointestinal tract, or a history of wound infections. Grade 3 hernias include those in patients with active or suspected wound contamination. Permanent synthetic mesh is not recommended for such patients; instead, biologic repair is a good option because it does not necessitate removal, even in the setting of active infections.

Grade 4 (infected) includes hernias with active infections, especially frankly infected synthetic mesh and septic dehiscence. Replacement of infected synthetic mesh with new permanent synthetic mesh leads to a high reoperation rate and to additional mesh infections and replacement. Before placement of repair material and definitive closure, infected wounds must be thoroughly prepared and the bioburden meticulously reduced. No repair material should be used in patients with gross, uncontrolled contamination; in such patients, the surgeon may consider a delayed repair [38–40].

Each grade relates to the aforementioned risk factors for SSIs but does not consider the defect's size or complexity or the proposed repair approach. A greater number of previous repairs substantially increases the risk of hernia recurrence [38–40].

Principles of Repair

The principles of incisional abdominal wall hernia repair are optimization of the patient's condition, wound preparation, centralization and approximation of the rectus muscles along the midline to the extent possible, and use of the appropriate prosthetic repair material to reinforce the closure. Optimization of the patient's condition includes encouraging smoking cessation (>4 weeks preoperatively), maintaining acceptable blood glucose levels (<110 mg/dL), improving oxygenation in patients with chronic hypoxia (e.g., by using bronchodilators, inhaled corticosteroids, or prostaglandin inhibitors), and setting realistic expectations. Wound preparation consists of two stages. The first occurs before surgery and may include percutaneous drainage of any abscesses and management of any skin irritation from an ECF. The second stage occurs in the operating room: Sharp debridement of all devitalized or infected tissue to reduce the bioburden of the wound is critical; contaminated wounds should be cleaned by pulse lavage. Approximation of the rectus muscles must be attempted to restore normal physiologic tension. Too little tension in a hernia repair results in wound edge separation and poor collagen organization in the incision; too much tension leads to ischemia and wound dehiscence. Physiologic tension attempts to

achieve a balance between those opposing outcomes. Techniques for repair of ventral hernias include retrorectus and component separation. Retrorectus allows for placement of repair material behind the defect without contacting the viscera. For larger defects, formal component separation, as first described by Ramirez et al. and modified by numerous authors, is the preferred approach for approximating the midline with minimal or no tension. Component separation creates a dynamic repair by using incisions that create fascial release to bring the rectus muscles together at the midline, thereby re-creating an innervated, functional abdominal wall. Open component separation has utility in patients with challenging defects and can reduce the recurrence rate; however, patients will still benefit from use of the appropriate prosthetic repair material, particularly if they have complex defects (e.g., degraded fascia, tight closure, multiple comorbidities, and wound contamination) [1, 41–47].

Mesh Placement

Resistance to infections for some biologic repair materials might be related to the ingrowth of cells and vasculature. The neovascularization demonstrated in several studies of biologic repair materials may allow such materials to better resist infections when placed in a potentially contaminated field. To date, however, no comparative trials have evaluated different biologic repair materials in patients with incisional hernias; currently, the differentiation between products is based on early findings with a limited number of available materials.

Studies of biologic repair materials have documented high rates of seromas, diastasis, bulging, and hernia recurrence. In one study, the hernia recurrence rate was reduced when component separation was combined with use of a biologic mesh material; conversely, in another study, bridging with a biologic repair material, without reducing the size of the defect, was associated with a hernia recurrence rate of 80 % [48–50].

In open incisional hernia repair, prosthetic materials may be placed to reinforce a primary repair or to bridge a remaining defect if approximation of the midline is not possible using one of the three techniques described elsewhere in this book. The surgeon's preference and experience as well as patient factors should also be considered [51, 52].

Other Surgical Approaches

Restoring the integrity of the abdominal wall after any abdominal catastrophe, such as necrotizing pancreatitis requiring multiple surgical interventions, represents a significant surgical challenge (Fig. 7.12). Most of the current literature supports staged closure of the acute abdominal wall defect and delayed abdominal wall reconstruction [53]. Abdominal wall reconstruction of massive ventral hernias (resulting from ACS and other serious clinical conditions) makes primary closure arduous. Massive abdominal hernias (i.e., those greater than 11 cm in the largest dimension) are complicated by the extent of the abdominal wall loss and subsequent tissue contraction.

Many surgical techniques used today to close these large defects are described throughout this book. Each technique has its limitations and challenges. Abdominal wall fascial release procedures without previous tissue expansion are limited in their ability to advance the fascia; such procedures frequently interrupt the innervations and blood supply to the abdominal wall [54].

Autogenous Reconstruction

A definitive abdominal wall reconstruction technique has not been fully defined currently. Yet, at present, autogenous reconstruction with myofascial flaps is the gold standard, especially in patients with contamination from chronic wounds and ECFs or ostomies [53, 54]. Patients with massive fascial defects (greater than 20 cm wide) or loss of tissue (from previous tumor extirpation or necrotizing infections) represent a profound challenge in terms of abdominal wall reconstruction. The reconstructive surgeon might be unable to close the abdominal fascia because of the limitations of flap advancement, the size of the defect, or an acute increase in abdominal pressure affecting pulmonary, cardiac, and mesenteric function. In such patients, fascial replacement is indicated. It can include permanent or absorbable mesh, autogenous fascial grafts, or allogeneic material (acellular dermal matrix). Reliable coverage of the reconstructed abdominal wall is essential and may warrant preoperative tissue expansion if skin quality or availability is questionable. Multiple techniques have been described in the literature. The component separation technique, in combination with epifascial mesh reinforcement (as appropriate), is the procedure of choice for most complicated abdominal wall hernias [54].

Tissue Expanders

Inserting tissue expanders between the external and internal oblique fasciae creates minimal disruption of the nerves and vessels of the lateral abdominal wall and avoids some of the disadvantages of other surgical techniques. During the initial stage of this two-stage procedure, tissue expanders are placed under the skin and subcutaneous tissue lateral to the defects. After adequate interval expansion, the second stage is performed: The expanders are removed, the visceral contents are reduced (easily), and the fascia is reapproximated with polypropylene mesh. The expanded skin is then closed (again, easily) over the fascial repair [55].

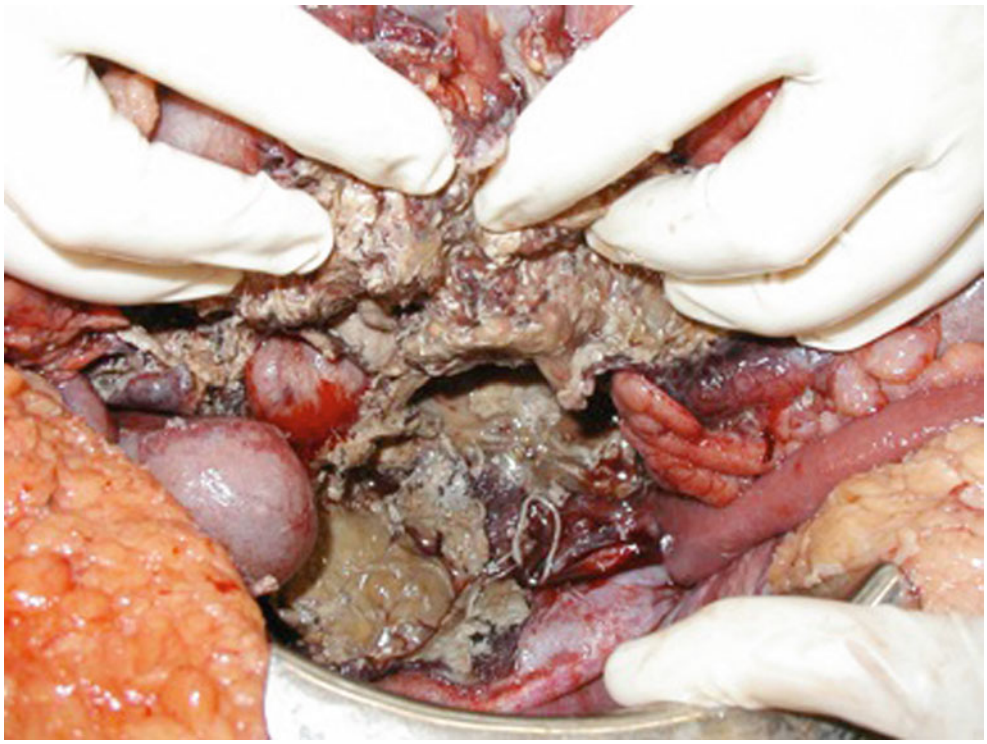


Fig. 7.12 Hemorrhagic necrotizing pancreatitis requiring multiple operations and "washouts"

Hobar et al. described placing tissue expanders through the rectus sheath in the plane between the internal oblique and transversalis fascial layers [56]. Carlson et al. described placing tissue expanders into the subcutaneous pocket above the abdominal wall musculature and fasciae [55].

Laparoscopy

The documented advantages of the laparoscopic approach include smaller incisions, a lower risk of complications, a shorter hospital stay, and patient preference. According to a recent meta-analysis of randomized controlled trials comparing open and laparoscopic incisional hernia repairs, open repair is associated with a significantly higher rate of complications. Reported complications included seromas, abscesses, hematomas, cellulitis, wound infections, bowel obstructions, and ileus. A single-institution cohort study comparing open and laparoscopic ventral hernia repair reported major morbidities in 15 % of the open group and in 7 % of the laparoscopic group; the mean follow-up period was 30–36 months. In addition to a higher rate of seroma formation, the limitations of laparoscopic repair include the inability to restore the functional abdominal wall anatomy as well as the inability to manage skin redundancy and the hernia sac. Current laparoscopic approaches do not routinely employ extensive tissue mobilization, meaning that the repair material is almost always bridging some aspect of the defect [57].

Minimally Invasive Techniques

Several investigators have described minimally invasive techniques of component separation. Experience with these techniques has been reported in cadavers, in a porcine model, in select human patients with infected repair materials, and in small comparative groups of human patients [45, 46, 57].

Summary

Abdominal wall reconstruction, both in the acute setting and as an elective or semielective procedure, presents a surgical challenge. Ventral hernia repair often involves significant loss of abdominal wall domain and inadequate soft tissue coverage. Incisional hernias are the most common wound complication after abdominal surgery, with a reported incidence rate of 2–11 % and a recurrence rate of 20–46 %. Careful evaluation of patients with complex abdominal defects should reveal predisposing factors for herniation, including inadequate local fascial and muscular layers caused by prior tissue loss; muscle denervation or vascular insufficiency because of prior irradiation or infections; wound infections; obesity; chronic pulmonary disease; malnutrition; sepsis; anemia; corticosteroid dependency; or concurrent malignant process.

Direct repair should be limited to patients with small defects (<5 cm in diameter) and with few associated risk factors for poor wound healing. With preexisting loss of abdominal wall layers, excessive tension at the closure site results in ischemia and eventual failure of the repair. This problem is avoided with the use of mesh, either alone or combined with a flap. In noninfected wounds with stable overlying skin, mesh is preferred to restore the integrity of

the abdominal wall. When soft tissue coverage is also inadequate, regional or distant flaps are necessary, either alone or combined with mesh. Both nonabsorbable polypropylene mesh (Marlex®, Davol, Warwick, RI; and Prolene®, Ethicon) and polytetrafluoroethylene (Gore-Tex®, W.L. Gore & Associates, Newark, DE) mesh are advocated for abdominal wall reconstruction. Prolene is inert, appears to have adequate strength, and unlike Gore-Tex, allows for tissue incorporation and ingrowth of granulation tissue. If available, the omentum should be placed between the bowel and the mesh.

The first step in treating patients with complex abdominal wall hernias is careful assessment, starting with risk factors and the size of the defect. Smaller defects (<2 cm) might be suitable for primary repair; larger defects, if the fascia does not meet without undue tension, should be reduced as much as possible. Most defects too large for primary repair can be closed with component separation and reinforced with prosthetic repair materials. For the rare patients in whom component separation is not feasible or is insufficient to reduce the defect completely, the surgeon might consider bridging the defect with prosthetic repair materials. Hernias that are grade 4 should be repaired with open procedures. Most grade 1, some grade 2, and a few grade 3 hernias are suitable for repair with permanent synthetic mesh; all patients considered high risk for SSIs should be considered for surgery with appropriate biologic mesh repair.

References

1. Ramirez OM, Ko MJ, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86:519–26.
2. DeFranzo AJ, Kingman GJ, Sterchi JM, et al. Rectus turnover flaps for the reconstruction of large midline abdominal wall defects. Ann Plast Surg. 1996;37:18–23.
3. de Vries Reilingh TS, van Goor H, Charbon JA, Rosman C, Hesselink EJ, van der Wilt GJ, et al. Repair of giant midline abdominal wall hernias: "components separation technique" versus prosthetic repair: interim analysis of a randomized controlled trial. World J Surg. 2007;31:756–63.
4. Vargo D. Component separation in the management of the difficult abdominal wall. Am J Surg. 2004;188:633–7.
5. Fabian TC, Croce MA, Pritchard FE, Minard G, Hickerson WL, Howell RL, et al. Planned ventral hernia. Staged management for acute abdominal wall defects. Ann Surg. 1994;219:643–50.
6. Rotondo MF, Schwab CW, McGonidal MD, et al. Damage control: an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma. 1993;35:375–82.
7. Stone HH, Fabian TC, Turkleson ML, Jurkiewicz MJ. Management of acute full-thickness losses of the abdominal wall. Ann Surg. 1981;193:612–8.
8. Saxe JM, Ledgerwood AM, Lucas CE. Management of the difficult abdominal closure. Surg Clin North Am. 1993;73:243–51.
9. Ledgerwood AM, Lucas CE. Management of massive abdominal wall defects: role of porcine skin grafts. J Trauma. 1976; 16:85–8.
10. Caro A, Olona C, Jimenez A. Treatment of the open abdomen with topical negative pressure therapy: a retrospective study of 46 cases. Int Wound J. 2011;8(3):274–9.
11. Espinosa-de-los-Monteros A, de la Torre JI, Marrero I, Andrades P, Davis MR, Vasconez LO. Utilization of human cadaveric acellular dermis for abdominal hernia reconstruction. Ann Plast Surg. 2007;58:264–7.
12. Kolker AR, Brown DJ, Redstone JS, Scarpinato VM, Wallack MK. Multilayer reconstruction of abdominal wall defects with acellular dermal allograft (AlloDerm) and component separation. Ann Plast Surg. 2005;55:36–41.
13. Dunne JR, Malone DL, Tracy JK, Napolitano LM. Abdominal wall hernias: risk factors for infection and resource utilization. J Surg Res. 2003;111:78–84.
14. Finan KR, Vick CC, Kiefe CI, Neumayer L, Hawn MT. Predictors of wound infection in ventral hernia repair. Am J Surg. 2005; 190:676–81.
15. Pessaux P, Lermite E, Blezel E, et al. Predictive risk score for infection after inguinal hernia repair. Am J Surg. 2006;192:165–71.
16. Guyatt G, Gutterman D, Baumann MH, Addrizzo-Harris D, Hylek EM, Phillips B, et al. Grading strength of recommendations and quality of evidence in clinical guidelines: report from an American College of Chest Physicians task force. Chest. 2006;129:174–81.
17. Shekelle PG, Woolf SH, Eccles M, Grimshaw J. Clinical guidelines: developing guidelines. BMJ. 1999;318:593–6.
18. Flum DR, Horvath K, Koepsell T. Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg. 2003;237:129–35.
19. Houck JP, Rypins EB, Sarfeh IJ, Juler GL, Shimoda KJ. Repair of incisional hernia. Surg Gynecol Obstet. 1989;169:397–9.
20. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg. 2004;240: 578–83.
21. Luijendijk RW, Hop WC, van den Tol MP, de Lange DC, Braaksma MM, IJzermans JN, et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med. 2000;343:392–8.
22. Awad ZT, Puri V, LeBlanc K, et al. Mechanisms of ventral hernia recurrence after mesh repair and a new proposed classification. J Am Coll Surg. 2005;201:132–40.
23. White TJ, Santos MC, Thompson JS. Factors affecting wound complications in repair of ventral hernias. Am Surg. 1998;64:276–80.
24. Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR. Guideline for prevention of surgical site infection, 1999. Hospital Infection Control Practices Advisory Committee. Infect Control Hosp Epidemiol. 1999;20:250–78.
25. Szczerba SR, Dumanian GA. Definitive surgical treatment of infected or exposed ventral hernia mesh. Ann Surg. 2003;237: 437–41.
26. Petersen S, Henke G, Freitag M, Faulhaber A, Ludwig K. Deep prosthesis infection in incisional hernia repair: predictive factors and clinical outcome. Eur J Surg. 2001;167:453–7.
27. Patton Jr JH, Berry S, Kralovich KA. Use of human acellular dermal matrix in complex and contaminated abdominal wall reconstructions. Am J Surg. 2007;193:360–3.
28. Paton BL, Novitsky YW, Zerey M, Sing RF, Kercher KW, Heniford BT. Management of infections of polytetrafluoroethylene-based mesh. Surg Infect (Larchmt). 2007;8:337–41.
29. Voyles CR, Richardson JD, Bland KI, Tobin GR, Flint LM, Polk Jr HC. Emergency abdominal wall reconstruction with polypropylene mesh: short-term benefits versus long-term complications. Ann Surg. 1981;194:219–23.
30. Jones JW, Jurkovich GJ. Polypropylene mesh closure of infected abdominal wounds. Am Surg. 1989;55:73–6.
31. van't Riet M, de Vos van Steenwijk PJ, Bonjer HJ, Steyerberg EW, Jeekel J. Mesh repair for postoperative wound dehiscence in the presence of infection: is absorbable mesh safer than non-absorbable mesh? Hernia. 2007;11:409–13.
32. Maurice SM, Skeete DA. Use of human acellular dermal matrix for abdominal wall reconstructions. Am J Surg. 2009;197:35–42.

33. Sarikaya A, Record R, Wu CC, Tullius B, Badylak S, Ladisch M. Antimicrobial activity associated with extracellular matrices. Tissue Eng. 2002;8:63–71.
34. Badylak SF, Coffey AC, Lantz GC, Tacker WA, Geddes LA. Comparison of the resistance to infection of intestinal submucosa arterial autografts versus polytetrafluoroethylene arterial prostheses in a dog model. J Vasc Surg. 1994;19:465–72.
35. Milburn ML, Holton LH, Chung TL, Li EN, Bochicchio GV, Goldberg NH, et al. Acellular dermal matrix compared with synthetic implant material for repair of ventral hernia in the setting of peri-operative *Staphylococcus aureus* implant contamination: a rabbit model. Surg Infect (Larchmt). 2008;9:433–42.
36. Butler CE, Langstein HN, Kronowitz SJ. Pelvic, abdominal, and chest wall reconstruction with AlloDerm in patients at increased risk for mesh-related complications. Plast Reconstr Surg. 2005;116:1263–75.
37. Nemeth NL, Butler CE. Complex torso reconstruction with human acellular dermal matrix: long-term clinical follow-up. Plast Reconstr Surg. 2009;123:192–6.
38. Kingsnorth A, LeBlanc K. Hernias: inguinal and incisional. Lancet. 2003;362:1561–71.
39. den Hartog D, Dur AH, Tuinebreijer WE, Kreis RW. Open surgical procedures for incisional hernias. Cochrane Database Syst Rev. 2008; Issue 3. Art. No.: CD006438. DOI: 10.1002/14651858.CD006438.pub2.
40. Korenkov M, Paul A, Sauerland S, et al. Classification and surgical treatment of incisional hernia. Results of an experts' meeting. Langenbecks Arch Surg. 2001;386:65–73.
41. Ennis LS, Young JS, Gampper TJ, Drake DB. The "open book" variation of component separation for repair of massive midline abdominal wall hernia. Am Surg. 2003;69:733–42.
42. de Vries Reilingh TS, van Goor H, Rosman C, Bemelmans MH, de Jong D, van Nieuwenhoven EJ, et al. "Components separation technique" for the repair of large abdominal wall hernias. J Am Coll Surg. 2003;196:32–7.
43. Lowe JB, Garza JR, Bowman JL, Rohrich RJ, Strodel WE. Endoscopically assisted "components separation" for closure of abdominal wall defects. Plast Reconstr Surg. 2000;105:720–9.
44. Milburn ML, Shah PK, Friedman EB, et al. Laparoscopically assisted components separation technique for ventral incisional hernia repair. Hernia. 2007;11:157–61.
45. Rosen MJ, Williams C, Jin J, et al. Laparoscopic versus open component separation: a comparative analysis in a porcine model. Am J Surg. 2007;194:385–9.
46. Rosen MJ, Jin J, McGee MF, Williams C, Marks J, Ponsky JL. Laparoscopic component separation in the single-stage treatment of infected abdominal wall prosthetic removal. Hernia. 2007;11:435–40.
47. DiBello Jr JN, Moore Jr JH. Sliding myofascial flap of the rectus abdominis muscles for the closure of recurrent ventral hernias. Plast Reconstr Surg. 1996;98:464–9.
48. Leber GE, Garb JL, Alexander AI, Reed WP. Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg. 1998;133:378–82.
49. Karakousis CP, Volpe C, Tanski J, Colby ED, Winston J, Driscoll DL. Use of a mesh for musculoaponeurotic defects of the abdominal wall in cancer surgery and the risk of bowel fistulas. J Am Coll Surg. 1995;181:11–6.
50. Iqbal CW, Pham TH, Joseph A, Mai J, Thompson GB, Sarr MG. Long-term outcome of 254 complex incisional hernia repairs using the modified Rives-Stoppa technique. World J Surg. 2007;31:2398–404.
51. Bauer JJ, Harris MT, Kreel I, Gelernt IM. Twelve-year experience with expanded polytetrafluoroethylene in the repair of abdominal wall defects. Mt Sinai J Med. 1999;66:20–5.
52. Martin-Duce A, Noguerales F, Villeta R, et al. Modifications to Rives technique for midline incisional hernia repair. Hernia. 2001;5:70–2.
53. Hultman CS, Pratt B, Cairns BA. Multidisciplinary approach to abdominal wall reconstruction after decompressive laparotomy for abdominal compartment syndrome. Ann Plast Surg. 2005;54(3):269–75.
54. Admire AA, Dolich MO, Sisley AC. Massive ventral hernias: role of tissue expansion in abdominal compartment syndrome. Am Surg. 2002;68:491–6.
55. Carlson GW, Elwood E, Losken A. The role of tissue expansion in abdominal wall reconstruction. Ann Plast Surg. 2000;44:147–53.
56. Hobar PC, Rohrich RJ. Abdominal wall reconstruction with expanded musculofascial tissue in a post-traumatic defect. Plast Reconstr Surg. 1994;94:379–83.
57. Turner PL, Park AE. Laparoscopic repair of ventral incisional hernias: pros and cons. Surg Clin North Am. 2008;88:85–100.

Surgical Strategies in the Management of Open Abdomen

8

James F. Whelan, Rahul J. Anand, and Rao R. Ivatury

Introduction

An increasingly common technique for the management of abdominal emergencies in both trauma and general surgery is the employment of a damage control strategy. The use of an abbreviated laparotomy has been shown to reduce mortality; however, the resulting open abdomen is a complex clinical issue for the intensive care unit (ICU) and surgical teams. Modern techniques and technologies are now available that allow for improved management of the open abdomen and the progressive reduction of the fascial defect. These techniques and technologies include the appropriate use of negative-pressure therapy and synthetic or biologic repair materials. It is essential that general and trauma surgeons understand the core principles underlying the need for and management of the open abdomen.

The most common use of the open abdomen technique occurs in the trauma population. At some busy academic level 1 trauma centers, the current rate of damage control surgery (DCS) among those undergoing emergent laparotomy can be as high as 30% [1]. Another important use of the open abdomen is in the treatment of abdominal compartment syndrome (ACS). ACS is defined as intra-abdominal hypertension (IAH) causing organ system dysfunction. Patients requiring postinjury DCS are at highest risk for developing ACS because they are given massive crystalloid and blood product resuscitation and commonly require intra-abdominal packing, which is an independent risk factor for ACS [2].

IAH and ACS are commonly encountered in nontrauma surgical patients as well. Depending on the etiology of the patient's surgical illness (ruptured abdominal aortic aneurysm, acute pancreatitis, burns, etc.), both the morbidity and mortality of IAH/ACS might be quite high. Recent advances in both the diagnosis and the resuscitation of these surgical patients have resulted in significantly improved survival over that seen in years past. Intra-abdominal pressure measurements should be performed in any surgical patient who demonstrates risk factors for IAH/ACS. When IAH or ACS is encountered, decompressive laparotomy is the mainstay of treatment. Despite the undisputed benefit of postinjury DCS as well as fascial release in ACS, these techniques not surprisingly are associated with the potential for major complications. Clinical observation shows that leaving an abdomen open for a prolonged period of time leads to retraction of the fascia and "shrinking" of the abdominal wall relative to the viscera. This retraction, if left unchecked, results in loss of domain.

Planning the closure of an open abdomen is a process that starts on the first day that the abdomen is opened. Multiple factors need to be addressed, optimized, and controlled to achieve the best outcome. Early fascial closure is an independent predictor of reduced complications in patients with open abdomen [3]. Therefore, approximation of the fascial edges should be performed at the earliest possible time. When early closure of this fascial plane is not possible because of ongoing resuscitation or contamination, temporizing measures should be employed. The goal of any temporary abdominal closure technique is no longer just abdominal visceral coverage because fluid control and facilitation of early fascial closure have now become important aspects. Recent evidence indicates that a large proportion of patients

J.F. Whelan, MD, FACS
Division of Trauma, Critical Care, and Emergency General Surgery, Department of Surgery, Virginia Commonwealth University Medical Center, Richmond, VA, USA
e-mail: jwhelan@vcu.edu

R.J. Anand, MD, FACS
Division of Trauma, Critical Care, and Emergency Surgery, Department of Surgery, Virginia Commonwealth University Medical Center, Richmond, VA, USA
e-mail: ranand@mcvh-vcu.edu

R.R. Ivatury, MD, FACS (✉)
Division of Trauma, Critical Care, and Emergency General Surgery, Virginia Commonwealth University, 1200 East Broad Street, W15E, Richmond, VA 23298, USA
e-mail: rivatury@hsc.vcu.edu

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_8,

treated with open abdomen can now have the abdomen closed during the initial hospitalization.

Multiple techniques have been introduced to obtain fascial closure for the open abdomen and to minimize morbidity and cost of care [4]. Although there are currently no standard approaches, some principles are universally accepted: control of effluent, protection of underlying viscera, preservation of intact fascia, prevention of fascial retraction, and minimization of future hernia requiring subsequent laparotomy [5]. The most widely used techniques include implantation of mesh, a vacuum-assisted closure (VAC) system, the "Bogota bag," or a synthetic patch sutured to the fascial edges. Each of these techniques is associated with its own inherent shortcomings.

Considerations Before Closure

In the patient with an open abdomen, the operating surgeon must "command the ship" and decide when the patient is ready to return to the operating room to attempt closure. At our center, we generally return a patient to the operating room 24–48 h after the initial operation. This usually allows adequate resuscitation and correction of metabolic abnormalities to take place. The adequately resuscitated patient who is ready for return to the operating room will generally no longer suffer from the "terrible triad" of hypothermia, acidosis, and coagulopathy. Return to the operating room at 36 h is by no means a "hard-and-fast rule," and if adequately resuscitated, patients can certainly have the abdomen closed earlier. Indeed, some authors have advocated leaving the patient in the operating room, resuscitating and warming there, with closure a few hours later as part of the same extended procedure.

Confirmation of resuscitation can be assessed by numerous factors. Absence of vasopressor requirement and evidence of end-organ perfusion such as adequate urinary output can be assessed. In our ICU, we trend lactate levels as a marker of end-organ perfusion, and normalization of lactate will suggest adequate resuscitation.

Conduct of the "Take-Back" Operation

At the reexploration, the operative surgeon must make a determination that bleeding and contamination have been successfully controlled. If they were left in, packs should be removed at this time, and raw surfaces should be inspected for control of "oozing." If the first operation was for "intra-abdominal catastrophe" such as a leaking feeding tube, the abdomen must be thoroughly inspected for undrained fluid collections. With regard to tube feeds or succus that might be contaminating various spaces in the abdomen, such as the paracolic gutters, the deep pelvis, and the lesser sac, these areas should be explored and irrigated with multiple liters of saline.

If the determination is made that there is still too much gross contamination, the operative surgeon may decide to "wash" out the abdomen again, with the rationale that simply closing at this time would place the patient at a prohibitive risk for formation of intra-abdominal abscess, with all of its attendant risks. A festering intra-abdominal process, for example, can place the patient at increased risk for fascial dehiscence, ongoing sepsis, and chronic wound problems.

Temporary Abdominal Closure Techniques

ABThera™

The ABThera™ (KCI, San Antonio, TX) device is a temporary abdominal closure device built on the principle of the VAC (Fig. 8.6a, b). The innermost layer of the ABThera is a plastic-coated visceral protective layer that contains perforated foam. A second foam layer is placed over this protective layer in a subfascial location, followed by a third foam layer applied "suprafascially" at the level of the skin. Negative-pressure therapy is applied to this system from a negative-pressure therapy unit. The innermost layer is designed to drain fluid all the way down from the paracolic gutters, and this allows for active fluid removal from the open abdomen, reducing edema. This system allows for provision of mechanical tension on the fascia, minimizing retraction, and making subsequent closure less technically challenging. The plastic-coated protective layer ensures the bowel is protected from both the abdominal wall and the atmosphere, minimizing chance of fistula formation. Importantly, the device does not require sutures for placement, and it is easily removable. It must be remembered that even with an ABThera dressing on, the patient can still develop ACS, and measurement of intra-abdominal pressure via bladder pressure in the ICU is of paramount importance.

Vacuum-Assisted Closure

Vacuum-assisted closure is a predecessor to the ABThera. Like the ABThera, VAC is commercially available. Application consists of an inner protective plastic coat, followed by a single foam sponge at the suprafascial skin level. The VAC allows for temporary abdominal closure, measurement of effluent, and application of a constant negative pressure.

Poor-Man's VAC

A "poor-man's VAC" (PMV) (Figs. 8.1 and 8.2) is a device that can be constructed in the operating room to simulate, although not perfectly, the effects of a VAC. Components of a PMV include an inner plastic drape, which can be fashioned from a "1010 drape" (3M™ Steri-Drape large towel

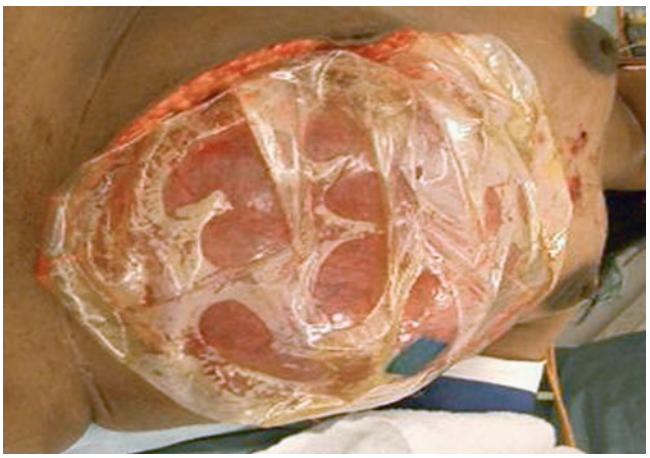


Fig. 8.1 "Poor-man's VAC." Separation of intraperitoneal contents from the fascia by placing a large plastic layer

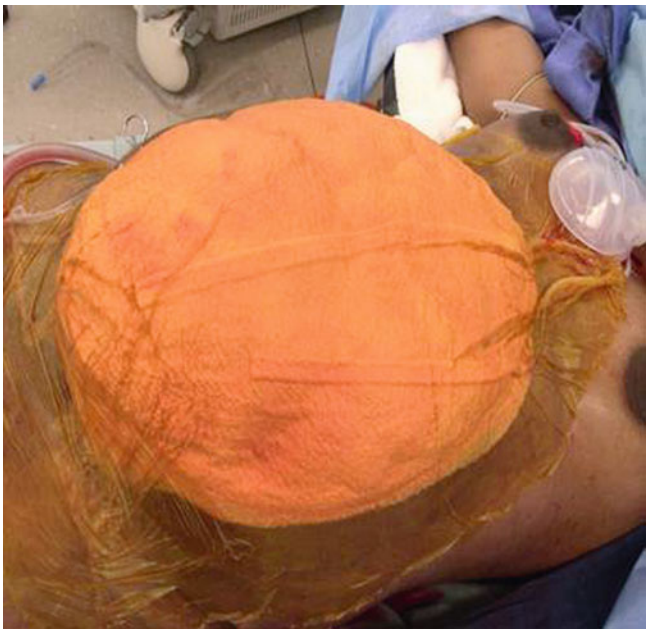


Fig. 8.2 "Poor-man's VAC." Application of Kerlix™ (Covidien) rolls and Jackson-Pratt drains over the Kerlix and creating a watertight dressing that can be connected to a vacuum seal

drape 1010; catalog no. 1010, 3M, St. Paul, MN) or "fluid warmer drape" in the operating room. The surgeon uses scissors to fenestrate this drape and then places it over the bowels. A layer of gauze roll is placed over the wound, covering the drape, followed by a Jackson-Pratt® (JP; Cardinal Health, Dublin, OH) drain. This is covered with another gauze roll, and finally the whole apparatus is covered with an adherent plastic drape (Fig. 8.2). Application of the JP drain to suction will allow for fluid drainage from the wound. There are numerous drawbacks to the PMV compared to a commercially available device. The PMV must be attached to "wall suction" as compared to a negative-pressure unit. This makes it difficult to regulate the pressure applied to the system. If the operator does not cut enough holes in the plastic drape, fluid removal might be inadequate, or the drape may become blocked with "clot." If too many holes are cut in the drape, the gauze roll can be exposed to the viscera, making formation of enteroatmospheric fistula (EAF) a concern.

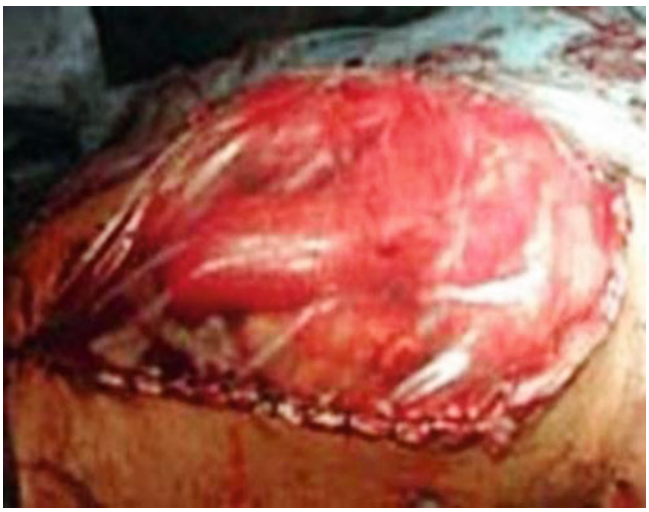


Fig. 8.3 "Bogota bag." Suturing a plastic layer to the skin edges

Bogota Bag

The Bogota bag (Fig. 8.3) is a temporary abdominal closure technique that makes use of a 3-L genitourinary irrigation bag that is sewn directly to the fascia or the skin. Unlike negative-pressure techniques, the Bogota bag does not allow for fluid removal. It does, however, allow for visual inspection of abdominal contents through the plastic.

Wittman Patch

The Wittman Patch® (Star Surgical, Burlington, WI) uses Velcro to permit progressive abdominal closure without necessitating serial operations. A "loop sheet" is sewn to the right-side abdominal fascia and a "hook sheet" to the left-side fascia. These sheets, when closely approximated, resist tangential forces. At subsequent operations, the Velcro sheets are trimmed, and the abdomen is tightened to the point at which fascial closure is possible. A potential drawback of this technique is that the device is sewn to fascia that might be compromised for future closure.

Surgical Zipper

In the spirit of the Wittman Patch and Bogota bag, "surgical zipper" devices are also available that allow for easy reentry to the previously opened abdomen. As with the Wittman

Patch, the device is sewn to fascia that might be compromised for future closure.

Skin-Only Closure

A surgeon may choose to close skin only as a temporary abdominal closure technique. This does not allow for either removal of fluid or inspection of the visceral content. Typically, a heavy nylon or Prolene™ (Ethicon, Somerville, NJ) stitch can be used for this purpose. This technique is quick and easy but may render the skin susceptible to pressure necrosis.

Considerations in the Patient with a Temporary Abdominal Closure

If the abdomen is left open for an extended period of time, efforts to feed the patient and maintain nutrition should be undertaken. However, with an open abdomen, surgical feeding tubes and definitive ostomies should be avoided. Patients may be fed using nasoenteric feeding tubes without the risk of surgical feeding tubes.

Definitive Closure Techniques

If, at the take-back operation, the fascia approximates in a tension-free manner, definitive fascial closure can be undertaken.

We prefer to use PDS™ (Ethicon) suture in a running fashion, but if there is any doubt regarding the integrity of the fascia, an interrupted closure can be used. There is, however, no definitive evidence that favors any type of closure. When closing fascia, the operating surgeon must take adequate purchase of the fascia, meaning at least 1 cm of fascia on either side of the incision, with no more than 0.5 cm between bites. Because of the risk for wound infection, contaminated wounds should be left open or stapled loosely enough that any type of wound infection would be obvious.

If definitive fascial closure is not achievable, skin-only closure is an option. For this technique to be a viable option, the skin must come tension free to the midline. The operating surgeon is, in effect, "accepting" a hernia that for possible repair at a later date. In the critically ill patient or in those patients with extremely limited mobility, the surgeon may decide "never" to fix the hernia. Skin-only closures are performed with running nylon or Prolene stitch on a large needle. Because of the nature of the closure, the wound may weep fluid. While in the hospital, patients should wear a binder, and all staff should be notified of the nature of the skin-only closure to prevent inadvertent disastrous suture removal.

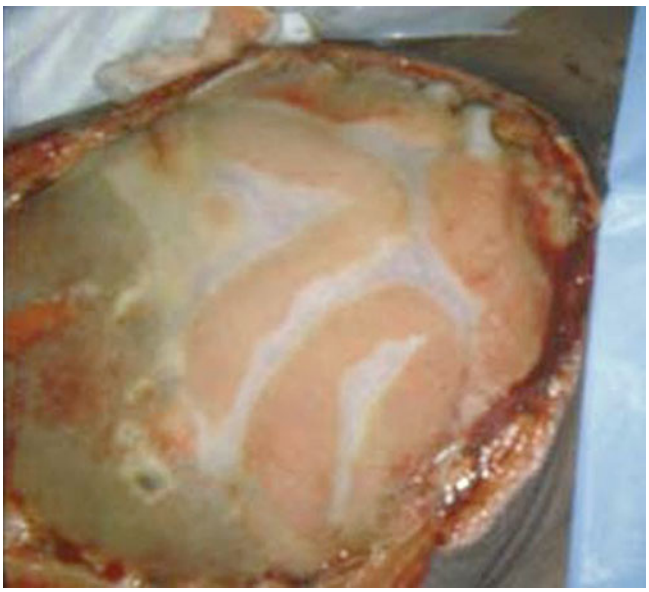


Fig. 8.4 Temporary abdominal closure with a Vicryl mesh

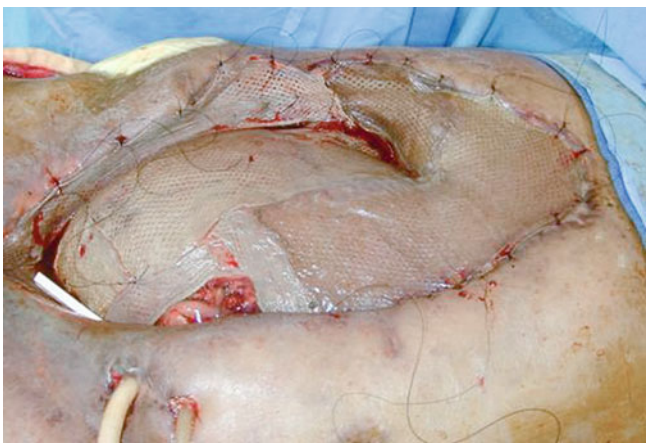


Fig. 8.5 Skin grafting over the naked bowel after the wound has been cleaned and is granulating

If skin closure and fascial closure are not possible, a temporary mesh such as Vicryl® (Ethicon) (Fig. 8.4) may be used. The goal of the Vicryl mesh is cover the intestines and lessen the chance of enteroatmospheric fistula. The Vicryl mesh will also allow for granulation tissue over the bowel. Once the abdomen has "granulated," the wound can be grafted with skin (Fig. 8.5). These patients by necessity will have large hernias that can be dealt with at a later date.

Biologic mesh (Fig. 8.7) is not typically recommended as a "bridge" to definitive closure. The biologic mesh is expensive, and with all of the other options listed, this plays little role in the management of the open abdomen.

Component separation (Fig. 8.8a, b, c) may be undertaken for definitive closure of the open abdomen in those patients for whom tension-free fascial approximation is not a viable option.

Fig. 8.6 (**a**) ABThera open abdomen negative-pressure system. (**b**) Cross-sectional view (Courtesy of KCI, San Antonio, TX)

Component separation might be the best way to "reapproximate" the midline. Preoperative evaluation may require a computed tomographic (CT) scan to confirm that the patient does in fact have an intact rectus abdominis muscle and fascia. Full component separation may allow reapproximation of fascia that is as much as 20 cm apart. The operation is begun by raising subcutaneous flaps just above the rectus sheath fascia, from the level of the costal margin to the level of the pubis, and

laterally to the anterior axial line. An incision is then made in the external oblique aponeurosis 2 cm lateral to the semilunar line. The external oblique muscle is then dissected free from the underlying internal oblique muscle. This allows for the rectus to "slide" over to the midline. Further release can be accomplished by incising the posterior rectus sheath. The operating surgeon should realize that component separation is a "one-shot deal," and that conditions such as infection and nutrition must be optimized before attempting this.

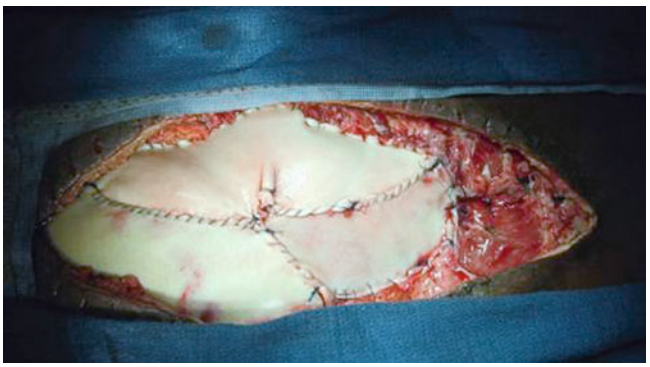


Fig. 8.7 Biologic mesh for the closure of fascial defect

Management of Complications of Open Abdomen

Abscess

Intra-abdominal abscess formation is a common complication in the patient treated with open abdomen technique. The current management of intra-abdominal infection includes immediate resuscitation, prompt source control, and appropriate use of antibiotics. For patients with septic shock, fluid resuscitation should begin immediately if hypotension is present. Fluid resuscitation should be combined with vasoactive drugs if necessary. Ultrasound or CT-guided percutaneous abscess drainage should be used when possible in this situation for source control. Rational use of anti-infective drugs could prevent prevalence of antibiotic-resistant strains of bacteria. Scheduled "washouts" of the patient with open

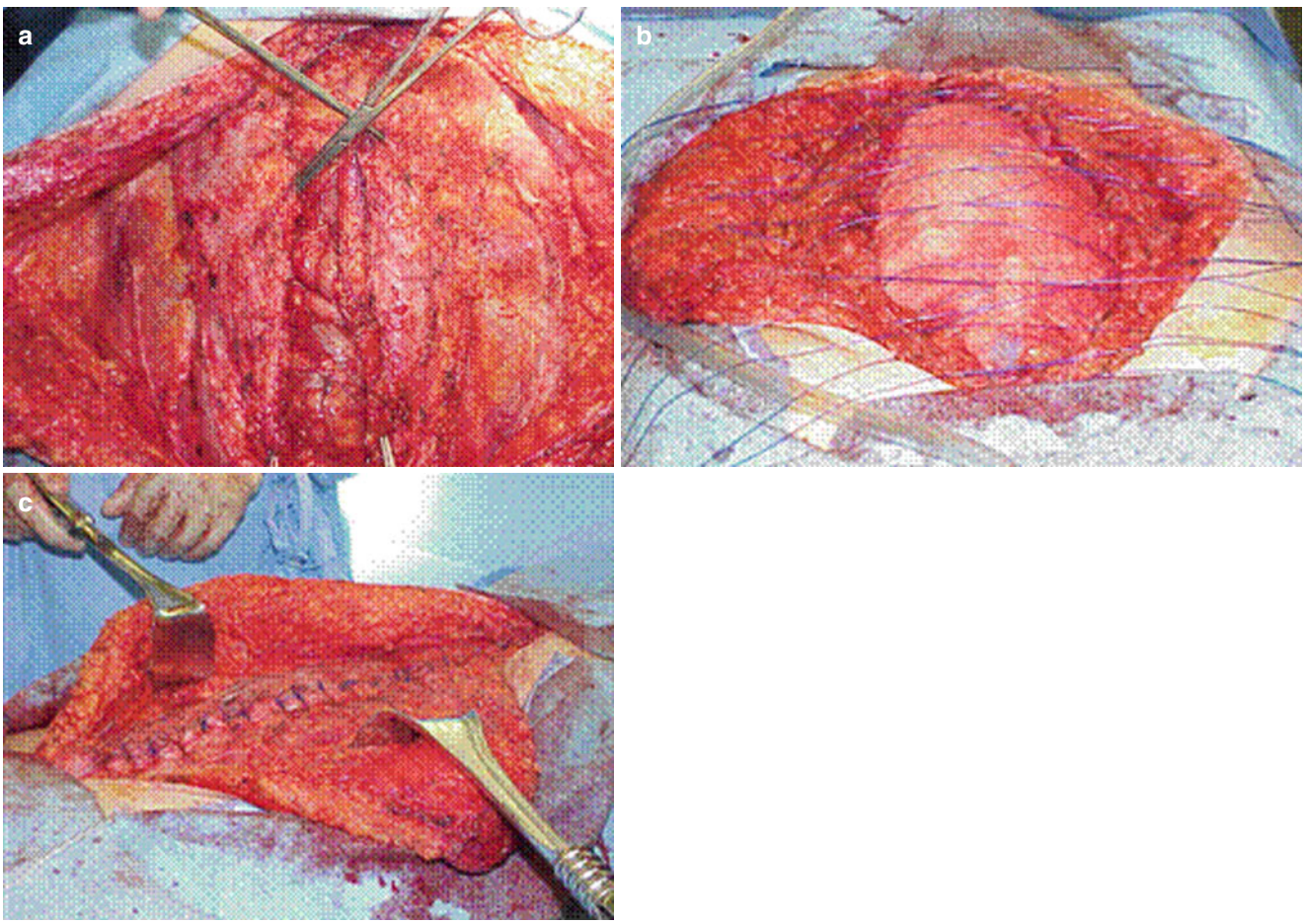


Fig. 8.8 (**a**–**c**) "Component separation" to close fascial defect

abdomen may serve to reduce the risk of morbidity from the development of intra-abdominal abscess formation. Utilization of "repeat washout" in severe necrotizing pancreatitis has correlated with decreased mortality, although there are mixed results in less-severe cases. Staging the abdominal reconstruction serves three main functions: (1) reduced contamination and controls intra-abdominal sepsis; (2) debridement of devitalized or contaminated tissue; and (3) reconstruction. Source control remains the priority in most patients managed with an open abdomen. Clinical parameters such as renal dysfunction, Acute Physiology and Chronic Health Evaluation (APACHE II), and multiorgan dysfunction score (MODS) might be predictive of ongoing intra-abdominal sepsis and can be used as indications for relaparotomy. Those patients with advanced ventilatory demands may safely undergo bedside relaparotomy with risks similar to those in the operative theater.

Hernia

Hernia is a common complication after DCS. In some cases, such as the patient left with a skin-only closure or the patient with Vicryl mesh closure followed by skin grafting (Fig. 8.5), development of hernia is expected and can be dealt with as an outpatient (Fig. 8.9).

In other cases, development of hernias is an unexpected complication. Though fascia may have been "brought to the midline" at the closure case, weak fascia or wound infection can predispose the patient to incisional hernia formation. When this happens, a discussion needs to take place with the patient in an outpatient setting regarding whether the hernia should be repaired. Simple presence of a hernia, for example, does not demand operative repair. Consideration of symptoms, such as pain, obstruction, enlargement, or interference with the patient's activities of daily living must be taken into account. Repair of incisional hernia in the patient treated at one point with open abdomen demands that the fascia be reapproximated to the midline, and this may require advanced techniques, such as component separation, with or without the addition of a permanent mesh.

The implantation of a permanent mesh is not without risk. All types of permanent mesh, including Parietex™ (Covidien, Dublin, Ireland) and Gore-Tex® (W.L. Gore & Associates, Neward, DE), are always at risk for mesh infection. Mesh infection might be treated by antibiotics, but ultimately the treatment for this problem may require removal of the mesh, which by definition will leave the patient with a hernia again. Repeat implantation of mesh to take care of a hernia that contained previously infected mesh is doomed to fail. The adage "once infected, always infected" reigns true in this case.

If a biologic mesh is used to repair an incisional hernia, development of mesh diastasis is a concern. This "stretching" of the biologic mesh creates a bulge in the midline that is not, in fact, a hernia. Reoperation for mesh diastasis is seldom warranted.

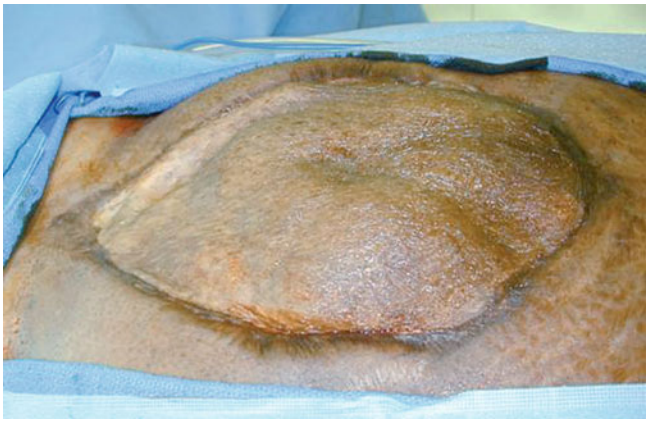


Fig. 8.9 Ventral hernia, created by skin grafting over an open abdomen, now ready for a delayed fascial closure

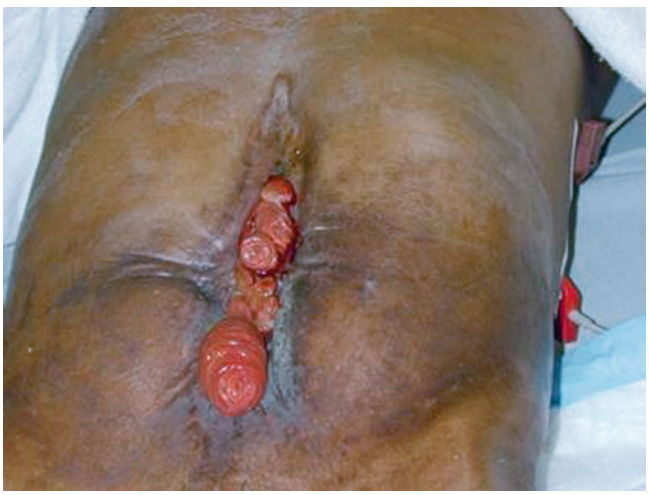


Fig. 8.10 Multiple enterocutaneous fistulas in the open abdomen. With local wound care, the fistulas were controlled, and the wound was allowed to heal. The patient underwent subsequent resection of fistula and anastomosis

Fistula

Enteroatmospheric fistulas are fistulas (Figs. 8.10 and 8.11) that occur in the midst of an open abdominal wound and pose many challenges in their management. The prevention of EAFs is highly important and is the most effective treatment. Coverage of exposed bowel using greater omentum or biologic dressings protects the bowel and prevents fistula formation. Negative-pressure dressings and gauze dressings should not be in direct contact with the bowel. Care should be taken during dressing changes to reduce risk of serosal injury, which may lead to fistula formation later. Once the fistula is

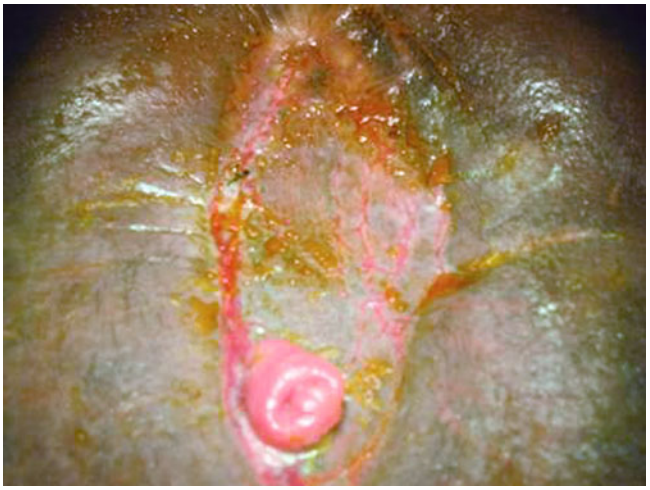


Fig. 8.11 Enterocutaneous fistula in an open abdomen. Local wound care and skin grafting of the rest of the wound allowed control of the fistula for a subsequent successful resection and enteric anastomosis

formed, attempts to close a fistula in the midst of an open abdomen are usually unsuccessful. Occasionally, fibrin glue and acellular dermal matrix can seal a small EAF; if the fistula occurs in an open abdomen that has not yet been sealed by adhesions, continued contamination will occur and predispose to intra-abdominal abscesses and sepsis.

Exteriorization of the fistula and proximal diversion are obviously the best solutions, but these are also often impossible because of massive edema and foreshortening of the mesentery. In such cases, the goal of therapy is to control the fistula effluent from contaminating the rest of the peritoneal cavity. Wound care consultants are helpful to accomplish this, and they have many "tricks" to seal the rest of the abdomen by isolating the fistula and treating it as a stoma.

The "floating stoma" is an interesting solution to this problem and consists of suturing the edges of the fistula to the plastic silo used for temporary coverage, creating a controlled stoma over which a stoma bag can be applied. If these measures fail, the only option is manual evacuation of the contaminating fluids by daily irrigation and watchful vigilance to identify and control sepsis. Once fistula isolation is accomplished and nutrition is optimized, the open abdominal wound is, essentially, a carpet of granulation tissue covering the exposed bowel and the omentum. The peritoneal cavity is sealed, and ongoing peritoneal contamination is not a major issue. However, control of the effluent and protection of the adjacent skin can still be problematic. In such cases, currently available VAC management is effective to control the fistula effluent. In a few cases, small fistulas may eventually close with this vacuum therapy. If this fails, coverage of the granulating abdomen around the fistula by skin grafts (Fig. 8.5) or mobilized skin flaps is the next step.

Occasionally, an open abdomen and fistula can be managed by soft tissue coverage with fascia or even skin as previously discussed, combined with fistula intubation to create a drainage tract. The fistula may have a chance to heal because of the coverage by well-perfused soft tissue. As mentioned, the combination of open abdomen and fistulas is extremely catabolic, and patients need to be supported by aggressive nutrition. If the fistula allows, enteral nutrition may take place by well-placed feeding tubes. Otherwise, total parenteral nutrition (TPN) might be necessary for the long term, which is best accomplished by peripherally inserted central catheter (PICC) lines and home TPN. These procedures often require extensive planning for complex abdominal wall reconstruction in close collaboration with a plastic surgery team.

Conclusion

The open abdomen technique has many excellent applications in the management of trauma and critical illness. Careful attention to detail, focusing on the physiology of the patient, will provide optimal outcomes and minimize complications. Experience is essential, and a multidisciplinary approach is crucial for success.

References

1. 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 take-back. J Trauma. 2011;70(6):1429–36.
2. Hatch QM, Osterhout LM, Podbielski J, Kozar RA, Wade CE, Holcomb JB. Impact of closure at the first take back: complication burden and potential overutilization of damage control laparotomy. J Trauma. 2011;71(6):1503–11.
3. Ivatury RR, Cheatham M, Malbrain M, Sugrue M. Abdominal compartment syndrome. Austin: Landes; 2006.
4. Ivatury RR. Update on open abdomen management: achievements and challenges. World J Surg. 2009;33(6):1150–3.
5. Boele van Hensbroek P, Wind J, Dijkgraaf MG, Busch OR, Goslings JC. 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.

Practical Approach to Patient with a Hostile Abdomen

9

Rifat Latifi and Ari Leppäniemi

Introduction

A hostile abdomen is defined as an abdomen that we as surgeons cannot enter freely, mostly because of adhesions (often fibrotic) that make the abdomen look like a frozen lake (Fig. 9.1a, b). Terms like *hostile*, *frozen*, *inaccessible*, and *difficult* are used interchangeably. Given its daunting nature, a hostile abdomen has been called a "disastroma." We also have called it "stoma city" (Fig. 9.2a, b). Most often, a hostile abdomen is associated with large abdominal defects [1]. The most complex cases of patients with hostile abdomen are associated with enterocutaneous fistulas (ECFs), enteroatmospheric fistulas (EAFs), or stomas (Fig. 9.2a, b). Often, patients with a hostile abdomen have lost the abdominal wall domain as a consequence of multiple operations and open abdomen management, resulting in severe fibrosis and a "cement-like" abdomen [2] (Fig. 9.3a, b, c).

R. Latifi, MD, FACS (✉)
Department of Surgery, University of Arizona,
1501 N. Campbell Ave, Tucson,
AZ 85724, USA

Trauma Section, Department of Surgery,
Weill Cornell Medical College,
Hamad General Hospital, Doha, Qatar
e-mail: latifi@surgery.arizona.edu

A. Leppäniemi, MD, PhD
Department of Abdominal Surgery,
Meilahti Hospital, University of Helsinki,
Haartmaninkatu 4, 340, Helsinki,
FIN-00029 HUS, Finland

Department of Emergency Surgery,
Meilahti Hospital, University of Helsinki,
Haartmaninkatu 4, 340, Helsinki,
FIN-00029 HUS, Finland
e-mail: ari.leppaniemi@hus.fi

Key Questions

In the care of patients with a hostile abdomen, the most serious question that we as surgeons must answer is: When should we operate, if at all? In other words: How long should we wait until we think it is the optimal time to operate? This question assumes other question: What should we tell the patient? How do we know that "things will get better with time" in an abdomen that looks like one large stoma city?

Subsequent questions (dealt with throughout this book) are also important: What surgical technique(s) should we use in approaching and repairing massive abdominal defects? What kind of mesh should we use? How should we place and fix the mesh? However, no question is as important as the timing of the operation, and that is the focus of this chapter. None of the many questions regarding a hostile abdomen has a straightforward answer [3]. Multiple factors affect our decisions, as surgeons, to wait to intervene, to adopt one technique or another, to use a certain mesh or another. Such factors include the individual patient's anatomy, physiology, and religious beliefs, but most critical are the surgeon's expertise, the hospital resources, and the support staff. A particular issue that is not often written about is the overall coping capacity—of both the patient and the surgeon—with the pathology at hand.

Preoperative Conditions

For the purposes of this chapter, we assume that patients have overcome the acute phase of their disease and have entered what we call a "status quo surgical condition." Each patient, however, must be evaluated and treated individually. For example, patients with a hostile abdomen who are acutely septic (from line sepsis or even from intra-abdominal sepsis) need sepsis control and should not be subjected to any major

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_9,

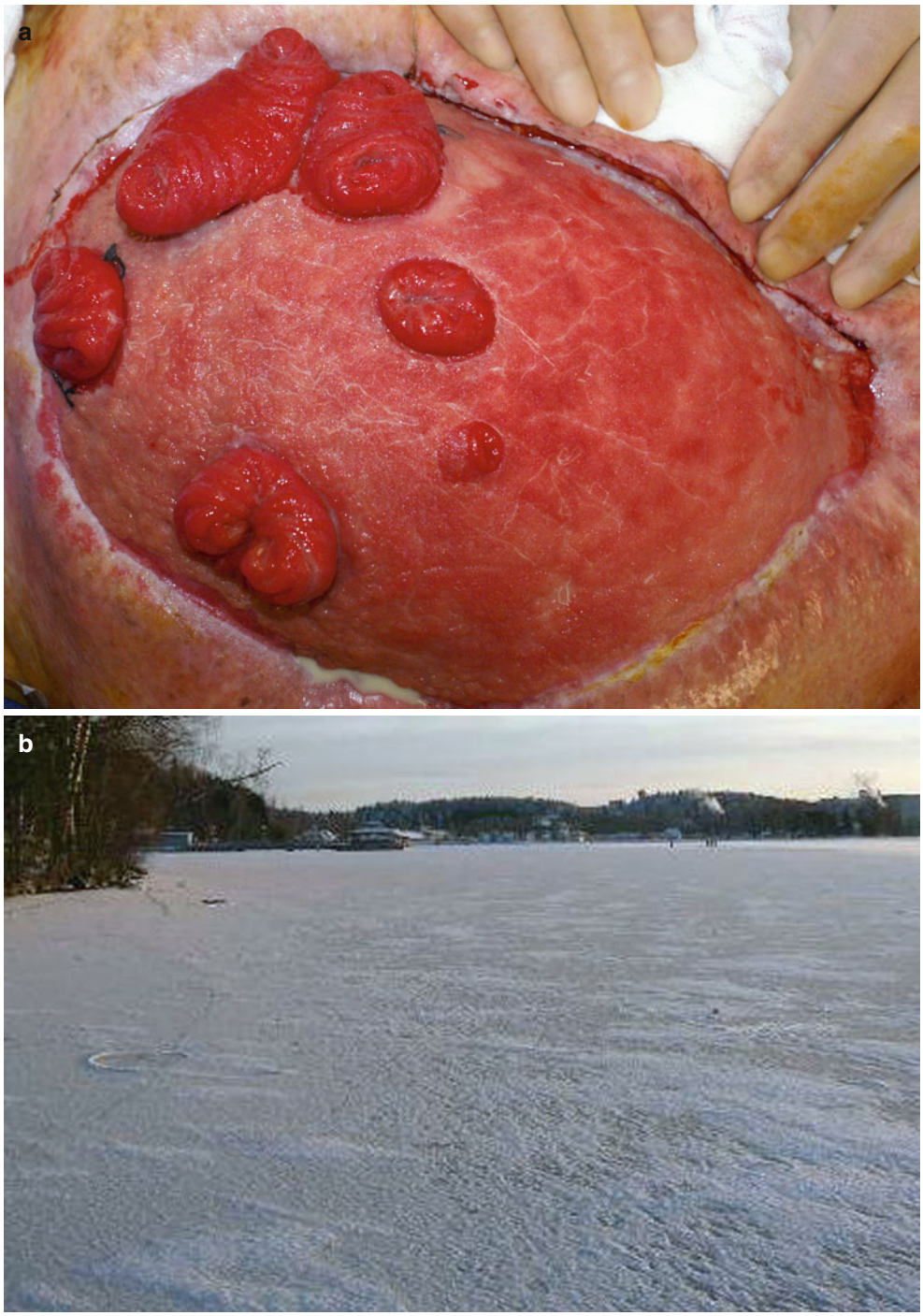


Fig. 9.1 (**a**) "Frozen" abdomen with multiple fistulas following open abdomen managed by a wound VAC. (**b**) Frozen lake in Finland

insult (i.e., to any sort of definitive operation). The same applies to patients with a hostile abdomen who are gravely malnourished; who have major abnormalities in their levels of electrolytes, trace elements, and vitamins; who are severely anemic; or who are in a state of depression [4].

All such preoperative conditions need to be addressed and corrected before definitive surgery is contemplated. In particular, the patient must be in an appropriate state of mind, completely ready for the operation. Usually, patients with a hostile abdomen have already gone through a lot; they need to understand fully what is at stake and what the surgical complications and benefits might be. The bigger dilemma, however, is with patients who think they are ready and wish to have the operation yet are still extremely ill and mired in a complex medical and surgical situation that offers no clear-cut answers. Next, we discuss three different

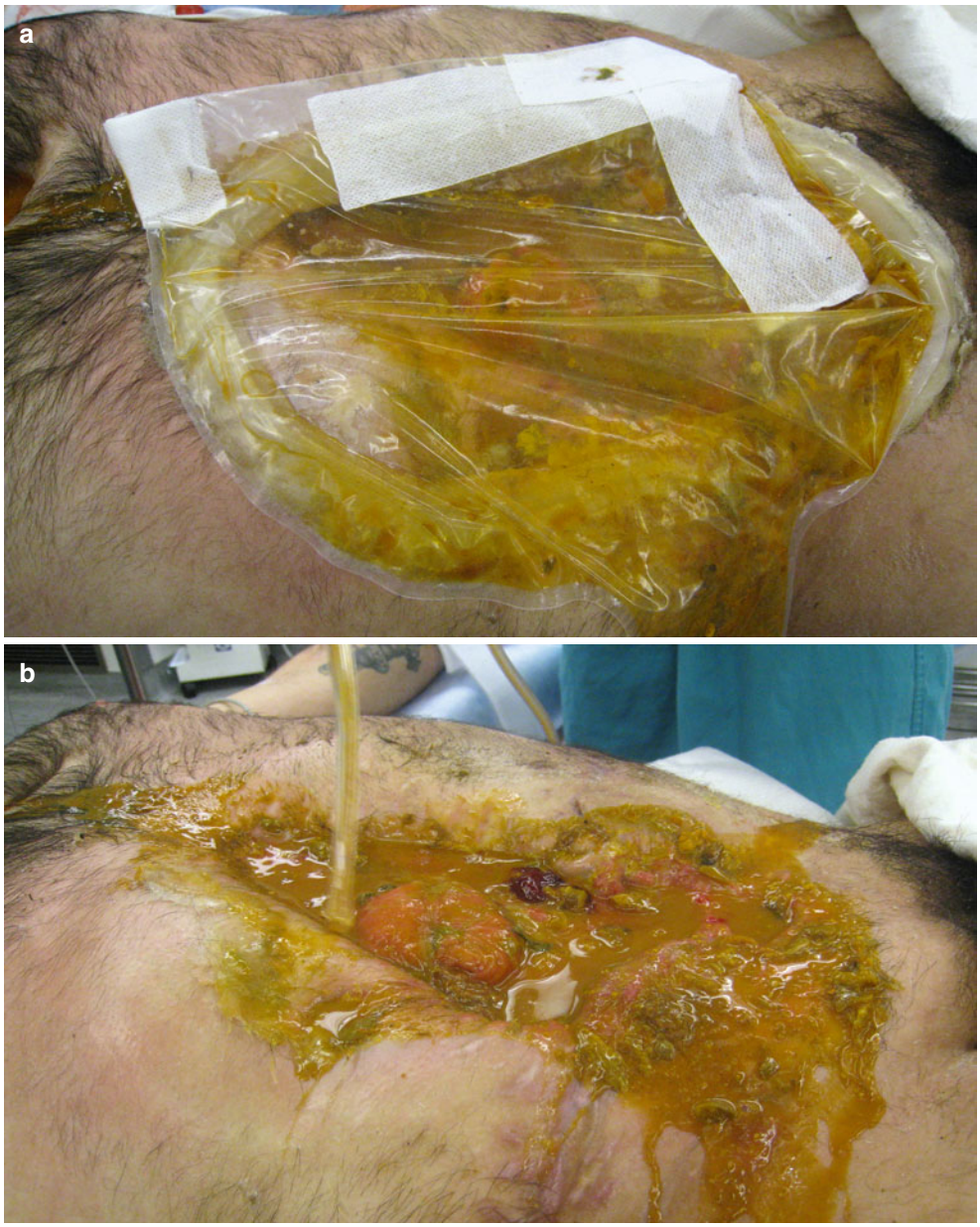


Fig. 9.2 (**a**, **b**) Twenty-four-year-old gentleman, status post high-velocity gunshot wound, following multiple operations and multiple enteroatmospheric fistulas managed as a single large stoma of the abdomen

scenarios, each a short case report describing real-life patients with a hostile abdomen, each requiring a different approach. Dissecting each of these scenarios, we hope, will help practicing surgeons implement the right action steps and tailor the ideal surgical approach for their own patients.

Scenario 1

A 41-year-old man has survived intra-abdominal sepsis after a catastrophic traumatic event that led to right hip disarticulation and open abdomen management. He now has a hostile abdomen with a few stomas and mushroom-like fistulas that drain a moderate amount of succus entericus, which is being managed by an individually tailored stoma bag (Fig. 9.2a). Although his sepsis has been controlled and electrolyte and fluid levels have been normalized, he is on total parenteral nutrition (TPN) and is unable to eat or to use his gastrointestinal (GI) tract. He lives in an extended care facility and has been out of work for almost a year. In recent months, on a regular basis, he has dealt with multiple nutritional deficiencies of trace elements, proteins, and fatty acids and with multiple bouts of line sepsis. Now, he wishes to be "put together."

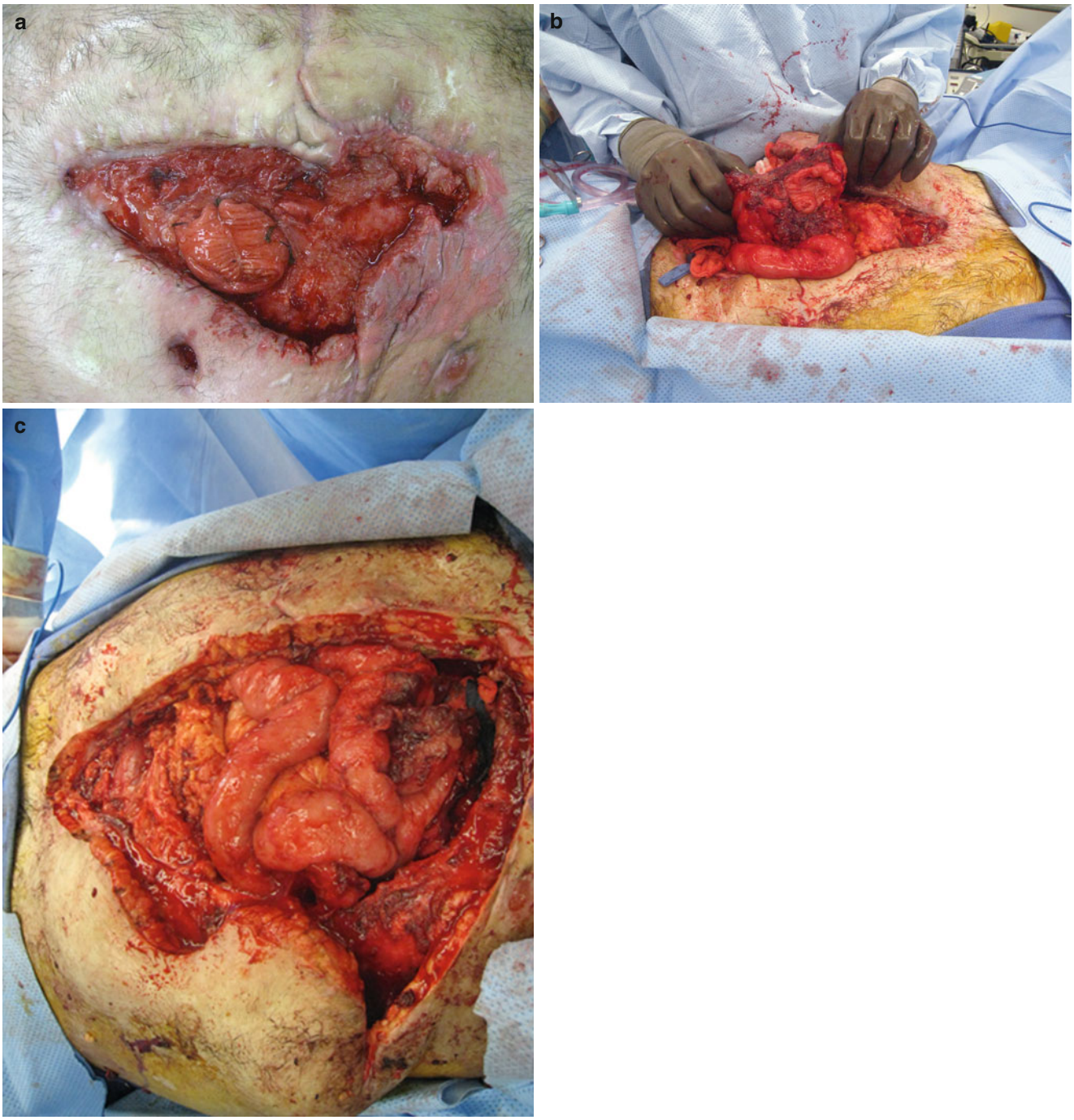


Fig. 9.3 Intraoperative view from the same patient as in Fig. 9.2a, b. As can be seen, he lost a significant mass of abdominal wall and has a fibrotic, cement-like, abdomen (**a**). (**b**) Multiple enteroatmospheric fistulas are identified. (**c**) Intraoperative view at the end of establishment of GI continuity but before abdominal wall reconstruction

Scenario 2

A 45-year-old morbidly obese woman has a giant abdominal wall defect after open abdomen management for trauma. That defect has been covered with a healed-over skin graft. She now requests reconstruction and abdominoplasty at the same time. She cannot work, cannot exercise, and wishes she were dead instead.

Scenario 3

A 58-year-old man has a colostomy 6 months after catastrophic intra-abdominal sepsis. His surgeon already attempted to reverse the colostomy (which the patient had undergone for perforating diverticulitis), but a leak occurred that required multiple abdominal washouts and, finally, diversion. The patient is now requesting another attempt to

reverse the colostomy. He cannot work, has no sex life, and is miserable at all times.

Creating a Surgical Plan

All three patients, although different clinically, have one thing in common: They want to be put together to move on with life. We as surgeons—when faced with sepsis, when faced with acute respiratory distress syndrome (ARDS), when faced with multiple-organ failure—tend to turn to one main strategy of action, namely, source control. For the problems of these three patients, source control is not different, but whether or when to operate (in an effort to achieve source control) is a fraught issue in such patients. Most surgeons who deal with complex abdominal defects probably have seen patients like these three often. For most such patients, it is basic human instinct to "forget" bad times and wish to "move on." Thus, most of them do not focus on the weeks or months in the surgical intensive care unit (ICU); all of the trips to the operating room; painful dressing changes; multiple episodes of fever, line sepsis, and blood draws, often every few hours; computed tomographic (CT) scans; and long days on many drugs and on TPN. They do not focus on all of the strangers—doctors, nurses, respiratory therapists, dietitians, and others—helping them every moment while they were really sick. All they remember now is that "this is not the way to live," and that they need an operation to be "fixed." The decision to operate is not an easy one, however.

The patient in scenario 1 patient represents the most difficult case: He needs to be treated like a patient with short-gut syndrome [5]. Most fistulas, especially high-output fistulas, require surgical treatment [6–14] and continuous meticulous attention to avoid or at least minimize sepsis (such as catheter-related sepsis in patients on TPN), electrolyte and fluid disturbances, and malnutrition [15]. So, when we are going to operate is really a matter of time. Week after week, the surgeon sees this patient in clinic. The patient looks sick, frail, and wasted, although he is not febrile. His muscle mass is shrinking, his abdomen looks like a large "stoma city," he has a zinc and fatty acid deficiency, and he wants to have his stomas reversed and move on with his life. With few exceptions, there are no published reports or recommendations that are of any use: The vague statements in the literature, which are not based on clear evidence, advise waiting until "nutritional support is adequate," "sepsis is controlled," "as long as possible," or "for the first 12 months." Such statements do not help in practical terms. No predictive index or score exists; no strategy has been tested in large-scale, randomized clinical trials. This patient is young and wishes to return to work and life, but he is not ready. Further waiting will only expose him to line sepsis, bacteremia, hospitalization, skin excoriations, foul odors, and possibly death. Operating, on the other hand, undoubtedly will expose him to a huge physiologic insult and probably to an anastomotic leak, further sepsis, a long intensive care stay, and a high risk of death.

Involving the Patient

To the patient in scenario 1, the first thing that the typical surgeon tends to feel like saying, in accord with the prevailing surgical dictum, is the following: "No, sir, no. I am not going to operate on you. If I do, you will die." But then, the patient says that, actually, he does not see himself anymore as a live human being. Things begin to change as the surgeon sits by the patient's bedside, smells the intestinal fistula output, looks at the patient's excoriating skin, and really sees the dying man; then, the surgeon vows to attempt to achieve source control, no matter what, because otherwise the patient is doomed to a slow death. The limited available evidence [16] suggests that when sepsis is controlled, nutritional status is optimized, and anatomy is defined, the surgeon may decide to operate on fistulas such as those of the patient in scenario 1. The patients in scenarios 2 and 3 are difficult as well. We do expect a patient such as the one in scenario 2 will lose enough weight to be ready for an operation. However, insisting on a huge amount of weight loss is not practical; it does not help and can be demoralizing to the patient. It is unfortunate, but these patients do enter a cycle without exit. Source control, via the operation, is the goal. For the patient in scenario 3, we would try to postpone as long possible, but at least 12 months, after the last operation.

In all cases like these, we have used a strategy that puts the patient partially in charge. If the surgeon is honest with patients with a hostile abdomen, most of them will understand (and, in fact, basically knew, on some level, all along) all the possible outcomes of either further waiting or surgery. In our practice, we explain to each patient, with the utmost compassion as well as clarity, that three main outcomes are possible with surgery:

1. My team and I might be able to complete our task to our satisfaction, that is, perform lyses of adhesions, take down stomas or fistulas, restore the continuity of the GI tract and reconstruct the abdominal wall, and then oversee a postoperative course that leads to good recovery, without major incidents.
2. My team and I may not be able to do any of what is specified in outcome 1 and in fact make the patient worse.
3. My team and I may successfully complete the initial operation, but the fistulas may recur, the anastomoses may leak, and a serious wound infection may develop that requires removal of the mesh, so that basically we end up back at square one; or, even worse, all of the possible complications prove fatal.

We review those three outcomes many times, not only with the patient but also with the family or friends and with the residents, medical students, fellows, and nurses on the team. It is imperative to go over all elements carefully, with absolute sincerity and empathy and without rushing. Proper mental preparation is essential for the surgeon and surgical team as well as for the patient, family, and friends.

Timing of the Operation

Once surgery is agreed to, then the team sets up a time—in 1 month, in 2 months, whatever works for everyone. Overall, with regard to the timing of the operation, there are two camps of surgeons: those who wait until things are "settled down" and those who choose to operate early [6–14] Because postoperative complications such as recurrence of ECFs are a major problem, the essential question is when to "attack" a hostile abdomen with ECFs or stomas that cannot be managed nonoperatively. Surgeons have wrestled with when to operate and how to succeed for years. Some authors have suggested waiting 4–5 weeks before operating, just long enough to make sure that patients are nutritionally sound and sepsis is controlled. Most surgeons, however, wait 3–6 months; others wait 12 months or more. In our practice, we choose to operate early (but not within the first 2–3 months if possible); what is "early" has not been defined clearly in the literature. Rather, the decision is clinical, based on the individual patient.

Preparing for the Operation

Ideally, the anatomy of complex ventral hernias, ECFs, and EAFs can properly be identified (preoperatively, if at all possible). Any previous operative reports should be obtained and studied. On many occasions, however, the surgeon must make a difficult decision even without completely discerning the anatomy, even without having a surgical road map in advance. Intraoperatively, the anatomy will become clear. In preparation for the operation, key laboratory and clinical issues must be addressed. For example, patients' blood sugar levels must be controlled. They must stop smoking. The bioburden must be reduced through wound vacuum-assisted closure (VAC) or through other stoma protection techniques. Hypovolemia and chronic anemia must be corrected. A complete biochemical profile (including levels of trace elements, vitamins [especially vitamin C], and essential fatty acids) must be obtained and any problems resolved. During these significant preoperative weeks, we make sure that the patient is receiving intensive nutritional therapy, extra doses of vitamins and trace elements, wound care, and physical therapy. The week before the operation and on the day of the operation, we review with the patient the entire proposed surgical procedure; we also check all laboratory data, ensure appropriate transfusion of blood and blood products, and discuss the case with the nursing staff and the anesthesia staff. We make sure that no other operations are on our schedule that day, and that we have no other clinical, administrative, or family obligations. It will be a long day, a surgical marathon. We also make sure to be in town for at least a week to 10 days after the operation.

Entering the "Frozen Lake"

In patients with a hostile abdomen and with ECFs or EAFs, the open surgical approach is standard. Often, not even millimeters of tissue are able to be dissected with each surgical move, made bluntly by fingers, scissors, or even a scalpel (Fig. 9.3a, b, c). Most important, dissection must be performed carefully and systematically, yet with the surgeon's intent on finding the easiest way and moving around, rather than insisting on completing one section at a time. Proceeding wherever it is easiest may minimize iatrogenic injuries to the bowel, blood vessels, liver, splenic capsule, and other organs and structures. It is paramount to handle the tissue gently and with the utmost care. Each surgeon will use creativity and a combination of different techniques and repairs—depending, primarily, on the mission at hand. If the goals of the operation are to take down fistulas, establish GI tract continuity, and concomitantly repair abdominal defects, then things do become more complicated, and the surgeon should plan accordingly. The majority of patients with ECFs or EAFs have a hostile abdomen, so entering the abdominal cavity will be extremely challenging. Whenever possible, the surgeon should avoid entering the abdomen initially through the same incision used in prior operations. Instead, attempts should be made to enter the abdomen from nonviolated areas of the abdominal wall, superiorly or inferiorly to the extent possible. Some authors have suggested alternative methods of entering the abdomen, such as through a transverse incision. In our practice, we have not used a transverse incision in patients with a hostile abdomen. Instead, when such an abdomen is covered with a skin graft, we prefer a meticulous dissection on either side of the abdomen. During skin and fascial dissection, however, the utmost care must be taken to avoid injury to the underlying bowel; the consequences of inadvertent enterotomies are not trivial [17]. If an enterotomy is recognized, it should either be repaired at once or be marked with a silk suture for later identification.

Mobilizing the Entire GI Tract

Most authors agree that the entire GI tract must be mobilized and identified from the gastroesophageal (GE) junction to the rectosigmoid junction. All adhesions must be taken down using a sharp or blunt finger technique. Often, this is easier

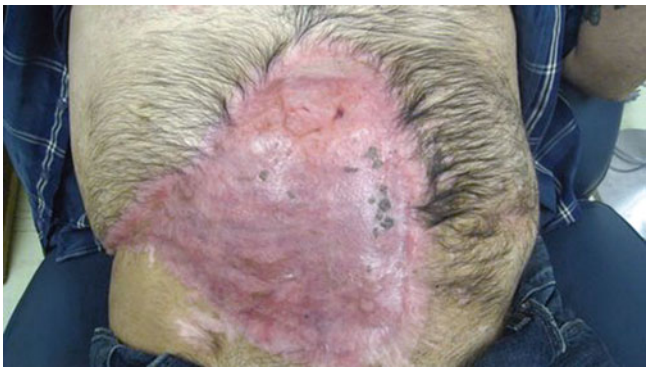


Fig. 9.4 Same patient as in Fig. 9.3a, b, c 6 months postoperatively following reconstruction

said than done. Surgical discipline must be executed, especially early in the surgical career and in any operation when things are not going well. The intestines, especially as they become swollen (e.g., from intraoperative fluids), are prone to injuries, so they must be handled gently. Furthermore, intestines that have not been used for a long period are thinner; they are easy to penetrate or avulse, even with finger dissection.

How Much of the Intestines to Resect and How to Create the Anastomoses

The two most important decisions concern the length of intestines that should be resected and the number of anastomoses that can be safely performed. Questions abound: Should large segments of small bowel be resected, potentially creating GI-crippled patients with possible short-gut syndrome? Should more than two or three anastomoses be created, running the risk of a leak or an ECF? Or, should the number of anastomoses be minimized? The senior surgeon should answer those questions by balancing the risk and benefits of the procedure. One good thing is that intestines look shorter than they in fact are. If at least 20–25 cm can be left between the anastomoses, a hand-sewn, double-layer technique should be used.

Close or Cover the Abdomen

Like most current surgeons, we do not favor leaving these patients postoperatively with an open abdomen. In an attempt to reduce the frequency of an open abdomen, those of us in the trauma community in particular have changed our mindset toward such patients. We have all departed from a "leave-them-open" to a "sew-it-up" strategy. Closing the abdomen as soon as technically and physiologically possible is becoming the new standard [18]. The postoperative care of these patients is complex, requires a multidisciplinary approach, and is detail oriented, so it must be vigilantly overseen by the operating surgeon at all times. Often, the abdominal wall defect is covered with a skin graft (Fig. 9.4).

Summary

Approach to patient with potentially hostile abdomen needs to be planned and carefully executed, as it involves a violated and changed anatomy and physiology as well as potentially significant complications both intraoperatively and postoperatively. A close partnership with the patient and his/her family are mandatory. Timing of the operation and preparation for the operation are also key elements of this multidisciplinary approach. Although each patient needs to be treated individually, the key principles such as optimal nutrition support, surgical discipline and aggressive preoperative support are the same for every patient. Utmost surgical discipline and creativity, often will play a major role on the patient's outcome. As there is a lack of literature on the timing of take-down of fistulas, surgeons are required to individualize the care of each patient and their surgical approach.

References

1. Latifi R, Gustafson M. Abdominal wall reconstruction in patients with enterocutaneous fistulas. Eur J Trauma Emerg Surg. 2011; 37:241–50.
2. Latifi R, Turegano F. Current management of enterocutaneous fistulas. Eur J Trauma Emerg Surg. 2011;37:207–8.
3. Leppäniemi A. The hostile abdomen—a systematic approach to a complex problem. Scand J Surg. 2008;97(3):218–9.
4. Lord Jr JW, Howes EL, Jolliffe N. The surgical management of chronic recurrent intestinal obstructions due to adhesions. Ann Surg. 1949;129(3):315–22.
5. Dudrick SJ, Maharaj AR, McKelvey AA. Artificial nutritional support in patients with gastrointestinal fistulas. World J Surg. 1999;23(6):570–6.
6. Ramsay PT, Mejia VA. Management of enteroatmospheric fistulae in the open abdomen. Am Surg. 2010;76(6):637–9.
7. Campos AC, Andrade DF, Campos GM. A multivariate model to determine prognostic factors in gastrointestinal fistulas. J Am Coll Surg. 1999;188:483–90.
8. Hollington P, Mawdsley J, Lim W, Gabe SM, Forbes A, Windsor AJ. An 11-year experience of enterocutaneous fistula. Br J Surg. 2004;91:1646–51.
9. Edmunds LH, Williams GM, Welch CE. External fistulas arising from the gastrointestinal tract. Ann Surg. 1960;152:445–71.
10. Berry SM, Fischer JE. Classification and pathophysiology of enterocutaneous fistulas. Surg Clin North Am. 1996;76:1009–18.
11. Evenson AR, Fischer JE. Current management of enterocutaneous fistula. J Gastrointest Surg. 2006;10:455–64.
12. Soeters PB, Ebeid AM, Fischer JE. Review of 404 patients with gastrointestinal fistulas: impact of parenteral nutrition. Ann Surg. 1979;190:189–202.
13. Levy E, Frileux P, Cugnenc PH, Honiger J, Ollivier JM, Parc R. High-output external fistulae of the small bowel: management with continuous enteral nutrition. Br J Surg. 1989;76:676–9.

14. Schecter WP, Hirshberg A, Chang DS, Harris HW, Napolitano LM, Wexner SD, et al. Enteric fistulas: principles of management. J Am Coll Surg. 2009;209:484–91.
15. Dudrick SJ, Abdullah F, Latifi R, Latifi R. Nutrition and metabolic management of short bowel syndrome. In: Latifi R, Dudrick SJ, editors. The biology and practice of current nutritional support. 2nd ed. Georgetown: Landes Bioscience; 2003. p. 261–74.
16. Visschers RG, Olde Damink SW, Winkens B, Soeters PB, van Gemert WG. Treatment strategies in 135 consecutive patients with enterocutaneous fistulas. World J Surg. 2008;32(3):445–53.
17. Van Der Krabben AA, Dijkstra FR, Nieuwenhuijzen M, Reijnen MM, Schaapveld M, Van Goor H. Morbidity and mortality of inadvertent enterotomy during adhesiotomy. Br J Surg. 2000;87(4):467–71.
18. Latifi R, Joseph B, Kulvatunyou N, Wynne JL, O'Keeffe T, Tang A, et al. Enterocutaneous fistulas and a hostile abdomen: reoperative surgical approaches. World J Surg. 2012;36(3):516–23.

Complex Abdominal Wall Reconstruction: The Plastic Surgeon's Perspective

10

Alexander T. Nguyen, Donald P. Baumann, and Charles E. Butler

Introduction

The goals of abdominal wall reconstruction are to reestablish the integrity of the musculofascial layer and provide external cutaneous coverage. Surgical planning for abdominal wall reconstruction must include the potential for true or relative loss of skin and musculofascial tissue. These tissue defects can be caused by tissue necrosis, infection, resection, incisional hernia with loss of domain, denervation of adjacent abdominal musculature, or scarred, retracted tissues that limit the ability to advance skin and fascia. Local wound conditions, including bacterial contamination, previous surgeries, and previous radiotherapy, can contribute to an increased risk of compromised wound healing, surgical site infection, and failure of the reconstruction. Patient factors such as advanced age, comorbidities, obesity, immunosuppression, poor nutritional status, tobacco use, and pulmonary disease also increase the risk of complications and thus must be considered in perioperative planning and management [1–5]. Abdominal wall reconstructive procedures themselves can result in significant complications, including bowel obstruction, fistula, mesh infection, seroma, dehiscence, recurrent hernia, abdominal compartment syndrome, and visceral injury. These may require complex, prolonged, or staged salvage procedures.

Direct suture repair of small ventral hernias can be performed with relatively low complication rates; however, hernia recurrences after direct suture repair are common, occurring in 10–60% of patients [3, 6]. When subsequent recurrent hernia repairs are performed, the recurrence rate increases each time, with progressively shorter intervals between repair and reherniation [7]. The use of mesh reinforcement reduces hernia recurrence rates compared with primary suture repair alone [3, 8]. However, the addition of mesh does not prevent all recurrences. For example, in one long-term follow-up study, synthetic mesh reinforcement during elective repair of small (<6 cm), uncontaminated ventral hernias was associated with a 32% 10-year cumulative recurrence rate [6]. Furthermore, despite advances in surgical technique and implantable mesh materials, other long-term outcomes of ventral hernia repair, including length of hospital stay and need for reoperation, have not significantly improved over time [2]. In addition, the incidences of surgical site occurrences, wound dehiscence, wound infection, seroma, and fascial separation in elective ventral hernia repair are higher than in other "clean" general surgery procedures [2, 3, 5]. In patients undergoing repairs of incisional hernias with previously documented wound infections, up to 41% will develop another wound infection, whereas only 12% of patients without a history of infection will develop a wound infection after hernia repair [2]. Clearly, there are ongoing difficulties with hernia repair, particularly with wound complications and infection, and further improvements are needed.

Various meshes have been developed to improve results in hernia repair. Commonly used implantable meshes include macroporous (monofilament and double-filament polypropylene); microporous (extended polytetrafluoroethylene [ePTFE]); composite (antiadhesive layer laminated to macroporous mesh); and bioprosthetic (decellularized, processed human or animal tissue) meshes [9, 10]. Macroporous meshes have large pore sizes that allow for ingrowth of scar tissue. When placed in contact with abdominal viscera, macroporous meshes are associated with the formation of bowel adhesions, bowel obstructions, and enterocutaneous fistulae [11, 12]. Therefore, these materials should be avoided or used in combination with omental coverage or antiadhesive barriers when placed in contact with bowel. Microporous meshes have a smaller pore size that does not allow for significant tissue ingrowth but may lead to encapsulation, periprosthetic fluid

A.T. Nguyen, MD • D.P. Baumann, MD
C.E. Butler, MD, FACS (✉)
Department of Plastic Surgery,
The University of Texas MD Anderson Cancer Center,
Houston, TX 77030, USA
e-mail: atnguyen3@mdanderson.org; dpbauman@mdanderson.org; cbutler@mdanderson.org

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_10,

collection, and bacterial overgrowth. Therefore, microporous mesh has a lower affinity for visceral adhesions but might be more susceptible to infection. A wide variety of composite materials is now available; these materials combine various qualities, such as having macroporous mesh on one side to promote tissue ingrowth and microporous mesh on the other to reduce the risk for adhesions to the mesh (polypropylene/ePTFE). In an attempt to take advantage of macroporous and antiadhesive characteristics, antiadhesive bilaminar mesh (such as Sepramesh™ [polypropylene/carboxymethylcellulose and hyaluronic acid]; Bard, Murray Hill, NJ) were developed to induce fibrovascular incorporation in the subcutaneous plane and minimize visceral adhesions with the microporous component or bioresorbable component when placed intra-abdominally. Clinical evidence suggests a reduced risk of adhesions for composite and coated synthetic meshes compared with traditional synthetic meshes [13–17]. The reported relative benefits of these different prostheses with regard to adhesion formation and risk for infection vary [11, 15, 18–21].

Bioprosthetic meshes are an equally diverse and expanding class of mesh materials. Certain characteristics are thought to contribute to the successful use of particular biologic repair materials in the setting of wound contamination or low-grade infection. These mesh properties include an intact extracellular matrix and the ability to support tissue regeneration through revascularization and cell repopulation. It has been hypothesized that resistance to infection for some biologic repair materials might be related to the ingrowth of cells and vasculature structures [22]. The neovascularization demonstrated in studies of some biologic repair materials may allow these materials to better resist infection when placed in a potentially contaminated field [22]. Data on the ability of some bioprosthetic meshes to support regeneration come from studies in animal models that describe the immunologic response of the host to the prostheses [23]. It should be emphasized that no clinical trials have been completed to date directly comparing different bioprosthetic meshes in incisional hernia repair, and the few clinical data suggesting benefits of one product over another come from small retrospective studies of a limited number of the available bioprosthetic mesh materials. Data from animal and clinical studies are needed for the majority of bioprosthetic mesh materials.

Current Indications for Utilization of Bioprosthetic Mesh

Indications for the use of bioprosthetic mesh are based mostly on animal data and short-term low-level evidence from clinical studies and case reports. The Ventral Hernia Working Group recommends the following indications [24]:

1. Contaminated wound (existing wound infection, adjacent ostomy, planned or inadvertent disruption of the gastrointestinal tract's continuity, enterocutaneous fistula)
2. Complex repair in a patient at high risk for the development of wound-healing problems, subcutaneous infection, or need for reoperation
3. Planned exposed bioprosthetic mesh or high likelihood of cutaneous exposure (open abdominal wound closure with a bridging repair or unreliable skin coverage in a patient with multiple comorbidities)
4. Unavoidable direct placement of mesh over bowel and other abdominal viscera

Surgeon preference and the variables of any given clinical scenario, including patient comorbidities, wound contamination, prior radiation, availability of omentum, posterior sheaths (retrorectus repair), and the quality of the overlying soft tissue will determine whether bioprosthetic mesh or synthetic mesh is implanted. Regardless of mesh type, the expectations are that the mesh will maintain the abdominal wall contour and not become attenuated, leading to a hernia or bulge. In addition, the mesh should be able to interface with the underlying viscera without forming extensive adhesions or erosion that can lead to fistulization. Both synthetic and bioprosthetic meshes can meet these expectations.

Abdominal wall defects require both reconstruction of the musculofascia and closure of the overlying skin. Musculofascial reconstruction is generally performed with component separation or implantable mesh. Many surgeons have described the use of component separation in complex abdominal wall repair with numerous variations, including reinforcement with mesh [20, 25–27], and component separation is now considered by many experienced hernia surgeons to be the first option [20, 26, 27]. Component separation provides an enhanced fascial closure technique and a dynamic repair of the abdominal wall without compromising motor innervation to the abdominal wall muscles.

Patient Selection

In elective hernia repair, patients should be "optimized" before surgery by improving controllable medical and surgical site comorbidities (see Chap. 5 on preoperative patient optimization). Factors such as serum glucose levels and nutritional status should be brought under control, and tobacco use should be eliminated for at least 3 weeks before surgery to decrease perioperative morbidity [5, 28]. Compelling data from the National Surgical Quality Improvement Program highlight the risk of morbid obesity, which increases perioperative complications and deaths [29]. Obese patients undergoing elective hernia repairs should be screened by a nutritional counselor and enrolled in a diet and exercise program to reduce the risk of complications. Patients

who are unable to lose weight on a personalized plan should be considered and evaluated for surgical laparoscopic bariatric procedures prior to elective hernia repair.

In nonelective or emergent abdominal wall reconstruction, contaminated wounds should be appropriately debrided and prepared to reduce the bacterial bioburden [30]. Severely contaminated wounds may require a staged approach consisting of serial debridement, dressing changes, negative-pressure wound therapy, and delayed fascial closure with bioprosthetic mesh or component separation. Systemically ill patients with numerous comorbidities and contaminated fascial defects may benefit from early musculofascial reconstruction with bioprosthetic mesh. Early fascial closure preserves the musculofascial domain, improves ventilatory support, reduces the risk of enterocutaneous fistula, and may reduce the acuity of subsequent wound management.

Abdominal Wall Reconstruction Principles

The general principles of abdominal wall reconstruction include optimization of the patient, preparation of the wound, centralization and reapproximation of the rectus abdominis muscles along the midline, and the use of appropriate synthetic or bioprosthetic material to reinforce the closure.

A key element of the inset of bioprosthetic mesh is to place the mesh in an inlay position under appropriate physiologic tension; this is in contradistinction to a tension-free repair. Mesh inset under physiologic tension facilitates and stimulates appropriate collagen remodeling to optimize mechanical strength and thus reduce the risk of bulge and laxity. The edges of the bioprosthetic mesh should overlap the undersurface of the musculofascial edge by at least 3–5 cm to allow for remodeling and fibrovascular incorporation. This method of bioprosthetic mesh inlay takes advantage of the mesh's remodeling mechanism, rather than simple scarring mechanisms, and increases the tensile strength of the junction between the mesh and the musculofascia [23, 31, 32].

The anatomic plane of the bioprosthetic mesh inlay has direct implications for the degree of incorporation at the mesh-musculofascia interface (Fig. 10.1). When possible, it is preferable to avoid setting the bioprosthetic mesh in direct contact with the peritoneum or preperitoneal fat. To avoid this, the preperitoneal fat pad is dissected away from the posterior rectus sheath, and the mesh is placed in direct continuity with the posterior sheath fascia. This improves the fibrovascular infiltration and mechanical strength at the mesh-musculofascia interface better than suturing the mesh to the preperitoneal flap/transversalis fascial layer would. Alternatively, a retrorectus repair can be used by which bioprosthetic mesh can be sutured to the semilunar line between the rectus muscle and the posterior rectus sheath.

Onlay mesh placement is technically easier but has several significant drawbacks and is not often recommended. If a reinforced repair is going to be performed, one generally must be able to close the fascia first, which is not possible before mesh placement in many cases of large defects; the inset of the mesh as an inlay actually helps reduce the tension needed to close the fascial defect. Thus, an inlay mesh placement facilitates primary fascial closure, whereas an onlay mesh placement can be performed only after primary fascial closure is achieved. However, there might be some situations for which an onlay repair is the only safe option, such as when it is impractical to reenter a hostile abdomen. Although onlay reinforcement avoids placement of mesh directly against intraperitoneal viscera, its positioning in the subcutaneous space may increase the risk of seroma formation or cutaneous mesh exposure if wound dehiscence occurs.

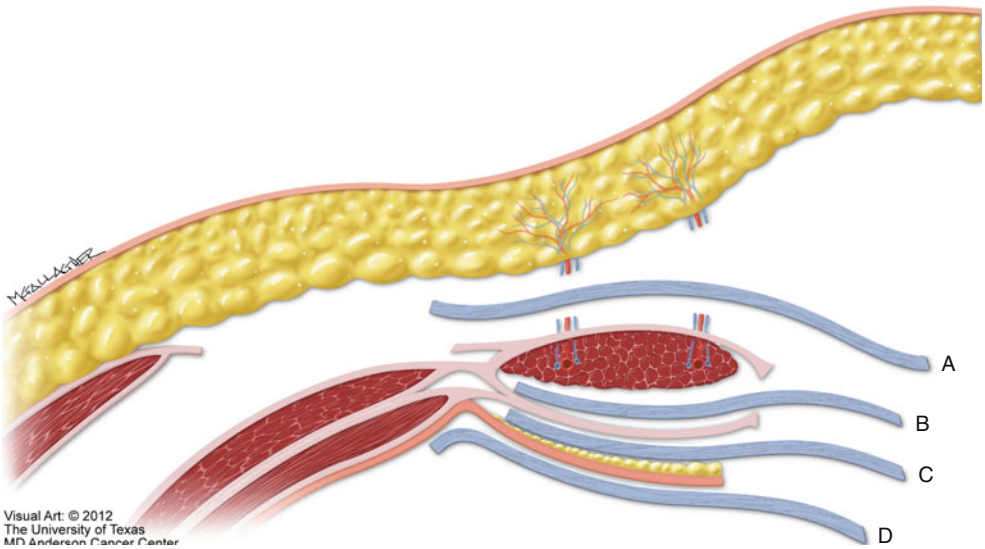


Fig. 10.1 Anatomic planes of mesh inset. *A* Onlay, *B* Retrorectus, *C* Preperitoneal, *D* Intraperitoneal (Copyright © 2012 The University of Texas MD Anderson Cancer Center)

When technically feasible, a bioprosthetic mesh inlay repair should be reinforced with a second layer of primary fascial closure over the mesh (Fig. 10.2). This dual-layer repair is preferred to a bridging interposition repair. Centralization of the rectus abdominis muscle complexes reduces the fascial defect and facilitates primary fascial closure. Primary fascial coverage also allows for complete apposition of the bioprosthetic mesh and the overlying musculofascial defect edge. When primary fascial closure is not attainable, a bridging repair is performed (Fig. 10.3). This is performed using a circumferential inlay technique, which allows two concentric suture lines to affix the bioprosthetic mesh directly to the musculofascia [33–35]. Creating direct apposition of the bioprosthetic mesh and the undersurface of the fascial defect itself prevents the collection of fluid between the two layers, which could prevent or delay fibrovascular incorporation and remodeling. To prevent the collection of fluid at the mesh-musculofascia interface, closed-suction drains are placed between the musculofascia and the bioprosthetic mesh.

Combining bioprosthetic mesh inlay repair with component separation may improve abdominal wall reconstruction outcomes further. Component separation has the ability to medialize the rectus complexes and reduce the defect size, with the ultimate goal of allowing primary fascial closure over the inlay bioprosthetic mesh and therefore a reinforced repair. Component separation also reduces the subsequent tension on the midline fascial incision closure and the mesh-musculofascia interface. Component separation involves releasing the external oblique aponeurosis and delaminating the external oblique muscle from the internal oblique muscle interface. This results in an offloading of the bilateral superolateral vector pull of the external oblique muscle on the central wound closure. Component separation can be performed as an open procedure that divides all cutaneous perforators or as a perforator-sparing minimally invasive procedure. Minimally invasive component separation (MICS), although technically more demanding than the open version, has been shown to decrease the incidence of skin dehiscence, wound-healing complications, and hernia

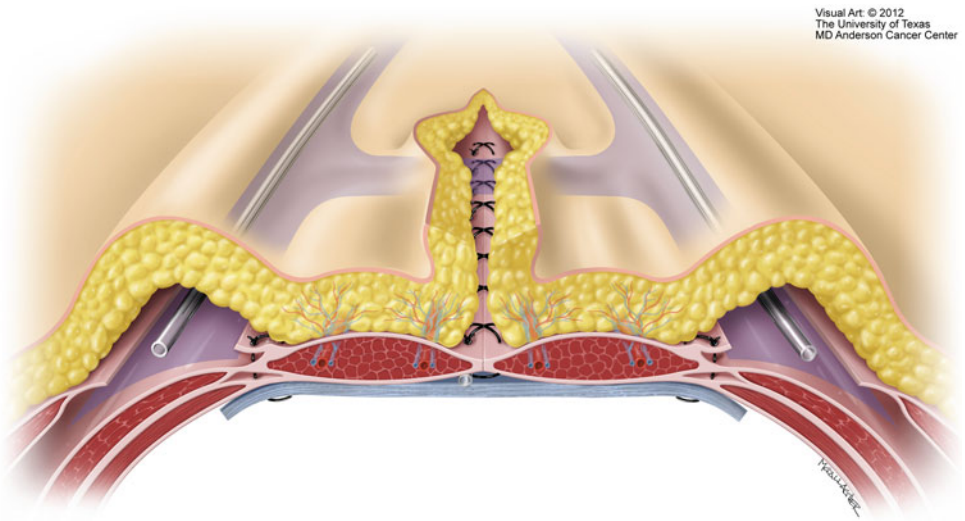


Fig. 10.2 Reinforced mesh repair with primary fascial closure and minimally invasive component separation (Copyright © 2012 The University of Texas MD Anderson Cancer Center)

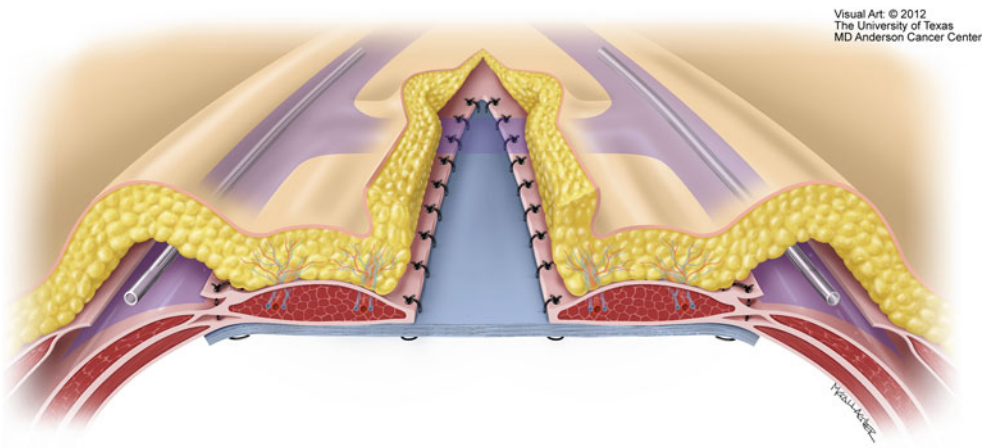


Fig. 10.3 Bridged mesh repair and minimally invasive component separation (Copyright © 2012 The University of Texas MD Anderson Cancer Center)

recurrence/bulge [36]. These improved outcomes are likely attributable to preservation of the vascularity of the overlying skin flaps and reduction of paramedian dead space, both of which MICS was designed to do.

Component separation can be performed in the face of a violation of the ipsilateral rectus sheath through either an ostomy or transection of the rectus abdominis muscle. At our institution, patients with a previously violated rectus myofascial complex who have undergone component separation have surgical outcomes (early complications and late recurrent hernia/bulge rates) equivalent to those of patients without such violation [37]. Although posterior sheath release was originally described as a maneuver included in component separation, it adds minimal additional medialization of the rectus complexes in most cases; the exception is cases with prolonged, severe loss of domain. These patients often have a "tubularized" rectus complex, and a posterior sheath release unfurls the rectus complex and enables considerable medialization of the rectus complex toward the midline. Posterior sheath release is also used as an access incision to the retrorectus space for mesh placement, as in the Rives-Stoppa ventral hernia repair technique [38].

Component Separation Technique

In the open component separation technique, after exploratory laparotomy, lysis of adhesions, and definition of fascial edges, bilateral subcutaneous skin flaps are elevated over the anterior rectus sheath circumferentially, transecting the medial and lateral rectus abdominis perforator vessels. The linea semilunaris is exposed, and the external oblique aponeurosis is incised 1–2 cm lateral to the linea semilunaris, from 5 to 6 cm above the costal margin to near the inguinal ligament. The external oblique and internal oblique muscles are separated by blunt and sharp dissection laterally to the midaxillary line. Release of the external oblique aponeurosis and separation of the internal and external oblique muscles allow medialization of the rectus sheath fascia to the midline fascia. The midline fascia is then closed with interrupted polypropylene sutures. Closed-suction drainage catheters are placed in the subcutaneous space, and absorbable quilting sutures are placed between Scarpa's fascia and the anterior abdominal wall fascia to obliterate dead space.

In MICS with inlay bioprosthetic mesh (MICSIB) technique [36], bilateral subcutaneous access tunnels 3-cm wide are created over the anterior rectus sheath from the midline to the linea semilunaris at the level of the costal margin (Fig. 10.4). Through these access tunnels, the external oblique aponeurosis is vertically incised 1.5 cm lateral to the linea semilunaris. The tip of a metal Yankauer suction handle (Cardinal Health, Dublin, OH), without suction, is inserted through the opening into the avascular plane between the internal and external oblique aponeuroses, separating them at their junction with the rectus sheath. The suction tip is advanced inferiorly to the pubis and superiorly to above the costal margin. A narrow (2.5-cm wide) subcutaneous tunnel is created with electrocautery and blunt dissection superficial to the external oblique aponeurosis, over the planned release location, using a narrow retractor and a headlight (Fig. 10.5). The external oblique aponeurosis is then released approximately 1.5 cm lateral to the lateral edge of the rectus sheath from 12 cm above the costal margin superiorly to near the pubis inferiorly. Next, lateral dissection between the internal and external oblique muscle is performed to the midaxillary line. Subcutaneous skin flaps are elevated over the anterior rectus sheath circumferentially to the medial row of rectus abdominis perforator vessels. The preperitoneal fat is dissected from the posterior sheath circumferentially to allow the bioprosthetic mesh to be inlaid directly against the posterior sheath or rectus muscle (below the arcuate line). Mesh is inset using a preperitoneal inlay technique; interrupted, number 1 polypropylene sutures are

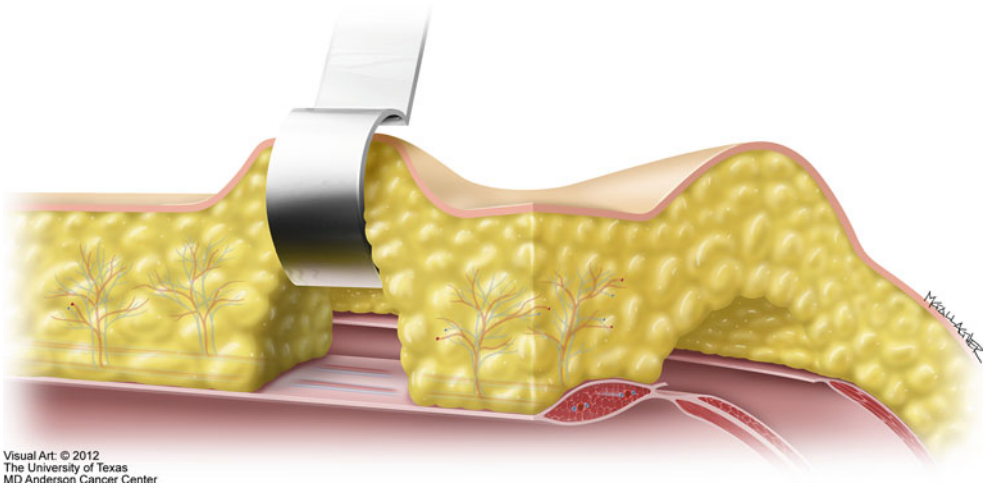


Fig. 10.4 Minimally invasive component separation (Copyright © 2012 The University of Texas MD Anderson Cancer Center)

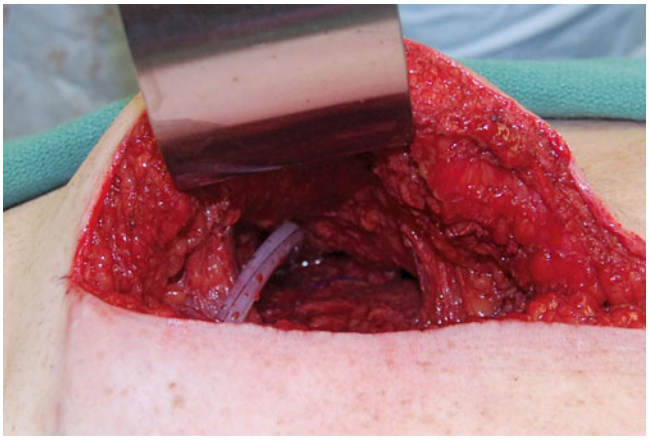


Fig. 10.5 Minimally invasive component separation subcutaneous access tunnel (Copyright © 2012 The University of Texas MD Anderson Cancer Center)

placed 3–5 cm peripheral to the true fascial edge through the bioprosthetic mesh and back through the musculofascial to create U stitches. All sutures are preplaced and tagged with hemostats to allow assessment, and potential adjustment, of the inset tension. Then, the musculofascial edges are advanced and coapted over the mesh with sutures placed through the musculofascia and bioprosthesis. Interrupted resorbable 3–0 sutures are placed to affix the posterior sheath to the mesh, thus reducing dead space and potential fluid collection. The fascial edges are closed with interrupted resorbable number 1 monofilament sutures. If complete musculofascial midline closure is not possible, the musculofascial edges are sutured to the surface of the mesh using interrupted number 1 polypropylene sutures to create a "bridged" repair, with the mesh spanning the defect between the musculofascial edges.

With both open component separation and MICSIB, the redundant medial aspects of the skin flaps are carefully excised in a vertical panniculectomy. Closed-suction drainage catheters are placed in each component separation donor site area, in the space between the rectus complex closure and bioprosthetic mesh, and in the subcutaneous space. The remaining undermined skin flaps are quilted to the musculofascia with resorbable 3–0 quilting sutures to reduce dead space and potential shear between the subcutaneous tissue and musculofascia. The midline skin incision is then closed in layers.

At times, unfavorable wound-healing scenarios will be encountered, and wound infection, dehiscence, or breakdown of overlying skin flaps will lead to exposure of the bioprosthetic mesh. Bioprosthetic meshes' tolerance of bacterial contamination and exposure allows an area of wound separation to be reclosed over drains after clearing any infection, assuming there is adequate skin laxity for closure. Small defects can be left open to heal by secondary intention with the use of standard saline-soaked dressing changes or negative-pressure wound therapy devices. The goal of negative-pressure wound therapy is to prevent desiccation and dehydration of the bioprosthetic material. Negative-pressure wound therapy can be used to develop a revascularized mesh granulation bed suitable for skin graft coverage or serve as a temporizing measure to facilitate a delayed primary closure or flap coverage, as the clinical circumstances dictate. The use of nonadherent barrier dressing materials between the negative-pressure wound therapy foam and the bioprosthetic mesh prevents trauma to the bioprosthetic mesh and helps retain the foam. Various materials can be used for this purpose, such as petroleum-impregnated wide mesh gauze or perforated silicone dressings. Alternatively, microporous foam, such as polyvinyl alcohol foam, can be placed directly over the bioprosthetic mesh. A skin graft can be applied onto granulated bioprosthetic mesh. If the defect is large with bioprosthetic mesh exposed at the base, the best choice is generally autologous skin flap tissue, whether a local advancement flap from the abdomen, a rotation advancement flap, a pedicled regional flap, or a free flap.

Management of the Skin: Deficiency and Redundancy

The success of any abdominal wall reconstruction depends on a stable wound-healing environment. Durable soft tissue coverage is required to avoid mesh exposure and reduce the risk of seroma formation, periprosthetic infection, and subsequent explantation. The goals of soft tissue coverage are to achieve a tension-free closure and obliterate any potential dead space. Redundant skin and subcutaneous flaps are often encountered after a bilateral component separation is performed because of the extensive medialization of the musculofascia. The paramedian skin edges in an open component separation can become marginally devascularized, a complication MICSIB is designed to eliminate. Compromised attenuated paramedian skin is resected as a vertical panniculectomy to minimize skin redundancy and subcutaneous dead space [39]. In patients who require resection of both horizontal and vertical redundancy, a Mercedes pattern skin excision can be performed to avoid skin necrosis at the confluence of the vertical and horizontal panniculectomy incisions [40].

In patients with large cutaneous defects and insufficient skin available for closure, wound coverage may require a local advancement flap, locoregional flap, or free flap. Coverage can generally be accomplished in the torso by local fasciocutaneous flap advancement. The overlapping angiosomes of the abdominal wall's skin allow for wide

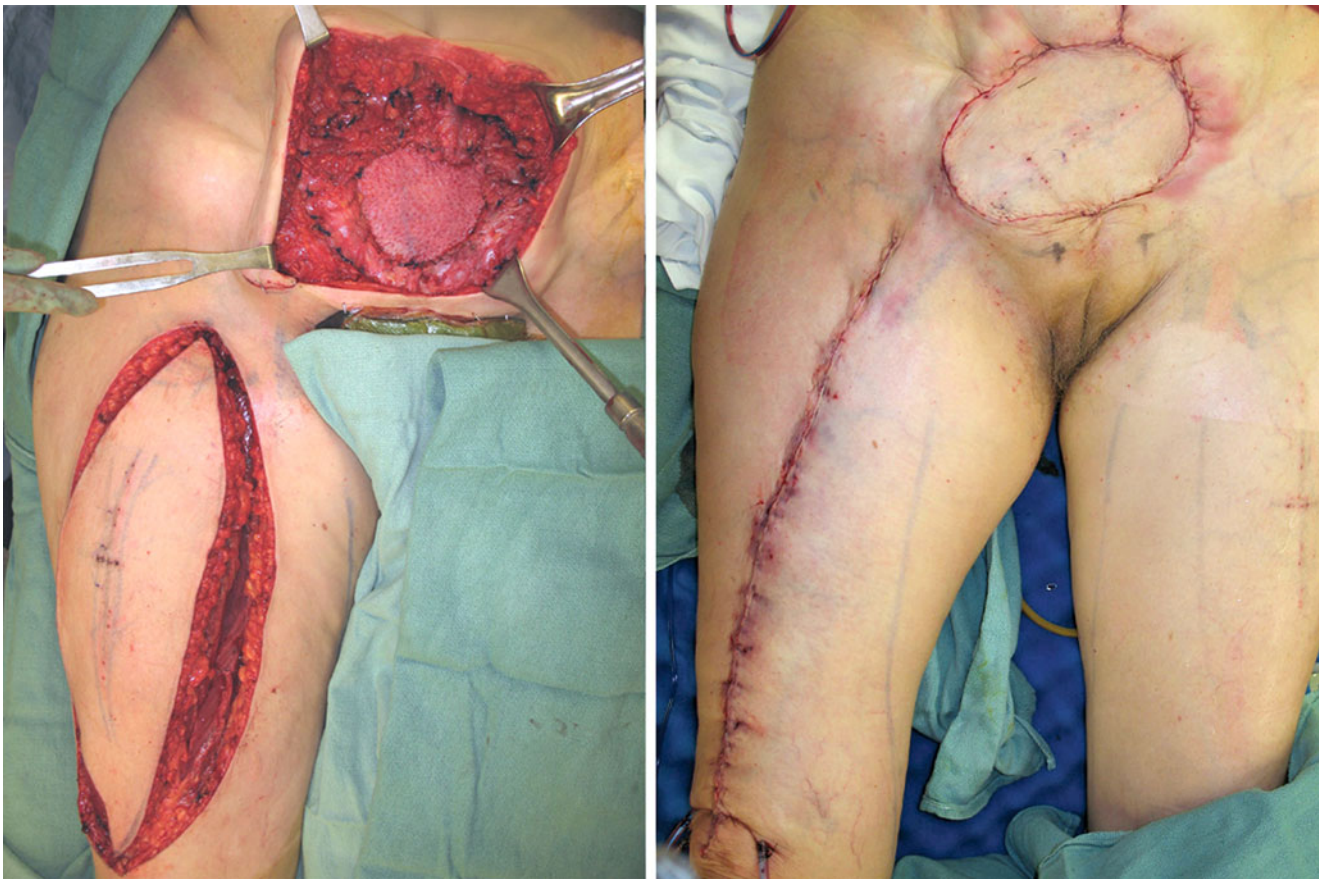


Fig. 10.6 Cutaneous deficit coverage with a pedicled anterolateral thigh flap (Copyright © 2012 The University of Texas MD Anderson Cancer Center)

undermining and skin advancement. In cases of prior radiation, prior surgery, or excessive skin resection, a pedicled regional flap or free flap might be required to provide adequate soft tissue coverage. Options for pedicled flaps in the upper abdomen include vertical rectus abdominis flaps and latissimus dorsi musculocutaneous flaps. Pedicled thigh-based flaps such as anterolateral thigh flaps, vastus lateralis flaps, and tensor fascia lata flaps are able to reach the lower abdomen and flank; for massive defects, a pedicled or free subtotal thigh flap [41] can be used (Fig. 10.6). If a pedicled flap is not available or feasible, a thoracoepigastric bipedicled fasciocutaneous flap may provide adequate local tissue and avoid a free tissue transfer. When these options are not feasible, a free flap is required for soft tissue coverage. The thigh can serve as a source of fasciocutaneous flaps and myocutaneous flaps that provide large skin paddles and significant muscle volume. Recipient vessels in the lateral abdominal wall include the deep inferior epigastric vessels, superior epigastric vessels, internal mammary vessels, intercostal artery perforators, and thoracolumbar perforators. When no local recipient vessels are available, vein grafts to the internal mammary or femoral vessels might be required.

Staged Abdominal Wall Reconstruction

Abdominal wall reconstruction at the time of unplanned bowel resection, excessive bowel edema, or extensive intraperitoneal inflammation presents formidable challenges in replacing the musculofascia and overlying skin. Intra-abdominal complications such as infection, obstruction, and fistula can be life threatening. Local wound conditions, including bacterial contamination, previous incisions, and abdominal wall radiation injury, can increase the likelihood of compromised wound healing, surgical site infection, and failure of the reconstruction. When local skin flaps and regional flaps are unavailable for soft tissue coverage in such cases, the remaining flap options might be limited. A useful strategy in these cases is to perform the reconstruction in stages. Bioprosthetic mesh is placed as an initial musculofascial replacement, and soft tissue wound closure is performed once bowel function has been reestablished. Early fascial closure preserves the musculofascial domain, reduces the risk of enterocutaneous fistula, and may reduce the complexity of subsequent wound management. After a period of optimal wound care, cutaneous coverage can be achieved by

delayed primary closure, healing by secondary intention, skin grafting, or flap reconstruction. Negative-pressure wound therapy provides temporizing wound care that allows for early flap coverage with preservation of the bioprosthetic mesh's integrity.

Early reoperation after complex abdominal wall reconstruction can be necessary for myriad reasons, including hematoma, bowel obstruction, and intra-abdominal sepsis. Reentry into the abdominal cavity under these circumstances can require conversion of a dual-layer "musculofascia-over-mesh" closure to a bridging mesh repair or make the further use of mesh impossible owing to intestinal edema, infection, or loss of domain. If mesh is temporarily removed during reoperation and reinset with less tension as a bridged repair, edema and friability of the abdominal wall fascia can lead to a weakened interface. One strategy to preserve the abdominal wall repair during reoperation for intra-abdominal complications is to perform a midline laparotomy incision through the midsubstance of the bioprosthetic mesh. This allows the lateral mesh-musculofascia interface to be preserved, and the mesh can be coapted to itself in the midline for abdominal closure.

Postoperative Care

After abdominal wall reconstruction, postoperative care includes gradual diet advancement based on intestinal function, epidural pain management transitioned to oral analgesics, and early ambulation (postoperative day 1). Patients are generally discharged from the hospital on postoperative day 4–7. Drains are removed when the output is 25 mL or less over 24 h. Patients are directed to avoid heavy physical activity and sports for 6 weeks postoperatively. Patients are typically followed up with a physical examination daily while in the hospital, then weekly for 1 month after discharge, and then every 3–6 months.

Conclusions

Surgical planning in complex abdominal wall reconstruction requires the combined efforts of plastic surgeons and general surgeons. To achieve the goals of reestablishing the integrity of the musculofascial unit and providing cutaneous coverage of the abdominal wall defect, surgeons must take into consideration local wound conditions, optimize the utility of remaining tissues, reinforce the abdominal wall with mesh, and provide durable skin replacement. To minimize hernia recurrences and maximize preservation of function, this type of complex abdominal wall reconstruction should be attempted only by teams of highly experienced surgeons.

Disclosure: Dr. Butler serves as a consultant for LifeCell Corporation. No other author has any relevant disclosures.

References

1. Diaz Jr JJ et al. Multi-institutional experience using human acellular dermal matrix for ventral hernia repair in a compromised surgical field. Arch Surg. 2009;144(3):209–15.
2. Houck JP et al. Repair of incisional hernia. Surg Gynecol Obstet. 1989;169(5):397–9.
3. Luijendijk RW et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med. 2000;343(6):392–8.
4. Finan KR et al. Predictors of wound infection in ventral hernia repair. Am J Surg. 2005;190(5):676–81.
5. Dunne JR et al. Abdominal wall hernias: risk factors for infection and resource utilization. J Surg Res. 2003;111(1):78–84.
6. Burger JW et al. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg. 2004;240(4):578–83; discussion 583–5.
7. Flum DR, Horvath K, Koepsell T. Have outcomes of incisional hernia repair improved with time? A population-based analysis. Ann Surg. 2003;237(1):129–35.
8. Espinosa-de-los-Monteros A et al. Utilization of human cadaveric acellular dermis for abdominal hernia reconstruction. Ann Plast Surg. 2007;58(3):264–7.
9. den Hartog D, et al. Open surgical procedures for incisional hernias. Cochrane Database Syst Rev 2008. Jul 16;(3):CD006438.
10. Korenkov M et al. Classification and surgical treatment of incisional hernia. Results of an experts' meeting. Langenbecks Arch Surg. 2001;386(1):65–73.
11. Harrell AG et al. Prospective evaluation of adhesion formation and shrinkage of intra-abdominal prosthetics in a rabbit model. Am Surg. 2006;72(9):808–13; discussion 813–4.
12. Novitsky YW et al. Comparative evaluation of adhesion formation, strength of ingrowth, and textile properties of prosthetic meshes after long-term intra-abdominal implantation in a rabbit. J Surg Res. 2007;140(1):6–11.
13. Bellon JM et al. Postimplant behavior of lightweight polypropylene meshes in an experimental model of abdominal hernia. J Invest Surg. 2008;21(5):280–7.
14. Emans PJ et al. Polypropylene meshes to prevent abdominal herniation. Can stable coatings prevent adhesions in the long term? Ann Biomed Eng. 2009;37(2):410–8.
15. van't Riet M et al. Prevention of adhesion to prosthetic mesh: comparison of different barriers using an incisional hernia model. Ann Surg. 2003;237(1):123–8.
16. Schug-Pass C et al. The use of composite meshes in laparoscopic repair of abdominal wall hernias: are there differences in biocompatibility? Experimental results obtained in a laparoscopic porcine model. Surg Endosc. 2009;23(3):487–95.
17. Pierce RA et al. 120-day comparative analysis of adhesion grade and quantity, mesh contraction, and tissue response to a novel omega-3 fatty acid bioabsorbable barrier macroporous mesh after intraperitoneal placement. Surg Innov. 2009;16(1):46–54.
18. Harrell AG et al. In vitro infectability of prosthetic mesh by methicillin-resistant *Staphylococcus aureus*. Hernia. 2006;10(2):120–4.
19. Schreinemacher MH et al. Degradation of mesh coatings and intraperitoneal adhesion formation in an experimental model. Br J Surg. 2009;96(3):305–13.

20. de Vries Reilingh TS et al. Interposition of polyglactin mesh does not prevent adhesion formation between viscera and polypropylene mesh. J Surg Res. 2007;140(1):27–30.
21. Burger JW et al. Evaluation of new prosthetic meshes for ventral hernia repair. Surg Endosc. 2006;20(8):1320–5.
22. Holton 3rd LH et al. Human acellular dermal matrix for repair of abdominal wall defects: review of clinical experience and experimental data. J Long Term Eff Med Implants. 2005;15(5):547–58.
23. Sandor M et al. Host response to implanted porcine-derived biologic materials in a primate model of abdominal wall repair. Tissue Eng Part A. 2008;14(12):2021–31.
24. Breuing K et al. Incisional ventral hernias: review of the literature and recommendations regarding the grading and technique of repair. Surgery. 2010;148(3):544–58.
25. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86(3):519–26.
26. Kolker AR et al. Multilayer reconstruction of abdominal wall defects with acellular dermal allograft (AlloDerm) and component separation. Ann Plast Surg. 2005;55(1):36–41; discussion 41–2.
27. Ghazi B et al. Current options in the management of complex abdominal wall defects. Ann Plast Surg. 2011;66(5):488–92.
28. Lindstrom D et al. Effects of a perioperative smoking cessation intervention on postoperative complications: a randomized trial. Ann Surg. 2008;248(5):739–45.
29. Mullen JT et al. Impact of body mass index on perioperative outcomes in patients undergoing major intra-abdominal cancer surgery. Ann Surg Oncol. 2008;15(8):2164–72.
30. Mangram AJ et al. Guideline for prevention of surgical site infection, 1999. Centers for Disease Control and Prevention (CDC) Hospital Infection Control Practices Advisory Committee. Am J Infect Control. 1999;27(2):97–132. quiz 133–4; discussion 96.
31. Burns NK et al. Non-cross-linked porcine acellular dermal matrices for abdominal wall reconstruction. Plast Reconstr Surg. 2010;125(1):167–76.
32. Holton 3rd LH et al. Comparison of acellular dermal matrix and synthetic mesh for lateral chest wall reconstruction in a rabbit model. Plast Reconstr Surg. 2007;119(4):1238–46.
33. Butler CE, Langstein HN, Kronowitz SJ. Pelvic, abdominal, and chest wall reconstruction with AlloDerm in patients at increased risk for mesh-related complications. Plast Reconstr Surg. 2005;116(5):1263–75; discussion 1276–7.
34. Jin J et al. Use of acellular dermal matrix for complicated ventral hernia repair: does technique affect outcomes? J Am Coll Surg. 2007;205(5):654–60.
35. Itani K. Prospective multicenter clinical study of single-stage repair of infected or contaminated abdominal incisional hernias using Strattice reconstructive tissue matrix. Washington: American College of Surgeons Clinical Congress; 2010.
36. Butler CE, Campbell KT. Minimally invasive component separation with inlay bioprosthetic mesh (MICSIB) for complex abdominal wall reconstruction. Plast Reconstr Surg. 2011;128(3):698–709.
37. Garvey PB et al. Violation of the rectus complex is not a contraindication to component separation for abdominal wall reconstruction. J Am Coll Surg. 2012;214(2):131–9.
38. Iqbal CW et al. Long-term outcome of 254 complex incisional hernia repairs using the modified Rives-Stoppa technique. World J Surg. 2007;31(12):2398–404.
39. Ko JH et al. Abdominal wall reconstruction: lessons learned from 200 "components separation" procedures. Arch Surg. 2009;144(11):1047–55.
40. Butler CE, Reis SM. Mercedes panniculectomy with simultaneous component separation ventral hernia repair. Plast Reconstr Surg. 2010;125(3):94e–8e.
41. Lin SJ, Butler CE. Subtotal thigh flap and bioprosthetic mesh reconstruction for large, composite abdominal wall defects. Plast Reconstr Surg. 2010;125(4):1146–56.

Staged Reconstructions of Abdominal Wall Defects

11

Ari Leppäniemi

Introduction

Staged abdominal wall reconstruction or planned ventral hernia is a management strategy of an open abdomen in which the fascial layer has been left unclosed and the viscera are covered with original or grafted skin. Most commonly, it is a result of prophylactic or therapeutic open abdomen that cannot or should not undergo primary fascial closure. Severe acute pancreatitis, damage control surgery for massive abdominal trauma, and surgery for ruptured abdominal aortic aneurysm are associated with primary abdominal compartment syndrome leading to an open abdomen. Loss of abdominal wall substance because of tumor excision or necrotizing infection and the removal of an infected mesh can also result in a situation requiring a planned hernia strategy. Under these circumstances, the hernia is a favorable outcome with the aim of repairing the hernia at a later stage when it is safe, possible, and tolerated by the patient.

Three Stages of Reconstruction

Stage 1: Temporary Abdominal Closure

Over the years, the methods for temporary abdominal closure (TAC) (stage 1 of reconstruction) have evolved through several stages [1]. The first-generation TAC consisted mainly of abdominal coverage, either by skin-only closure (with running suture or towel clips) or a synthetic cover, such as a plastic silo (Bolsa de Borraez, Bogota bag), mesh, or a Velcro burr. The second-generation TAC methods introduced the concept of fluid control (e.g., the vacuum pack). Third-generation TAC methods are mainly commercially manufactured negative-pressure therapy sets such as the VAC™ Abdominal Dressing (Kinetic Concepts, San Antonio, TX) or ABThera™ (Kinetic Concepts). Recently, the combined use of a temporary mesh and the negative-pressure dressing has resulted in delayed primary fascial closure rates of about 90% [2] (Fig. 11.1).

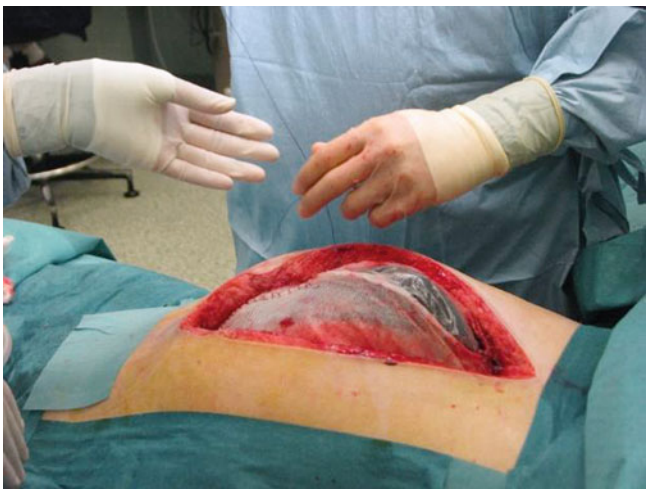


Fig. 11.1 Vacuum and mesh-mediated fascial traction closure method of open abdomen

Stage 2: The Maturation Period

If the TAC techniques do not achieve fascial approximation at the midline within a reasonable time frame (stage 2, the maturation period), a more sustainable cover of the abdominal viscera is needed. If there is enough viable skin to be closed without too much tension, this skin-only technique is an acceptable and preferred method as long as there is no risk for further loss of abdominal skin. If the patient's original skin does not allow skin-only closure, a split-thickness skin

A. Leppäniemi, MD, PhD
Department of Abdominal Surgery,
Meilahti Hospital, University of Helsinki,
Haartmaninkatu 4, 340, Helsinki FIN-00029 HUS, Finland

Department of Emergency Surgery,
Meilahti Hospital, University of Helsinki,
Haartmaninkatu 4, 340, Helsinki FIN-00029 HUS, Finland
e-mail: ari.leppaniemi@hus.fi

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_11, © Springer Science+Business Media New York 2013

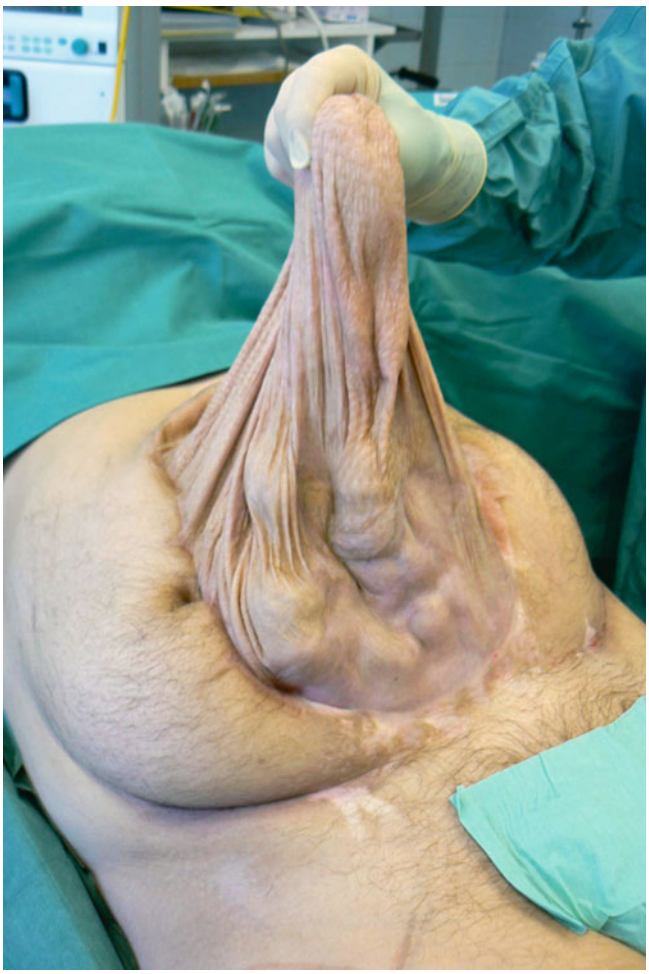


Fig. 11.2 Matured skin graft closure

graft provides a readily available, cheap, foreign-body-free, and infection-resistant coverage that closes the "catabolic drain" of the open abdomen and protects the viscera from erosion (Fig. 11.2). A skin graft can be applied over exposed bowel at a relatively early stage without having to wait for mature granulation tissue to appear.

Stage 3: Definitive Abdominal Wall Reconstruction

Based on the type of skin coverage used for staged repair of an open abdomen, the abdominal wall defect can be reconstructed with several different methods in stage 3. To achieve the best functional result, the rectus muscles should be brought together in the midline using component separation or other local tissue transfer technique if possible. In patients with intact original skin, the hernia can be repaired with a mesh. However, in patients with large skin-grafted defects in the midline or extensive hernias reaching the epigastrium or in the presence of contamination or infected mesh, the tissue transfer or mesh-based techniques might not be possible or appropriate, and a more complex reconstruction technique is required.

Vascularized flaps provide healthy autologous tissue coverage and usually do not require any implantation of foreign material at the closure site. Small and midsize defects can be repaired with pedicled flaps within the arch of the rotation of the flap. In extensive upper midline abdominal wall and thoracoabdominal defects, a free flap that offers a completely autologous, single-stage reconstructive solution is in most cases the best option available.

Tensor Fascia Latae Flap for Abdominal Wall Reconstruction

The tensor fascia latae (TFL) myocutaneous free flap was first described by Hill and coworkers in 1978 [3]. Besides reconstructing large abdominal wall defects, it can also be used for reconstruction of complex head and neck, composite extremity, and perineal defects [4–16]. To date, the microvascular TFL flaps, sometimes in combination with the anterolateral thigh flap, have been used in more than 90 patients with abdominal wall defects [3–17].

The deep inferior epigastric vessels are the most commonly used recipient vessels for the TFL flap, but utilizing intraperitoneal vessels, such as the gastroepiploic vessels, allows the use of flaps with shorter pedicles and tight, continuous, circumferential fascial closure between the flap and native abdominal wall [11]. In contrast to the anterolateral thigh flap, however, the anatomy of the TFL pedicle is constant, and it offers large-caliber vessels matching the vessel size of the great saphenous vein loop. Furthermore, the size of the flap can be large (up to 20×35 cm). However, in extremely wide flaps, the relative thinness of the anteromedial portion of the fascia, especially in women, sometimes requires mesh enforcement [17].

Functionally, the TFL flap is passive, resembling a mesh. A functional dynamic reconstruction of full-thickness abdominal wall defect with an innervated free latissimus dorsi musculocutaneous flap has been described by Ninkovic and coworkers [18].

The technique used at our institution is now described [17]. A musculofasciocutaneous flap with a skin component measuring 30–35×15–20 cm and underlying fascia as well as the TFL muscle is harvested from the thigh, and its pedicle is dissected free toward the deep femoral artery and vein. In patients with large defects, the rectus femoris muscle can be included in the flap to ensure adequate perfusion of the distal tip. The ipsilateral great saphenous vein is divided distally above the knee, and its distal end is reflected proximally and anastomosed end to side to the common femoral artery, creating an arteriovenous loop (Fig. 11.3). The loop is tunneled subcutaneously to the edge of the defect and divided at its apex. Arterial and venous anastomoses with the flap

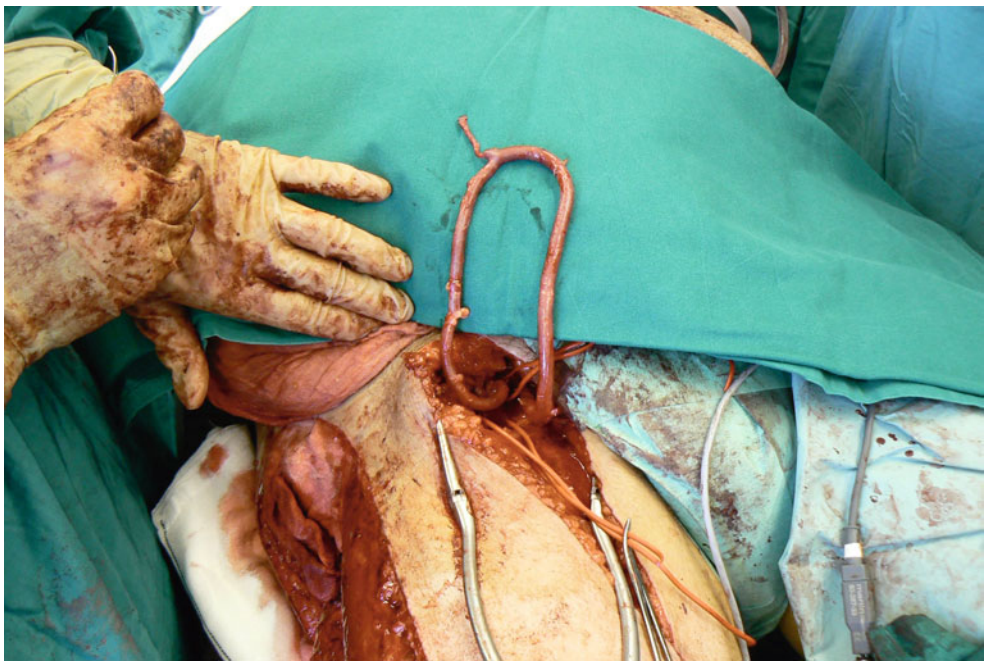


Fig. 11.3 Free tensor fascia latae flap with arteriovenous (AV) loop (Reprinted with kind permission of Springer Science + Business Media from Leppäniemi and Tukiainen [19])

vessels are performed with continuous 7–0 or 8–0 vascular sutures. The flap fascial edges are sutured to the fascial edges of the original defect, carefully avoiding any obstruction or kinking of the flap vessels. Drains are placed subcutaneously, and the subcutaneous space and skin are closed with interrupted sutures or staples. The donor site is closed directly as far as possible, and the remaining defect is covered with a split-thickness skin graft. Postoperatively, the viability of the flap is monitored clinically for flap color, temperature, and capillary refill. In addition, the intra-abdominal pressure is measured at regular intervals.

Since 1990, 20 patients with large abdominal wall defects have been operated on with TFL flap in our institution [17]. The perioperative mortality was zero, and there were no intra-abdominal or deep surgical site infections. There was one flap failure, and two patients had minor distal tip necrosis requiring only revision and primary skin closure. During a follow-up period of 0.5–13 years, there was only one hernia recurrence 3 months after the TFL repair. Because of a large defect or if the fascial component of the TFL flap was found to be thin, an additional component separation procedure was used in one patient, mesh enforcement in nine patients, and a combination of both techniques in one patient.

Selection of the Appropriate Reconstruction Method

Abdominal wall defects may be categorized as type I and II defects depending on the type of skin coverage over the defect. In type I defects, there is intact or stable skin coverage, whereas type II defects have absent or unstable skin coverage [20]. Even relatively large type I defects can usually be repaired with component separation or mesh repair alone.

The most important aspect of reconstructing a functional abdominal wall is the re-creation of the linea alba and achieving midline closure, allowing the abdominal wall to be encompassed by functional muscular components in a manner similar to normal anatomy [21]. In contrast to inert material, the abdominal musculature provides dynamic support of innervated tissue to redistribute the stress applied from intra-abdominal forces. In that respect, the component separation technique is preferred over a mesh repair.

Fascial repair alone is inappropriate in abdominal wall defects with absent or unstable skin coverage (type II) because the repair needs to be covered with healthy skin, often requiring reconstruction techniques that are more complex. The criteria for special reconstruction techniques have been listed as a large-size (40-cm^2) defect, absence of stable skin coverage, recurrence of the defect after prior closure attempts, infected or exposed mesh, systemic compromise (intercurrent malignancy), local tissue compromise (irradiation, corticosteroid dependence), or concomitant visceral complications (enterocutaneous fistula) [20].

However, complex reconstruction techniques are rarely used and are required mainly in extensive or recurrent defects. In a series of 954 patients undergoing autologous tissue repair techniques of large abdominal wall defects, 94% of the patients underwent either local tissue repair (component separation, rectus sheath) or repair with autologous grafts (free fascial latae, autodermal graft). Pedicled or free vascularized flaps were used in only 59 patients, with 35 of these TFL flaps (pedicled in 15 and microvascular in 20 patients) [22].

Table 11.1 Management options in abdominal wall defects

	Primary procedure	Additional (+) or optional procedures
Small or midsize hernia, intact skin		
No contamination	CS	M
Contamination	CS	Mb
Small or midsize hernia, grafted skin		
No contamination	CS	+M or flap
Contamination	CS	+Mb or flap
Large hernia, intact skin		
No contamination	CS	+Flap or M
Contamination	CS	+Flap or Mb
Large hernia, grafted skin		
No contamination	Flap	+CS + M
Contamination	Flap	+CS + Mb

CS component separation, *M* mesh repair, *Mb* biological mesh
Reproduced with kind permission of Springer Science + Business Media from Leppäniemi and Tukiainen [19]

Summary

The choice of the most appropriate late abdominal wall reconstruction method after planned hernia strategy is always an individualized process requiring a multispecialty approach and close collaboration with the plastic and abdominal surgeons. The guidelines used at our institution in selecting the appropriate reconstruction method are presented in Table 11.1 [19].

References

1. De Waele JJ, Leppäniemi A. Temporary abdominal closure techniques. Am Surg. 2011;77:S46–50.
2. Acosta S, Bjarnason T, Petersson U, et al. Multicentre prospective study of fascial closure rate after open abdomen with vacuum and mesh-mediated fascial traction. Br J Surg. 2011;98:735–43.
3. Hill HL, Nahai F, Vasconez LO. The tensor fasciae latae myocutaneous free flap. Plast Reconstr Surg. 1978;61:517–22.
4. O'Hare PM, Leonard AG, Brennan MD. Experience with the tensor fasciae latae free flap. Br J Plast Surg. 1983;36:98–104.
5. Caffee HH. Reconstruction of the abdominal wall by variations of the tensor fasciae latae flap. Plast Reconstr Surg. 1983;71:348–51.
6. Penington AJ, Theile DR, MacLeod AM, et al. Free tensor fasciae latae flap reconstruction of defects of the chest and abdominal wall: selection of recipient vessels. Scand J Plast Reconstr Surg Hand Surg. 1996;30:299–305.
7. Williams JK, Carlson GW, Howell RL, et al. The tensor fascia lata free flap in abdominal-wall reconstruction. J Reconstr Microsurg. 1997;13:83–90.
8. Sasaki K, Nozaki M, Nakazawa H, et al. Reconstruction of a large abdominal wall defect using combined free tensor fasciae latae musculocutaneous flap and anterolateral thigh flap. Plast Reconstr Surg. 1998;102:2244–52.
9. Lyle WG, Gibbs M, Howdieshell TR. The tensor fascia lata free flap in staged abdominal wall reconstruction after traumatic evisceration. J Trauma. 1999;46:519–22.
10. Heitmann C, Pelzer M, Menke H, et al. The free musculocutaneous tensor fascia lata flap as a backup procedure in tumor surgery. Ann Plast Surg. 2000;45:399–404.
11. Chevray PM, Singh NK. Abdominal wall reconstruction with the free tensor fascia lata musculofasciocutaneous flap using intraperitoneal gastroepiploic recipient vessels. Ann Plast Surg. 2003; 51:97–102.
12. Kuo YR, Kuo MH, Lutz BS, et al. One-stage reconstruction of large midline abdominal wall defects using a composite free anterolateral thigh flap with vascularized fascia lata. Ann Surg. 2004; 239:352–8.
13. Bulstrode NW, Kotronakis I, Baldwin MA. Free tensor fascia latae musculofasciocutaneous flap in reconstructive surgery: a series of 85 cases. J Plast Reconstr Aesthet Surg. 2006;59:130–6.
14. Sarabahi S, Bajaj SP, Bhatnagar A, et al. Reconstruction of abdominal wall by whole thigh flap. J Plast Reconstr Aesthet Surg. 2006;59:1429–32.
15. Wong CH, Lin CH, Fu B, et al. Reconstruction of complex abdominal wall defects with free flaps: indications and clinical outcome. Plast Reconstr Surg. 2009;124:500–9.
16. Kuo YR, Yeh MC, Shih HS, et al. Versatility of the anterolateral thigh flap with vascularized fascia lata for reconstruction of complex soft-tissue defects: clinical experience and functional assessment of the donor site. Plast Reconstr Surg. 2010;125:757–8.
17. Tukiainen E, Leppäniemi A. Reconstruction of extensive abdominal wall defects with microvascular tensor fasciae latae flap. Br J Surg. 2011;98:880–4.
18. Ninkovic M, Kronberger P, Harpf C, et al. Free innervated latissimus dorsi muscle flap for reconstruction of full-thickness abdominal wall defects. Plast Reconstr Surg. 1998;101:971–8.
19. Leppäniemi A, Tukiainen E. Planned hernia repair and late abdominal wall reconstruction. World J Surg. 2012;36(3):511–5.
20. Mathes SJ, Steinwald PM, Foster RD, et al. Complex abdominal wall reconstruction: a comparison of flap and mesh closure. Ann Surg. 2000;232:586–94.
21. Harth KC, Rosen MJ. Endoscopic versus open component separation in complex abdominal wall reconstruction. Am J Surg. 2010; 199:342–7.
22. De Vries Reilingh TS, Bodegom ME, van Goor H, et al. Autologous tissue repair of large abdominal wall defects. Br J Surg. 2007;94: 791–803.

Selection of Prosthetic Materials in the Repair of Complex Abdominal Wall Defects

12

Marcos Campos W. Reis, Bruno Monteiro T. Pereira, Bartolomeu Nascimento, and Gustavo Pereira Fraga

Introduction

The development of an incisional ventral hernia is a common complication following open abdominal surgery and represents a major management challenge for the surgeon. Its incidence varies between 2 and 20 %, and it is estimated that approximately 250,000 hernias repairs are performed each year at a cost of $2.5–$3 billion in the United States [1–3]. Complex abdominal wall defects (CAWD) can be defined by the presence of any of the following, either in isolation or in combination: hernia that is recurrent with multiple failed repairs; multiple sites of abdominal wall defects; loss of abdominal domain; damage control skin graft closure, infection or other local tissue compromise; and resection of abdominal wall musculature with inadequate soft-tissue coverage [4, 5].

The incidence of CAWD has increased with bariatric surgery procedures due to the epidemic of obesity in the United States, "damage control" trauma laparotomies, the increase in visceral transplantation rates, and the increase in rates of failed primary herniorrhaphies [4, 6]. CAWD are associated with potentially serious complications such as intestinal obstruction, gangrene, peritonitis, intestinal perforations, and death. Therefore, the need for their correction is well-established. In general, the surgical management of CAWD can be performed with or without the use of a prosthetic material.

The direct surgical repair is associated with high risk of complications including bleeding, wound infection, skin necrosis, abdominal compartment syndrome, bowel ischemia, prolonged intubation, and death. With this approach, the incidence of recurrent incisional hernia is as high as 58 % [1, 4, 7, 8]. Contrarily, some studies confirmed significantly lower recurrence rates and better outcomes with repairs utilizing synthetic meshes, as compared to direct surgical repairs for the correction of ventral hernias [1, 9–11]. The tension-free procedures achieved by the utilization of prosthetic materials have rapidly gained popularity, and an impressive variety of synthetic materials are now commercially available for the management of CAWD. However, controversy exists over what the best approach would be and over which type of material should be employed. The selection of a prosthetic material should take into consideration not only the synthetic material properties and its biologic response, but also factors related to the technique to be performed and the particular patient´s characteristics.

Considerations When Selecting Prosthetic Materials for the Management of CAWD

Modern hernia surgery is no longer imaginable without the application of prosthetic meshes. The recurrence rate using prosthetic repair is approximately half of the recurrence rate after suture repair [1, 9–11]. The use of prosthetic materials allows the repair of defects of any size without tension; and the mesh induces an inflammatory response, which promotes the synthesis of collagen.

M.C.W. Reis, MD, TCBC
Department of General Surgery, Baleia Hospital,
Faculdade de Sociologia e Ecologia Humana (FASEH),
1341 Odilon Braga St, Belo Horizonte 30310390, Brazil
e-mail: drmarcosreis@gmail.com

B.M.T. Pereira, MD, MSC
Division of Trauma Surgery,
Department of Surgery, University of Campinas,
Campinas, SP, Brazil
e-mail: drbrunomonteiro@hotmail.com

B. Nascimento, MD, MSc, CTBS
Department of Surgery,
University of Toronto/Sunnybrook Health Sciences Center,
2075 Bayview Ave, Toronto, ON M4N 3M5, Canada
e-mail: barto.nascimento@sunnybrook.ca

G.P. Fraga, MD, PhD (✉)
Division of Trauma Surgery,
Department of Surgery, University of Campinas,
Campinas, SP, Brazil
e-mail: fragagp2008@gmail.com

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_12, © Springer Science+Business Media New York 2013

There is debate on which type of material should be used and how the material should be employed in the repair. Open surgery for prosthetic repair is a safe and common technique, but laparoscopic mesh repair is a new procedure with several documented advantages, including smaller incisions, lower risk for complications, shorter hospital stay, and patient preference [12, 13]. The decision between open or laparoscopic repair requires a detailed assessment of the individual patient's risks and benefits.

Prosthetic repair is associated with a higher incidence of haematoma, seroma, and infection. Other complications in the mesh repair group are small bowel obstruction, fistula from mesh to skin, enterocutaneous fistula, long-term pain, abdominal wall immobility, and foreign body sensation [5, 6, 14].

Prosthetic Mesh

The ideal surgical mesh should be inert, flexible, non-carcinogenic, biologically inactive, have long-term strength to prevent recurrence, have fast body incorporation, and should not affect human tissue distensibility. Unfortunately, nowadays, surgical mesh may have most but never all of the qualities noted here [15].

The mesh may have mono- or multifilament structures knitted to provide pores, and the pore variety determines the mesh's characteristics and its successful usage. The pore size is a determinant of the tensile strength; it also affects neovascularization, the infection resistance, and collagen fiber growth.

There are three different categories of prosthetic meshes used in ventral hernia repair: synthetic polymers, composites and biologic prosthetics (Table 12.1). Synthetic polymers can be classified in absorbable and non-absorbable [15, 16].

Table 12.1 Types of prosthetic material for the repair of complex abdominal hernias

Synthetic	
Nonabsorbable polymers	Polypropylene
	Polyester
	Expanded polytetrafluoroethylene (ePTFE)
Absorbable synthetic polymers	
Composites	
Biologic prosthetics	Human
	Bovine
	Swine

Synthetic Non-absorbable Polymers

This category includes polypropylene, polyester, and expanded polytetrafluoroethylene (ePTFE).

Polypropylene

This type of mesh is the most widely used because of its strength, ease of handling, and versatility (Fig. 12.1a). It was first used in the 1950s and has a rough surface which prevents the mesh from slipping. This mesh is extremely resistant to biodegradation, is not destroyed by tissue enzymes, and is very flexible in surgical use. The mesh is arranged in mono- or multifilament combination and classified in lightweight or heavyweight. Heavyweight meshes consist of pore sizes smaller than 1 mm; meanwhile, meshes with pores larger than 1 mm are called lightweight. Lightweight meshes result in a reduced amount of mesh material after incorporation and cause less abdominal stiffness. The heavyweight mesh supports six times normal abdominal tension. This leads to high resistance, but also to higher rates of severe chronic pain and abdominal stiffness when compared to lightweight mesh, which simulates more closely human tissues. Furthermore, heavyweight meshes trigger more adverse inflammatory response, although animal studies showed that the 1 month after surgery tensile strength seems to be similar. Both lightweight and heavyweight polypropylene prosthetics were noted to shrink 30–50 % in a 6 month period of time. Due to this shrinkage, a 3–5 cm overlap of meshes is recommended during hernia repair to avoid recurrences at the mesh margins (Fig. 12.1b). Complications such as migration, infection, hernia recurrence, and functional impairment may occur when using polypropylene mesh. In the long term, restriction of abdominal wall movement can be observed due to mesh stiffness which is caused by an intense inflammatory response (Fig. 12.1c). Many studies have also shown that polypropylene is very adhesive to intestinal serous when used in direct contact with abdominal organs. This explains why this type of mesh is rarely used in direct contact with the peritoneal cavity as well as in laparoscopic repairs (Fig. 12.2). Among all the absorbable prosthetic meshes, the polypropylene meshes are the type which best handle acute infection [17, 18].

Polyester

Polyester is a carbon polymer, multifilament, and nonabsorbable material which was used the first time in 1956. Although this type of mesh is less popular than polypropylene meshes, it has the same indications of usage. However, studies have shown higher rates of recurrence and infection with this mesh when compared to polypropylene meshes [17].

Expanded Polytetrafluoroethylene (ePTFE)

The ePTFE mesh is also a non-absorbable prosthetic mesh which varies from both polypropylene and polyester due to its micropores and its advantages in intraperitoneal hernia repair. This fluorocarbon polymer, which came on the scene

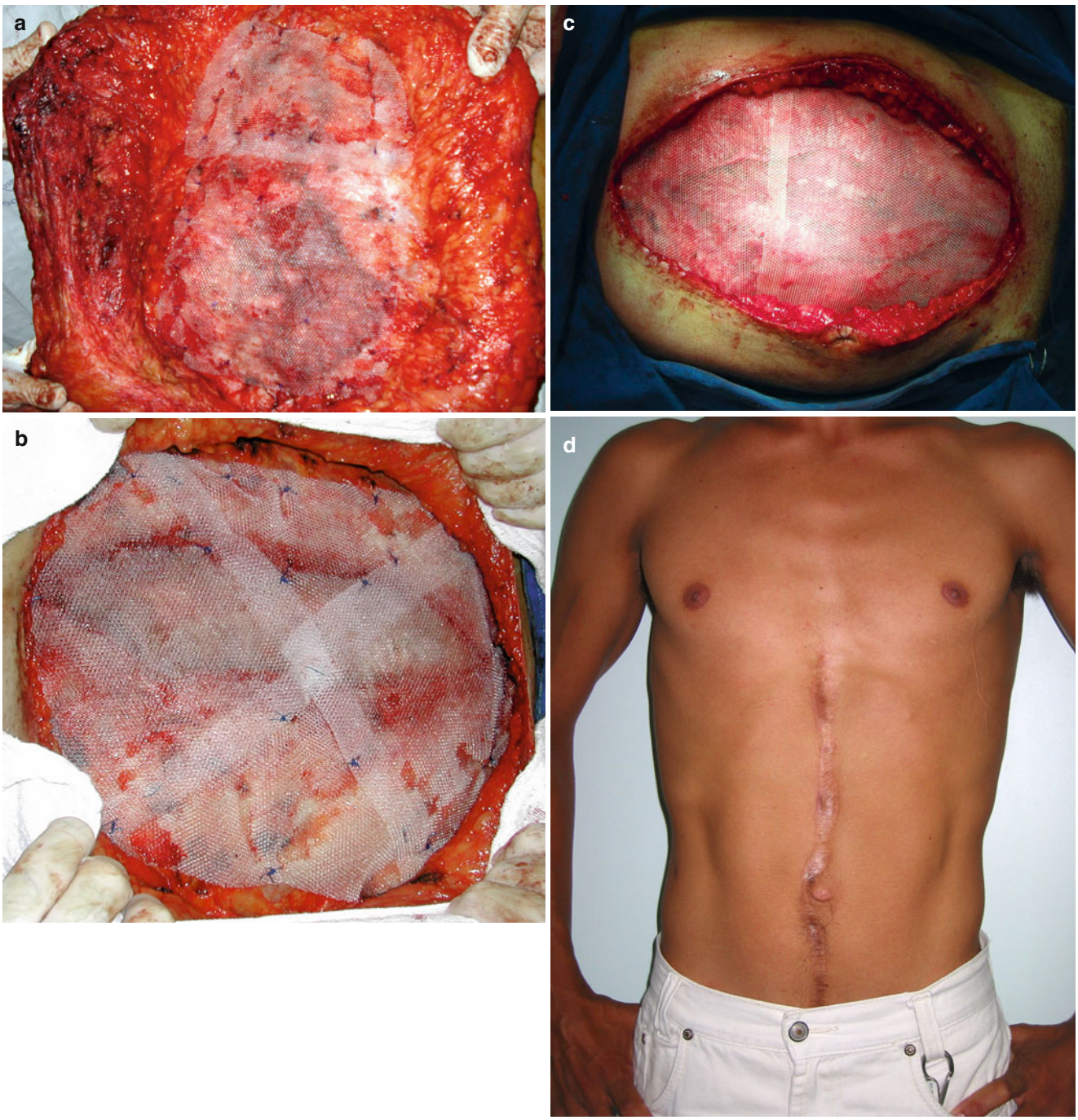


Fig. 12.1 (**a**). Polypropylene mesh repairing a small abdominal wall defect. (**b**). Polypropylene mesh repairing a hernia after peritoneostomy covering scar area and part of the aponeurosis. (**c**). Polypropylene mesh repairing complex abdominal wall defect. (**d**) Postoperative (1-year) anterior view demonstrates stable abdominal wall reconstruction

in 1963 and has a favorable biologic behavior and smooth surface with pore sizes smaller than 3 μm, can be placed in direct contact with abdominal viscera due to its low adhesive risk. Furthermore, ePTFE meshes are stiffer and can be double-faced, meaning that they have both a regular side and a side with larger pores. The viscera side is anti-adhesive while the other side allows cellular penetration and adhesion formation. Although the ePTFE mesh is a good option for intraperitoneal contact and laparoscopic surgeries, it has less tensile strength than other meshes. Its smaller pores allow less fibrotic formation and have higher rates of infection. Finally, the ePTFE prosthesis has higher shrinkage rates when compared to polypropylene, which leads to more recurrence [17].

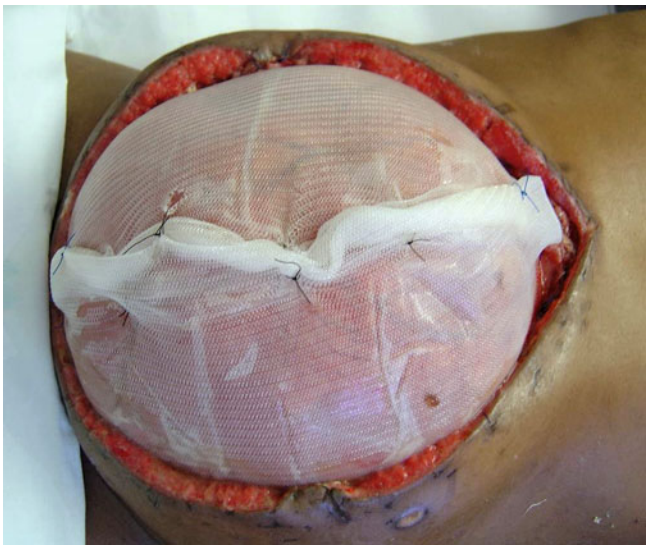


Fig. 12.2 Polypropylene mesh associated to Bogota bag to contain recurrent peritoneostomy evisceration

Absorbable Synthetic Polymers

These polymers consist primarily of polyglycolic acid, which can be or cannot be associated with lactic acid. The use of synthetic polymers is normally restricted to temporary abdominal closure. As opposed to nonabsorbable polymers, absorbable prostheses are hydrolyzed with time. This mesh was developed in the 1980s due to high infection rates of non-absorbable meshes when applied to contaminated surgical fields. The absorbable mesh is more flexible and easier to handle. Theses meshes have been used for temporary closure of contaminated surgical wounds. Due to their absorbable characteristics, the development of postoperative incisional hernia is expected. Therefore, these meshes should not be used alone for the repair of hernias in clean surgeries. The absorbable synthetic polymers are also used together with non-absorbable polymers. This combination results in a mesh with partial absorption and less prosthetic volume after tissue incorporation, allowing long-term comfort [17].

Composites

Composite prosthetics are meshes produced with more than one type of material and are designed to be placed in contact with the peritoneal cavity because of their non-adhesive properties. They are usually made of polypropylene or polyester, and one of the sides is covered with a product which will form a barrier between the abdominal content and the mesh when applied. This product can be non-absorbable (titanium, polyurethane, ePTFE) or absorbable (omega-3 fatty acid, collagen hydrocel, oxygenated regenerated cellulose). When the protective layer is absorbable, there is a chance of adherence after degradation. Polypropylene and ePTFE composites are widely used intraperitoneally. They offer both the polypropylene's advantages, such as resistance and fibroplasias, as well as the ePTFE's safeness due to its low adhesive properties. These composite meshes have been successfully applied on inlay position in order to repair complex and multirecurrent anterior abdominal wall hernias in association with flaps and muscular sheath advancements [8, 17, 19–22].

Biologic Prosthetics

Biologic prosthetics are acellular collagen backbones derived from allogenic (cadaver) or xenographic (non-human) sources. These are the most recent materials used in hernia repair. The tissues used (human, bovine, or swine) undergoes procedures that eliminate cellular material, leaving a matrix that retains a structurally intact basement membrane, intact collagen fibers, and intact elastin and laminin filaments, serving as a supporting surface for cellular repopulation and neovascularization. The most used biologic meshes are the ones derived from human dermal matrix, porcine small intestine sub mucosa, porcine dermis, and bovine pericardium. The use of biological meshes in ventral hernia was first described in 2003. These prostheses can be used on contaminated wounds, and in general they do not cause adhesion when placed in direct contact with abdominal viscera. Although its tensile strength is similar to synthetic prosthetic meshes, biologic grafts have been used mostly for reconstructive surgery, particularly during contaminated and complex cases. These meshes may be applied intraperitoneally or extraperitoneally. Some biological prostheses need to be stored in a refrigerator, while others may be stored in natural temperatures. Rehydration may be necessary 30–40 min before implanting certain biologic mesh types. The biological meshes have the highest costs. Meshes from human tissues cost approximately \$26.00/cm^2 while meshes from porcine and bovine tissues can cost from \$8.60 to \$22.00/cm^2. The synthetic absorbable and non-absorbable meshes cost approximately \$100/cm^2 [17, 19].

Fibrin Sealant in Hernia Repairs

Fibrin sealant is proven to be an efficacious alternative to mechanical methods for the sealing of meshes used in CAWD surgery. It offers several advantages over mechanical methods. Fibrin sealant reproduces the final steps of the human coagulation cascade, making it biocompatible with the surrounding tissue. Furthermore, the results obtained in inert simulation models and experimental animals were similar to those observed in the sealing of mesh with mechanical means in patients. In patients treated with fibrin sealant, a lower

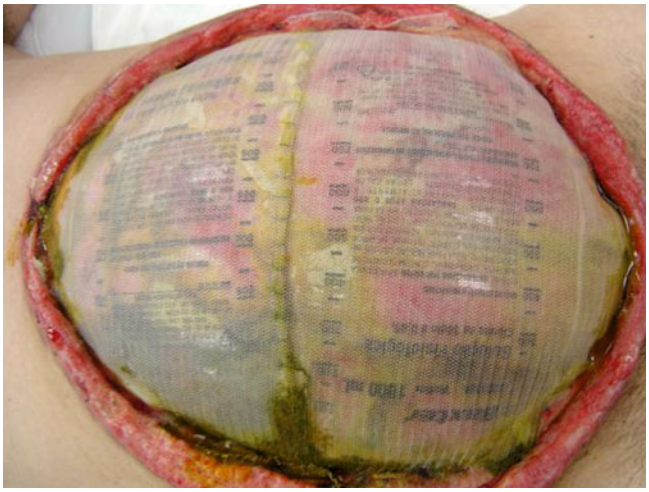


Fig. 12.3 Enterocutaneous fistula in a patient with peritoneostomy contained by Bogota bag and polypropylene mesh

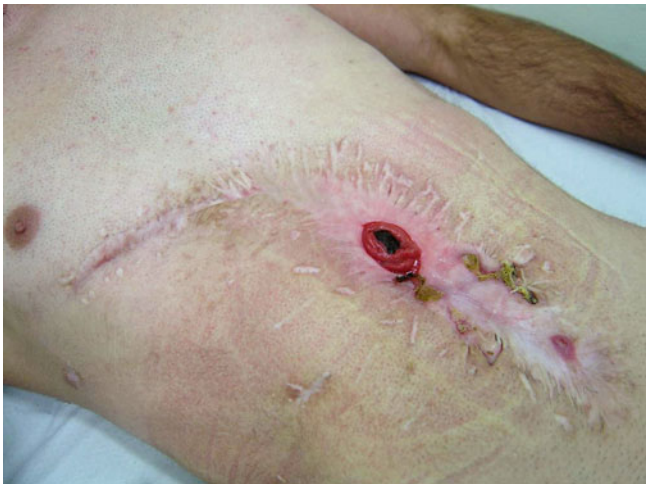


Fig. 12.4 Patient who underwent damage control procedure with an impaired wound healing and colocutaneous fistula and mesh rejection

prevalence of acute and chronic postoperative pain is observed, as is a lower number of hemorrhagic problems (hematoma, ecchymosis, bleeding). At the experimental level, the intraperitoneal formation of adhesions with fibrin sealant was less than that observed with the use of mechanical sealing methods. However, there are no data indicating that fibrin sealant decreases the appearance of seroma [6, 14, 23–25].

Very few studies evaluating cost effectiveness and satisfaction of the health-care professional with this technique are available, and those that exist are not consistent. However, it is possible to hypothesize that the use of fibrin sealant might reduce the costs associated with abdominal hernia surgery. Two randomized clinical trials demonstrated significant reductions in hospital stay and in acute and chronic pain, faster return to normal activity, and significant reductions in bleeding complications when fibrin sealant were used in hernia surgery [23–25].

Complications

Complications include migration, infection, delayed healing, skin necrosis, enterocutaneous fistula formation, functional impairment, and hernia recurrence (Figs. 12.3 and 12.4). Hypertension, smoking, body mass index (BMI) > 30, and diabetes are relevant risk factors for complications following CAWD surgery. Patients with two or more risk factors are at a greater risk for complications, including hernia recurrence, as compared to those with a single risk factor [5, 6].

Complication rates have been described as significantly higher when mesh is used compared with primary closure without mesh [6, 10].

Conclusion

The surgeon should apply the principles of reconstruction to serve as the basis of an individualized strategy that will offer the best outcome. Meticulous attention to technique, timing, utilization of new technology, and tension-free repair in a clean, well-vascularized wound continue to be the cornerstones of the ideal repair. Focus on an individualized strategy is also important when selecting the correct prosthetic material.

The management of complex abdominal wall defects remains challenging. The abdominal wall has a variety of functions, all of which rely on an established complex interaction between dynamic muscle layers and a static fascial framework. Various reconstructive options exist, ranging from simple to more complex. When addressing abdominal wall defects, the surgeon constantly must be focused on recreating a stable core that is both structurally strong and functional.

Risk factors, comorbidities, hernia recurrence, and presence of contamination are indispensible issues to be considered prior to facing the challenge of approaching an abdominal wall defect.

References

1. Luijendik RW, Hop WC, Van Tol MP, de Lange DC, Braaksma MM, IJzermans JN, et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med. 2000;343:392.
2. Shell DHIV, de la Torre J, Andrades P, Vasconez LO. Open repair of ventral incisional hernias. Surg Clin North Am. 2008;88:61–83.
3. Millenium Research Group. US Markets for Soft Tissue Repair 2009. Toronto: Millennium Research Group, Inc; 2008.
4. Davison SP, Parikh PM, Jacobson JM, Iorio ML, Kalan M. A "buttressed mesh" technique for fascial closure in complex abdominal wall reconstruction. Ann Plast Surg. 2009;62(3):284–9.

5. Hadeed JG, Walsh MD, Pappas TN, Pestana IA, Tyler DS, Levinson H, et al. Complex abdominal wall hernias: a new classification system and approach to management based on review of 133 consecutive patients. Ann Plast Surg. 2011;66(5):497–503.
6. Ghazi B, Deigni O, Yezhelyev M, Losken A. Current options in the management of complex abdominal wall defects. Ann Plast Surg. 2011;66(5):488–92.
7. Lipman J, Medalie D, Rosen MJ. Staged repair of massive incisional hernias with loss of abdominal domain: a novel approach. Am J Surg. 2008;195(1):84–8.
8. Kolker AR, Brown DJ, Redstone JS, Scarpinato VM, Wallack MK. Multilayer reconstruction of abdominal wall defects with acellular dermal allograft (AlloDerm) and component separation. Ann Plast Surg. 2005;55(1):36–42.
9. Burger JW, Luijendijk RW, Hop WC, Halm JA, Verdaasdonk EG, Jeekel J. Long-term follow-up of a randomized controlled trial of suture versus mesh repair of incisional hernia. Ann Surg. 2004;240(4):578–85.
10. Sauerland S, Schmedt CG, Lein S, Leibl BJ, Bittner R. Primary incisional hernia repair with or without polypropylene mesh: a report on 384 patients with 5-year follow-up. Langenbecks Arch Surg. 2005;390(5):408–12.
11. den Hartog D, Dur AH, Tuinebreijer WE et al. Open surgical procedures for incisional hernias. Cochrane Database Syst Rev. 2008; 16(3):CD006438.
12. Tagaya N, Mikami H, Aoki H, Kubota K. Long-term complications of laparoscopic ventral and incisional hernia repair. Surg Laparosc Endosc Percutan Tech. 2004;14(1):5–8.
13. Kurmann A, Visth E, Candinas D, Beldi G. Long-term follow-up of open and laparoscopic repair of large incisional hernias. World J Surg. 2011;35(2):297–301.
14. Ventral Hernia Working Group, Breuing K, Butler CE, Ferzoco S, Franz M, Hultman CS, et al. Incisional ventral hernias: review of the literature and recommendations regarding the grading and technique of repair. Surgery. 2010;148(3):544–58.
15. Pandit AS, Henry JA. Design of surgical meshes - an engineering perspective. Technol Health Care. 2004;12(1):51–65.
16. Anurov MV, Titkova SM, Shchegoleva NN, Mikhaleva LM, Tsitovich IG, Galushkina NV, et al. Experimental study of the impact of the textile structure of mesh endoprostheses for the efficiency of reconstruction of the anterior abdominal wall. Bull Exp Biol Med. 2008;145(5):642–6.
17. Shankaran V, Weber DJ, Reed 2nd RL, Luchette FA. A review of available prosthetics for ventral hernia repair. Ann Surg. 2011; 253(1):16–26.
18. Schug-Pass C, Tamme C, Tannapfel A, Köckerling F. A lightweight polypropylene mesh (TiMesh) for laparoscopic intraperitoneal repair of abdominal wall hernias: comparison of biocompatibility with the DualMesh in an experimental study using the porcine model. Surg Endosc. 2006;20(3):402–9.
19. Chavarriaga LF, Lin E, Losken A, Cook MW, Jeansonne LO, White BC, et al. Management of complex abdominal wall defects using acellular porcine dermal collagen. Am Surg. 2010;76(1):96–100.
20. Rosen MJ. Biologic mesh for abdominal wall reconstruction: a critical appraisal. Am Surg. 2010;76(1):1–6.
21. Limpert JN, Desai AR, Kumpf AL, Fallucco MA, Aridge DL. Repair of abdominal wall defects with bovine pericardium. Am J Surg. 2009;198(5):e60–5.
22. Lin HJ, Spoerke N, Deveney C, Martindale R. Reconstruction of complex abdominal wall hernias using acellular human dermal matrix: a single institution experience. Am J Surg. 2009;197(5):599–603.
23. Morales-Conde S, Barranco A, Socas M, Alarcón I, Grau M, Casado MA. Systematic review of the use of fibrin sealant in abdominal-wall repair surgery. Hernia. 2011;15(4):361–9.
24. Matthews RD, Neumayer L. Inguinal hernia in the 21st century: an evidence-based review. Curr Probl Surg. 2008;45:261–312.
25. Hultman CS, Tong WMY, Kittinger BJ, Cairns B, Overby DW, Rich PB. Management of recurrent hernia after components separation: 10-year experience with abdominal wall reconstruction at an academic medical center. Ann Plast Surg. 2011;66:504–7.

Reconstruction of Abdominal Wall in Trauma Patients After Damage Control

13

Mayur Narayan, Eduardo D. Rodriguez, and Thomas M. Scalea

Introduction

The appearance of crack cocaine in the United States in the late 1980s led to a marked increase in interpersonal violence on the streets of American cities. High-caliber weaponry and multiple trajectories became common. Major urban level 1 trauma centers were flooded with badly injured patients. Traditional therapy dictated that definitive operative care be performed on the night of presentation. It soon became clear that this was not wise. Patients with multiple injuries required many hours of operative care, as well as large volumes of fluid and blood transfusions to support them through their initial resuscitation. The chest, abdomen, or both body cavities were typically open for hours. Patients developed what was described as the lethal triad of hypothermia, coagulopathy, and acidosis. Patients were then transported to the intensive care unit (ICU) postoperatively, where they often died early of the consequences of prolonged shock. Those who survived developed multiple organ failure and often succumbed days or weeks later.

To a large extent through trial and error, surgical practice evolved to using sequential therapy, limiting operative care the night of injury to only absolutely necessary procedures, deferring non-life-threatening procedures until later. Packing had been used as adjunctive hemostasis in the liver for some time. These concepts were broadened, and this therapy became commonly used throughout the abdomen and then the chest. The technique was termed "damage control" by Rotondo and Schwab in the early 1990s. Adjunctive hemostatic techniques, such as angioembolization, were often employed to supplement operative hemostasis. Further therapy was staged over the next hours or days, depending on the patient's physiologic response to injury and critical organ performance [1].

Damage Control

The use of damage control or the abbreviated laparotomy has four early phases. The first phase takes place in the field or trauma resuscitation unit with recognition that the patient is severely injured. Initial care involves rapid emergency medical system (EMS) transport, early resuscitation, and initiation of the massive transfusion protocol. Ideally, the decision to initiate damage control should be made at this time. The second phase of damage control takes place in the operating room and involves identification of injury, control of hemorrhage and contamination, liberal use of intra-abdominal packing, and a temporary abdominal closure. During the third phase, the patient is then transported to the ICU for further stabilization and resuscitation, rewarming, correction of accompanying coagulopathy, as well as maximization of hemodynamic and ventilatory support. Adjunctive hemostasis can be accomplished with angiographic embolization. Patient trajectory during this phase will determine the optimal time for the final phase of damage control surgery, a return to the operating room for planned reexploration, pack removal, definitive repair of all remaining injuries, and closure of the abdomen, if possible.

M. Narayan, MD, MPH, MBA • T.M. Scalea, MD (✉)
Program in Trauma, R Adams Cowley Shock Trauma Center,
University of Maryland School of Medicine,
22 South Greene Street, Room P1G01,
Baltimore, MD 21201, USA
e-mail: mnarayan@umm.edu; tscalea@umm.edu

E.D. Rodriguez, MD, DDS
Department of Plastic, Reconstructive, and Maxillofacial Surgery,
University of Maryland School of Medicine,
22 South Greene Street, Baltimore, MD 21201, USA
e-mail: erodriguez@umm.edu

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_13, © Springer Science+Business Media New York 2013

Table 13.1 Patient selection for damage control

High-energy blunt torso trauma/Injury Severity Score (ISS) > 35
Multiple torso penetrations
Hemodynamic instability
Presenting coagulopathy or hypothermia
Multifocal or multicavitary exsanguination with concomitant visceral injuries
Major abdominal vascular injury with multiple visceral injuries
Multiregional injury with competing priorities
Severe metabolic acidosis (pH < 7.2)
Hypothermia (temperature < 34 °C)
Resuscitation and operative time > 90 min
Coagulopathy as evidenced by development of nonmechanical bleeding
Massive transfusion (>10 units packed red blood cells)

Reprinted with permission of Elsevier from Rotonda and Zonies [2]

Table 13.2 Reasons for leaving the abdomen open

Distended midgut/inability to close
Prevention of primary abdominal compartment syndrome
Planned second-look reoperations
Lap pad removal
Reassessment of injury/evolving injury
Definitive repair of visceral injuries
Loss of abdominal domain

The decision to utilize damage control techniques must be made early, before the patient develops terminal shock, often characterized by refractory hypotension despite seemingly adequate resuscitation. The so-called lethal triad of acidosis, hypothermia, and coagulopathy is often present. If the surgical team initiates damage control late, the chances of a good outcome are markedly diminished.

Patient selection (Table 13.1) and the correct timing to utilize damage control are probably the two most important steps in maximizing survival [1, 3]. A number of parameters have been described to alert the surgeon to utilize damage control. Some of these involve anatomic injury, such as penetrating torso trauma with hypotension, the need for resuscitative thoracotomy, large-volume intraperitoneal hemorrhage with accompanying hypotension after blunt trauma, pelvic fracture with hypotension, and multicavitary trauma [4]. Others have utilized physiologic variables, which include the need for cardiopulmonary resuscitation (CPR) in the field, an admission systolic blood pressure less than 70 mmHg, and an early transfusion need of more than 10 units of packed red blood cells [5]. Laboratory values may also be helpful. These include pH less than 7.2, hypothermia (defined as body temperature < 34 °C), blood loss of more than 4 L, initial base deficit greater than −6 (particularly in patients over 55 years of age), and a lactate greater than 5 mmol/L. Coagulation parameters, including prothrombin time greater than 16 s or partial thromboplastin time greater than 50 s, have also been described as defining patients who benefit from damage control surgery [3, 6, 7].

When utilizing damage control for abdominal injury, it is almost always wise to leave the abdomen open during the first several days of resuscitation. These patients are physiologically exhausted and have more pressing needs than fascial closure. Definitive closure of the abdomen during ongoing resuscitative or immediate postresuscitative phase may lead to primary abdominal compartment syndrome (ACS), requiring emergency decompression and increasing morbidity and mortality. Temporary abdominal closure techniques are quick, easily maintained, and provide access to the intra-abdominal contents for second-look or staged repair (Table 13.2).

Extending Damage Control in the ICU

While damage control was originally described as a surgical technique, it is more correctly termed a philosophy that is used to treat the most critically injured patients. Changes in resuscitation have recently been developed that may limit the amount of visceral edema. In addition, ventilatory strategies and the use of early nutritional help reduce the number of complications. Early control of sepsis and treatment of intra-abdominal infections are also a key part of extended damage control. All of these can help limit visceral edema, prevent multiple organ failure, and perhaps prevent loss of domain and protect the fascia for an attempt at early fascial closure.

Damage Control Resuscitation

The goal of any resuscitative strategy is to ensure adequate tissue perfusion and maintain cellular aerobic metabolism. Traditionally, initial resuscitation after significant blood loss involved 2 L of isotonic crystalloid fluid infused as quickly as possible, with a goal of raising blood pressure to a normal value. Additional crystalloid fluid and blood when available was given if resuscitation was deemed incomplete. Plasma and platelets were given only after 10 units of red cells were used. However, several new strategies are now employed. Hemostatic clot forms when patients bleed to the point of hypotension. Raising blood pressure to normal simply displaces this clot, leading to a cycle of recurrent hemorrhage and resuscitation. This ultimately leads to hemodilution, ongoing shock, and utilization of clotting factors, causing coagulopathy. Unless this cycle is broken, hemorrhage will continue, and hypothermia, acidosis, and coagulopathy will soon follow [8–10].

Permissive hypotension is now a commonly utilized strategy following significant blood loss. Systolic blood pressure is allowed to remain between 70 and 80 mmHg until

hemorrhage is controlled. That level of hypotension seems to preserve the hemostatic clot but still provides sufficient critical organ perfusion. Once hemorrhage is controlled, in the operating room, interventional radiology suite, or both, raising blood pressure to normal further improves perfusion without causing recurrent hemorrhage [11].

Intravascular volume should be used as an inotrope to support cardiovascular function. In the 1980s, hyperdynamic resuscitation was commonly used. This involved large volumes of crystalloid fluid and blood, targeting fixed cardiovascular parameters as goals. Shoemaker et al. demonstrated that a cardiac index of 4.5 L/min/m^2, oxygen delivery index of 650 L/min/m^2, and oxygen consumption index of 170 L/min/m^2 correlated with survival. Later, a more tailored resuscitation strategy emerged, and lactate was demonstrated to be the most important predictor of survival [12]. In addition, Moore and colleagues demonstrated that targeting lower, more reasonable oxygen transport parameters still allows lactate to be cleared quickly but does so with less crystalloid fluid and blood. The additional blood and crystalloid fluid used to support hyperdynamic goals have been shown to correlate with increases in intra-abdominal pressure (IAP) and intra-abdominal hypertension (IAH). These all lead to ACS [8].

Intra-abdominal hypertension also leads to significant physiologic dysfunction in many organ systems. For instance, increases in IAH lead to increases in IAP and cause a decrease in filtration grading of the kidney, causing prerenal oliguria. Increases in IAP also compromise renal vein flow. These pressures are transmitted to the renal parenchyma, increasing renal vascular resistance. Low cardiac output then results from decreases in venous return and increases in intrathoracic pressures. All of these decrease renal blood flow and glomerular filtration, ultimately leading to acute renal failure. These changes are most marked in patients with closed fascia. Even in patients with an open abdomen, however, the visceral and retroperitoneal edema causes increases in IAP, and many of these same physiologic derangements can be seen [13, 14].

Damage control resuscitation involves utilizing red cell transfusions earlier. In addition, plasma and platelets are given with red cells in a 1:1:1 ratio. Data from the U.S. military suggest that when transfusion ratios of blood, plasma, and platelets approach 1:1:1, survival is improved. This has also been demonstrated in large civilian series. However, other authors have questioned whether the correct ratio is 1:1:1, and the exact ratios to optimize survival remain unclear [9].

Thus, newer resuscitation strategies utilize permissive hypotension and restricted crystalloid fluid. Blood and plasma are used earlier. All of these tend to limit edema and decrease IAP. Some patients may tolerate primary fascia closure early after injury. In those patients whose fascia must be left open, there should be a less-frequent loss of domain and less facial edge retraction [11, 15].

Once hemorrhage is controlled and the patient is in the ICU, patients often become hyperdynamic and vasodilated with relative hypotension. It can be unclear at this point whether more fluid is helpful. Invasive monitoring, using central venous and pulmonary artery pressures, is often utilized to help guide therapy. However, in these dynamic patients, cardiac compliance changes quickly, particularly during active resuscitation. The relationship between cardiac pressures and intravascular volume is unclear, limiting the use of central venous or pulmonary artery pressures when determining cardiac filling volumes. At our institution, an echocardiographic exam has been developed to estimate volumes more precisely. The focused rapid echocardiographic exam (FREE) is a transthoracic exam incorporating hemodynamic information from the echo exam combined with the patient's clinical scenario to generate treatment recommendations for the use of fluid, inotropes, and vasopressures [16].

After resuscitation and hemodynamic optimization has taken place, early diuresis should be initiated. This will allow for removal of resuscitation fluid. Use of loop diuretics is usually sufficient. If the patient is on continuous renal replacement therapy for acute renal failure, fluid can gently be removed. Either method will assist with reducing the volume of the abdomen and potentially aid in obtaining fascial closure. The risks and benefits of diuresis must be defined clearly. Improper use in the setting of ongoing resuscitation will lead to further shock and organ dysfunction [17–20].

Damage Control Ventilation

During ongoing resuscitation of the critically ill patient with an open abdomen, it is important to optimize oxygenation and ventilation while minimizing alveolar injury caused by volutrauma. Increases in intrathoracic pressures promote visceral edema formation, increasing IAP as well as decreasing cardiac output. Strategies to limit peak plateau pressures to the minimum level necessary should be initiated.

These patients are also at particularly increased risk for developing acute respiratory distress syndrome (ARDS), given the massive inflammatory response and large resuscitative volumes of fluid used. Patients with ARDS are often treated with high driving pressure ventilatory strategies. Using lower tidal volumes (6–8 mL/kg), consistent with ARDS Network (ARDSNet) recommendations, and using pressure-regulated modes of ventilation will decrease the chances for injury to the lung, limit intrathoracic pressures, and keep IAP at a minimum [21]. At the Shock Trauma Center (Baltimore, MD), airway pressure release ventilation (APRV) has become the preferred mode of ventilation. Using APRV, we have documented a reduction in ARDS-related mortality and multisystem organ failure. In addition, patients

Table 13.3 Temporary closure techniques

Technique	Materials	Advantages	Disadvantages
Skin approximation	Towel clips, running suture	Rapid; inexpensive	Skin damage; ACS (abdominal compartment syndrome) risk; prevents X-ray studies; poor fluid loss control
Silastic/plastic closure device	Intravenous saline bag, 10–10 drapes, Bogota bag	Inexpensive; inert; nonadherent	Evisceration risk; loss of domain; risk of domain; risk of ACS
Marlex ± zipper	Wittmann Patch	Ease of reexploration; maintains domain	Special equipment; multiple fascial manipulations
Vacuum pack closure	Polyethylene sheet; fenestrated; nonadherent	Inexpensive; moderate fluid control	Loss of domain; difficulty with constant suction
Vacuum-assisted closure	V.A.C./ABThera	Nonadherent; protects fascia	Specialized equipment

Wittmann Patch™ (Starsurgical, Burlington, WI)
V.A.C.®/ABThera™ (KCI, San Antonio, TX)
Marlex® (Davol, Providence, RI)

treated with APRV had a significant reduction in mortality compared to patients in the ARDSNet trial (21.4% vs. 31%). Additional advantages of APRV include reduced sedation needs, promoting greater spontaneous ventilation when compared to conventional ventilator modes [22].

Damage Control Nutrition

All patients who are treated with damage control are intensely catabolic. Thus, good nutritional support is critical. Numerous studies have shown the benefit of nutritional support in improving infection-related complications, particularly for septic, trauma, and burn patients. Nutritional support helps prevent acute calorie malnutrition, modulates immune response, and supports gastrointestinal structure and functions. Many clinicians withhold enteral feedings in patients with an open abdomen for fear of intestinal necrosis. We believe that early tube feedings in the open abdomen patient help set the stage for definitive closure. Enteral feedings provide a hyperemic response in both hemorrhagic and septic shock. They may also diminish pressor requirements during the shock state by increasing intestinal blood flow and preserving intestinal integrity [23]. Enteral feedings also provide anti-inflammatory or immunomodulation action to the small bowel. Provision of 30–50% of feeding goal appears to be adequate to maintain the mucosal barrier and prevent bacteria translocation and a subsequent, secondary hit of sepsis.

By preventing the secondary hit, the patient's systemic inflammatory response is diminished, and swelling and edema are decreased. This will facilitate earlier abdominal closure. Nutritional support in this context is clearly beneficial as early abdominal closure may lead to a decrease in intra-abdominal contamination and fistula formation [24]. Other studies have shown that permissive underfeeding, or providing nutritional support below goal levels, can result in fewer infectious outcomes and mechanical ventilator days [25].

Damage Control Infection Management

Antimicrobial therapy should be started early and directed against the most frequently expected pathogens. An antimicrobially active concentration of drugs at the site of infection must be achieved for the antibiotics to be effective. Specific, directed treatment is usually not possible initially because the infecting microorganisms and their sensitivities are not known. Source control for sepsis is a major priority in patients with an open abdomen. Teixeira et al. found that the presence of a deep soft tissue infection or intra-abdominal abscess was an independent risk factor associated with failed abdominal closure. However, they found no difference in the complication rate between prophylactic and prolonged antibiotic use [26]. Antibiotic use should be directed toward any underlying disease process that could have led to the development of the open abdomen. It can be tailored to the narrowest spectrum possible when culture-specific data and sensitivities are available.

Vogel et al. found that the inability to achieve primary abdominal closure was associated with infectious complications, including ventilator-associated pneumonia, blood stream infection, surgical site infection, and large transfusion requirements [27]. Several studies have shown that blood transfusions increase the risk of postoperative bacterial infection, ARDS, and death [28]. Once active bleeding has been controlled, physiologic derangements corrected, and hemodynamic stability restored, the risk of blood transfusion may well outweigh its benefit.

Temporary Abdominal Wall Closure Options

Several options exist to temporarily close the abdomen in a critically ill patient prior to leaving the operating room and continuing resuscitation in the ICU (Table 13.3). The simplest of these techniques is skin closure performed either by a simple running suture of skin or using a sequential towel

clip closure. Skin closure can be performed rapidly and with minimal cost using either of these techniques. However, Rutherford et al. showed that these simple closure techniques have an increased risk of skin necrosis and infection. Most important, the pressure caused by skin closure can cause ACS. In addition, the use of the towel clip closure technique will obscure radiographs and may lead to limitations in the effectiveness of any other radiographic studies, such as angiographic embolization [29].

Use of temporary fascial bridging substitutes is another option. The Bogota bag or other silo techniques are inexpensive and rapidly placed after damage control surgery. The Bogota bag is the most common method used. The saline is discarded from a 3-L bag of genitourinary irrigation, and the bag is opened. It is then cut to the appropriate size and is sewn to the skin or fascia. None of these silo techniques is able to remove abdominal fluid, and they are prone to leaking and evisceration. In addition, the patient remains at risk for enterocutaneous fistula development. Nevertheless, they remain a viable option for damage control temporary abdominal closure, especially if other techniques are not available [30–34].

Newer modalities include negative-pressure wound devices. While practices at individual institutions vary, all methods are similar. These systems do not stabilize fascia but do manage the effluent from the peritoneal cavity, allowing the nursing staff to quantify it. These systems prevent skin maceration by keeping both the patient and the bed dry. As the fluid losses are sometimes measured in liters per day, these advantages can be helpful. Commercially available devices include the KCI (San Antonio, TX) V.A.C.® Abdominal Dressing System and ABThera™ system. A homemade vacuum pack can also be used. Homemade vacuum packs can be fashioned by the surgeon, placing a 10×10 drape over the viscera as an interface to protect the bowel. Moist Kerlex™ (Covidien, Dublin, Ireland) gauze covers the abdominal defect, and nasogastric tubes clear abdominal fluid effluent. A seal is achieved using an Ioban™ (3M, St. Paul, MN) dressing. The nasogastric tubes are then connected to a suction canister via a Y connector [35–39].

The KCI Wound V.A.C. also uses a plastic barrier to protect the bowel contents. Once placed, a macroporous black sponge is placed over the plastic. Some authors recommend holding the sponge in place with several staples before applying the adhesive occlusive dressing. A suction drainage lily pad is then applied to the superficial foam layer for removal of peritoneal fluid. The ABThera system differs slightly from the traditional vacuum-assisted closure (VAC) in that it has a visceral protective layer prepackaged to cover the entire abdominal contents. This provides a theoretical advantage of preventing adhesions to the visceral mass and allowing for mobilization on subsequent washouts. Both of these systems drain effluent while attempting to minimize loss of abdominal domain [38–41].

Early Abdominal Wall Closure

Early abdominal closure is appealing because it returns the abdominal contents to their natural position, protects from external infectious sources, and limits loss of protein and fluid when compared to the open abdomen. In addition, unprotected bowel is prone to desiccation, trauma, and the dreaded complication of fistula formation. Later complications also include infections, renal failure, polyneuropathy, and formation of heterotopic ossification. Failure to achieve early closure can be a logistical nightmare, particularly if any of these serious complications occur, especially fistula formation. These patients place a heavy burden on hospital resources, requiring multiple reoperations and prolonged stay in the ICU. Thus, early definitive closure, if possible, is the best idea [42, 43].

Timing and patient selection are key factors in determining likelihood of successful early abdominal closure. However, attempts at early definitive closure may burn bridges for later repair should the repair fail. Late closure usually occurs electively 6–12 months after the initial operation and allows time for the patient to rebound from the initial insult and improve nutritional status.

After damage control laparotomy and the resuscitation often needed, the abdominal wall is typically edematous and noncompliant. There is often significant swelling of the intra-abdominal contents and massive midgut distention. In this setting, a tension-free closure of the abdominal incision is generally not possible at the time of initial reexploration. Even if closure can be performed, if ACS develops, necessitating a reopening of the recently closed abdomen, the patient will have some degree of fascial loss from the tight closure. In these cases, a better approach is to place a temporary closure device to contain and protect the exposed visceral mass (Table 13.3).

Most noncritically ill patients should start to mobilize their third-space edema by postoperative day 3–5. However, in a subset of critically injured patients with ongoing inflammation and sepsis, this process may persist for several weeks. The resultant loss of abdominal domain will lead to a larger gap, making it impossible to reapproximate fascial edges in a safe manner (Fig. 13.1).

The traditional approach to this problem has been to allow this wound to granulate and heal by secondary intention, a process that can take several weeks. Unfortunately, this process results in the difficult to manage “frozen abdomen” as there is little or no access to the abdomen for some months. It also becomes impossible to mobilize the abdominal wall off the adherent bowel loops. Any attempt at mobilization at this stage increases the likelihood of bowel injury and subsequent fistula formation [36, 44].

In this setting, the safest approach is for the granulating wound to be covered with a skin graft. After several months

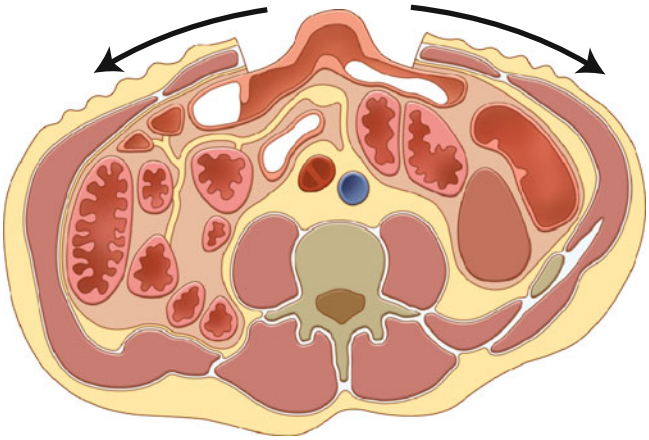


Fig. 13.1 Loss of abdominal domain can occur as the unopposed forces of the oblique muscles pull the abdominal wall in a lateral direction

of contraction, the wound will heal, and the patient will be left with a large abdominal wall hernia. Several months after the recovery from acute illness and nutrition optimization, a "planned ventral hernia" can be performed. First introduced by Fabian et al., this is a safe strategy for managing the open abdomen. Though there are few data to guide timing of hernia repair, we generally wait about 6 months to allow as much inflammation as possible to abate [45–47].

At the Shock Trauma Center, when we cannot achieve primary fascial closure, we generally place an interpositional polyglactin mesh followed by split-thickness skin grafting as a bridge to anticipated abdominal wall reconstruction. The polyglactin mesh helps prevent evisceration and further retraction of the fascial edges while the patient is rehabilitated and nutrition is optimized. At the time of reconstruction, the polyglactin mesh has been absorbed, and only split-thickness skin graft, adherent to bowel, remains. An alternative to skin grafting is to mobilize soft tissue flaps and close the skin over the mesh if there is sufficient skin [48].

Unless the fascia is stabilized early after a damage control laparotomy, it will retract laterally over time, increasing the defect and decreasing the chances of primary closure during the first admission. Techniques available early on to stabilize fascia and prevent retraction include, but are not limited to, the Wittmann Patch™ (Starsurgical, Burlington, WI), polytetrafluoroethylene (PTFE), and polyprolene mesh. All of these fascial bridges help prevent loss of abdominal domain. Cothren et al. and Weinberg et al. recommended using these techniques, particularly when the open abdomen is unlikely to be closed within the first week [49, 50]. Several studies have shown that fascial closure rates using the Wittmann patch technique range from 78% to 100%; the rates of complications, including enterocutaneous fistula, remain low [51–53].

Potential downsides to these techniques are that they are more costly than the simpler techniques and have the ability to increase fascial trauma and necrosis as the devices are anchored to the fascia. In addition, these methods do nothing to prevent adhesion formation of the viscera to the anterior abdominal wall, and they do not allow for drainage of peritoneal fluid, which may compromise the healing wound or, even worse, lead to IAH or compartment syndrome.

Use of negative-pressure therapy can aid in primary fascial closure by fluid removal or immunomodulation. Boel van Hensbroek et al. reported average primary fascial closure rates of 67% using negative-pressure therapy. Fistula rates vary between 0 and 15% [54]. Modifications to these vacuum dressings to help prevent loss of abdominal domain have also been described. Miller and colleagues have recommended placing interrupted fascial sutures every 48 h in addition to the VAC abdominal dressing. Using this technique, they were able to achieve primary closure in 86% of patients [55]. Others, such as Cothren et al., have recommended not only placing of sutures but also sequential tightening of the fascial sutures on subsequent washouts to apply tension continuously to fascia and help approximate the defect. The group reported a 100% fascial closure rate in their subset of temporary open abdominal closure patients [39, 49].

Definitive Closure and Abdominal Reconstruction

Despite the push for early aggressive abdominal closure and widespread recognition of the complications of the open abdomen, there are still subsets of injured patients who will require delayed complex abdominal wall reconstruction. There have been many techniques described to reestablish the integrity of the abdominal wall after the initial hospitalization. These include primary fascial repair and component separation with or without the use of interpositional materials such as biological or synthetic mesh (Fig. 13.2a, b, c). Tensor fasciae latae grafts have also been used as fascial substitutes. Unfortunately, no technique is without potential complications, such as recurrence of the hernia, wound infection, enterocutaneous fistulas, bowel adhesions, and the frozen abdomen [56–58].

Figure 13.3 is a proposed algorithm for late complex abdominal wall reconstructions at the R Adams Cowley Shock Trauma Center. After damage control laparotomy, the first step involves a thorough assessment of the patient's skin and fascia. If there is inadequate skin, there are three options: placement of tissue expanders, rearrangement of skin using local tissue advancement, or distant flaps. Tissue expanders must be placed several months before abdominal wall reconstruction. The expanders are filled with increasing amounts of saline each week until the skin reaches the necessary size (Fig. 13.4). A stoma or fistula increases the risk of infection in the tissue expanders. Conversely, if there is excess skin or

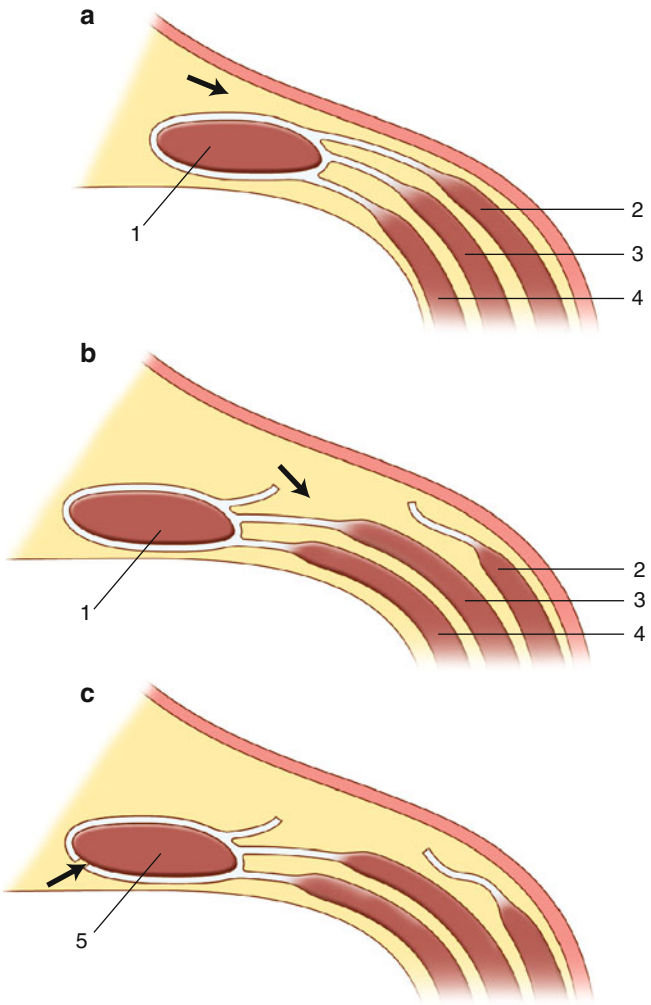


Fig. 13.2 Component separation technique. *1* Rectus abdominis muscle; *2* external oblique muscle; *3* internal oblique muscle; *4* transversus abdominis muscle; *5* posterior rectal sheath. (**a**) Dissection of skin and subcutaneous fat. (**b**) Transaction of aponeurosis of external oblique muscle and separation of internal oblique muscle. (**c**) Mobilization of posterior rectal sheath and closure in the midline (Adapted with permission of Elsevier from Bleichrodt et al. [55])

fat, the surgeon may consider a panniculectomy at the same time as the fascial reconstruction. Panniculectomy in this setting reduces the tension on the fascial closure, removes poorly vascularized tissue, and eliminates a portion of the subcutaneous dead space (Fig. 13.5a, b). However, we recognize that this creates an additional incision, increasing the chance of wound infection or incisional breakdown [48].

The surgeon must then evaluate the quantity and quality of the fascia at the time of reconstruction. Skin and soft tissue flaps can be mobilized laterally to identify the true fascial edges. Sometimes, the fascia comes together without tension. On other occasions, mobilizing the skin and subcutaneous tissue further laterally releases the fascia sufficiently to allow the edges to come together in the midline. This is the simplest method to achieve fascial closure. Even if the fascial edges come together without tension, the chance of recurrent hernia has been reported to be as high as 54%. Thus, we generally place a piece of mesh as an overlay to buttress the repair. If there clearly is insufficient fascia, there are two options: component separation with supportive mesh onlay (if primary fascial closure is obtainable) (Fig. 13.6a, b) or interpositional mesh [48, 59].

Component separation, initially described by Ramirez et al., involves incision of the external oblique fascia and muscle lateral to the rectus abdominis muscle, allowing medial advancement of the rectus abdominis muscles [57]. This facilitates primary closure of fascia under less tension. Usually, component separation allows fascial defects of 12–15 cm to be closed primarily. Additional release of the posterior rectus sheath may be added to gain an additional 1–2 cm of advancement. A mesh onlay can be placed to cover both the midline fascial repair and the relaxing external oblique incisions. This onlay is placed in an effort to decrease the risk of a lateral abdominal wall hernia, remove tension from the primary repair, and provide additional reinforcement should the primary repair fail. Mesh is used as an interpositional material to bridge the fascial defect when primary repair is not possible and is sutured underneath as an underlay several centimeters behind the leading fascial edge [60–64].

Even when there appears to be sufficient fascia, component separation can be performed to achieve a better tension-free repair. Given the incidence of hernia recurrence following component separation, we prefer to reinforce the repair with either a biological or a synthetic mesh onlay. The operating team must carefully monitor the peak airway pressures during the operation. Peak airway pressures should be measured during the closure. Pressures below 15–20 cm H_2O are considered acceptable. Bladder pressures are also monitored postoperatively to ensure that ACS does not occur [65–69]. In some patients, clinically important ACS occurs with relatively normal IAP. While the explanation for this is not clear, we believe it is the change in pressure that is important. In some patients, the change from a negative IAP to 15–20 mmHg creates ACS.

When selecting a mesh material, for use either as an onlay patch or as an interpositional fascial substitute, the risk for infection must be considered. Synthetic material such as Prolene™ (Ethicon, Somerville, NJ) mesh or Gore-Tex® (W.L. Gore & Associates, Newark, DE) should be avoided in any patient with increased risk for infection, such as patients with a contaminated abdominal wall, such as those who need ostomy takedowns or repair of an enterocutaneous fistula, and those who have an enterotomy during the procedure, open wounds, previous wound infections, or unstable wound coverage (i.e., skin grafts or secondarily healed wounds). Additional relative indications for the avoidance of synthetic mesh include general risk factors for infection, including diabetes, smoking, obesity, and immunosuppression. When

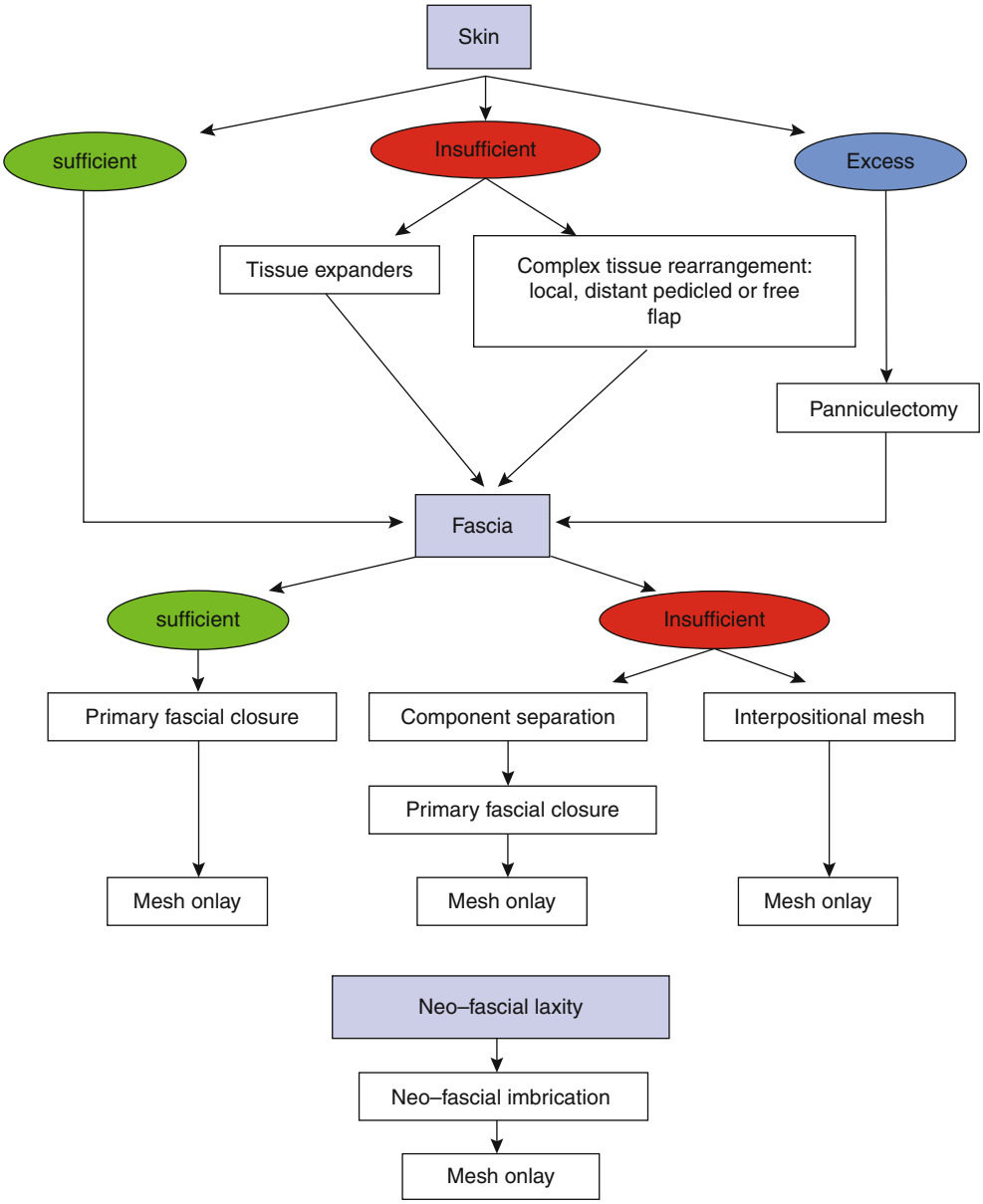


Fig. 13.3 Shock Trauma Center algorithm for abdominal wall reconstruction (Adapted with permission from Rodriguez et al. [48])

synthetic materials are contraindicated, a biological material that becomes revascularized is more likely to resist infection and is preferable [44, 56–58, 60, 61, 70–74].

The use of human acellular dermal matrix (AlloDerm®, LifeCell, Branchburg, NJ) or porcine-derived acellular dermal matrix (Strattice™, LifeCell) is attractive because of their allogenic origin and proven ability to revascularize. In patients with a high risk for infection or wound breakdown, acellular dermal matrix has several distinct advantages. First, there is neovascularization, which reduces the risk of infection. Second, there is minimal inflammatory reaction; thus, there are fewer bowel adhesions. Third, should the wound break down and the acellular dermal matrix becomes exposed, it can granulate so it does not have to be removed. However, AlloDerm is expensive, and the cost must be justified. In any situation in which temporary mesh is placed, the patient is committed to at least one more surgical procedure for hernia repair and is at risk for further complication. The cost associated with ventral hernia repair in this setting regardless of mesh used well exceeds the cost of acellular dermal matrix on a per patient basis. Currently, the long-term strength of AlloDerm is unknown [62, 73].

An important technical point is that AlloDerm must be placed under considerable tension until it will no longer physically stretch. If it is not placed under enough tension, it has a higher chance of becoming lax over time (Fig. 13.7a, b, c)

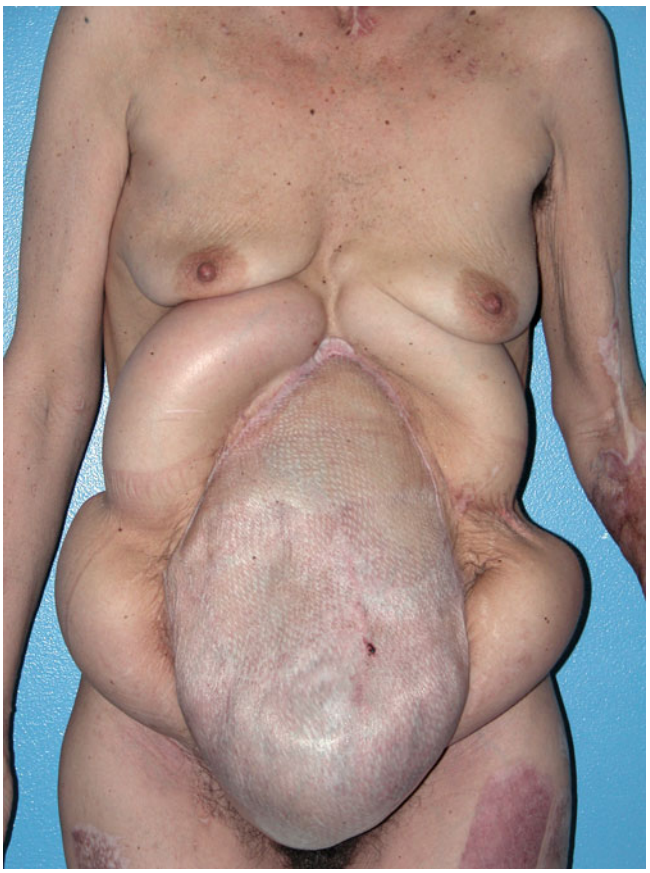


Fig. 13.4 Preoperative view of patient with four tissue expanders in place and a split-thickness graft on the bowel (Reprinted with permission from Rodriguez et al. [48])

[41]. Each piece of AlloDerm increases in length by 30–50% when placed under maximal tension because of the elastin fibers within the graft. Lateral laxity as a result of external oblique release is another possible cause of laxity, though this is quite rare. To address the possibility of laxity, we generally place an onlay of either AlloDerm or polypropylene mesh on the laterally released areas as additional support (Fig. 13.8a, b, c) [48]. Although acellular dermal matrix performs remarkably well in contaminated situations, it is important to differentiate between contamination and gross infection. Acellular dermal matrix cannot be placed in a grossly infected wound with large amounts of purulence or enteric contents. In contaminated wounds, meticulous debridement and generous irrigation still need to be performed to

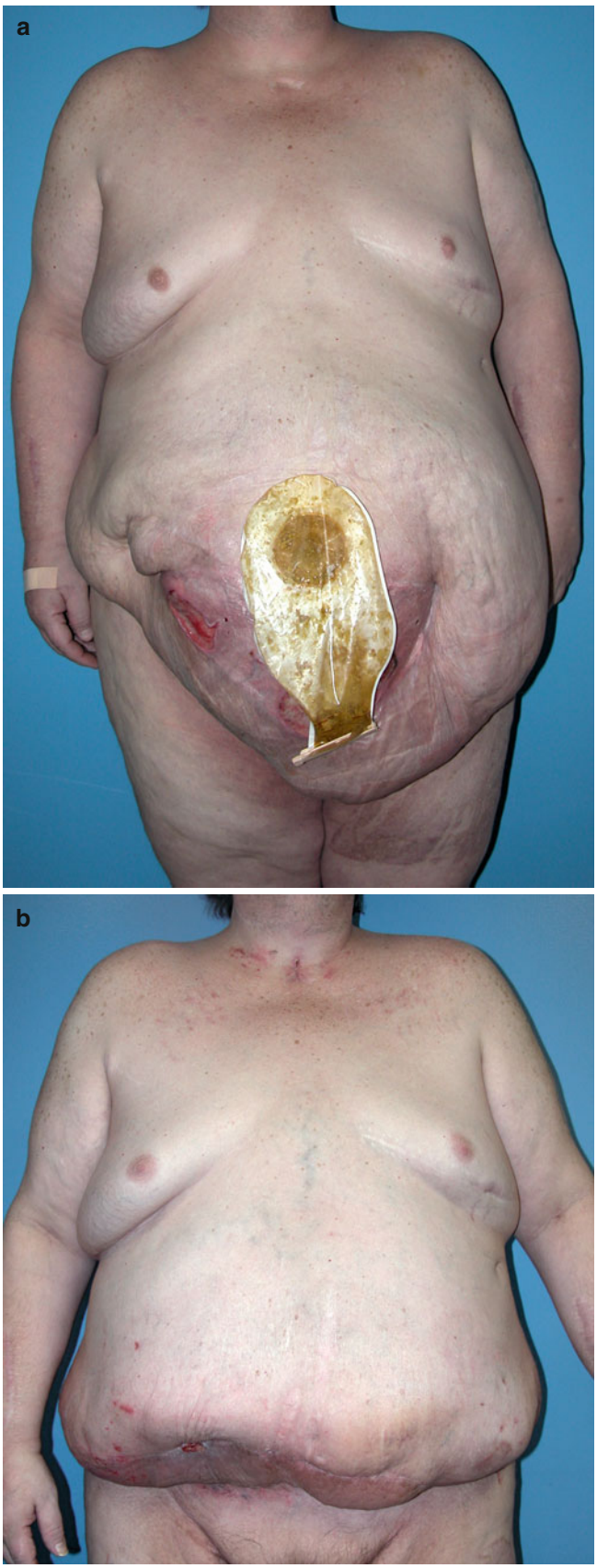


Fig. 13.5 (**a**) Preoperative view of large hernia with a split-thickness skin graft on the bowel and central ostomy. (**b**) Clinical photograph at 6-month follow-up. The wound in the right lower quadrant is superficial and did not fistulize (Both **a** and **b**: Reprinted with permission from Rodriguez et al. [48])

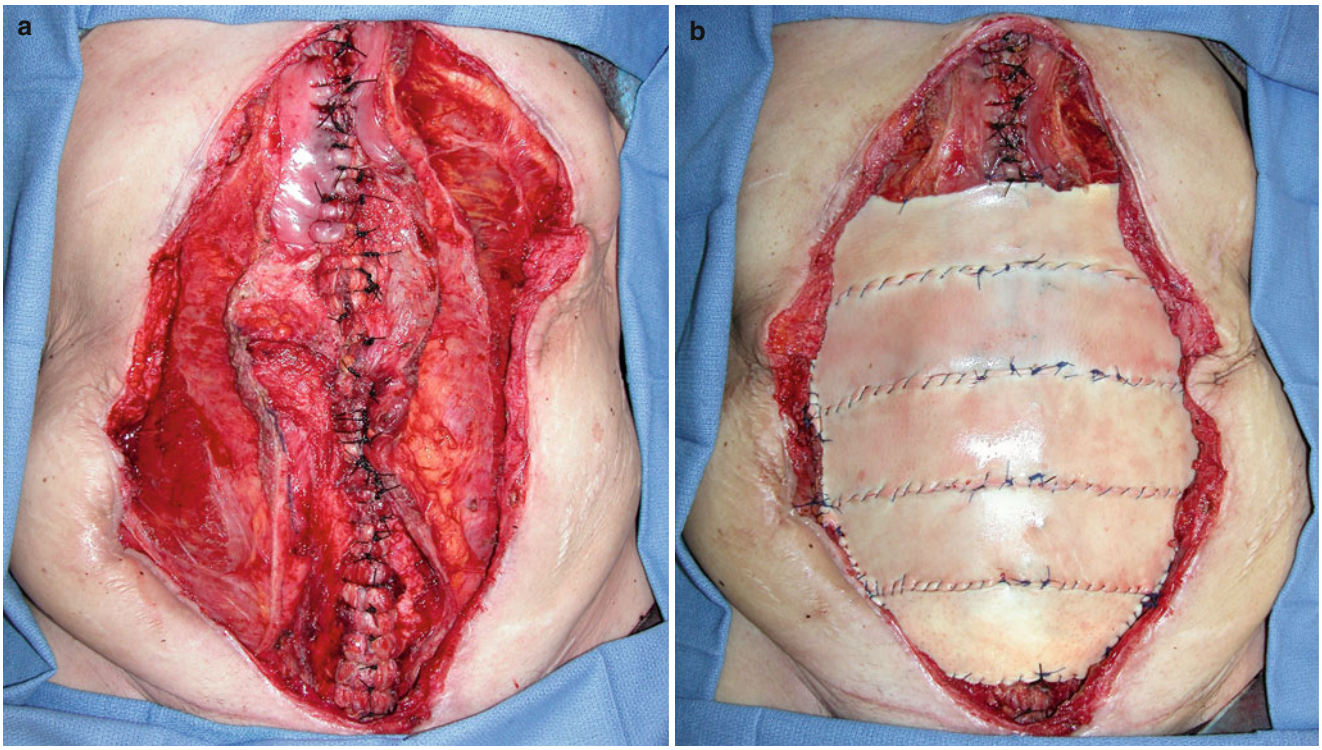


Fig. 13.6 (**a**) Primary fascial closure after compartment separation (**b**) AlloDerm only (Both **a** and **b**: Reprinted with permission from Rodriguez et al. [48])

remove debris. Acellular dermal matrix has an additional advantage in that it is resistant to adhesion formation when placed against the bowel.

Strattice is a surgical mesh derived from porcine skin that acts as a structural scaffold for wound healing. Other biological materials available are from xenogenic sources and thus may carry a risk for a gradual xenogenic response and potential resorption. All of these have properties similar to AlloDerm.

In completely sterile situations when synthetic materials are indicated, there are several options. The most commonly used products in the United States are polypropylene, Marlex® (Davol, Providence, RI); expanded polytetrafluoroethylene; and combination polypropylene/polytetrafluoroethylene. They are stronger than the biologic meshes but have some downsides.

There are three major considerations when selecting a specific synthetic product: contact with the bowel, incorporation into the surrounding tissue, and risk for wound breakdown leading to mesh exposure. If the bowel will be in contact with the mesh, expanded polytetrafluoroethylene has traditionally been the material of choice because of its resistance to adhesion formation. Polypropylene evokes a vigorous inflammatory response that can lead to dense adhesions and increased risk for fistula formation. However, when fascial closure is possible and mesh is used as an onlay, we prefer polypropylene over expanded polytetrafluoroethylene. Unlike expanded polytetrafluoroethylene, which is encapsulated by the surrounding tissue, polypropylene induces fibrous ingrowth, strengthening the repair. Finally, if vascular supply to the skin flaps is at all questionable, polypropylene is preferred because it does not need to be removed if it becomes exposed [75]. Synthetic materials have an increased risk of infection, bowel adhesions, fistulization, ulceration, and extrusion. In contaminated cases, placement of mesh carries a 50–90% risk of infection [76–79].

Composite polypropylene and expanded polytetrafluoroethylene products use the advantages of each of the material types. The expanded polytetrafluoroethylene portion faces the bowel and therefore minimizes adhesion formation. The polypropylene portion faces the more superficial soft tissues and encourages incorporation. Of course, the product never becomes completely incorporated because of the expanded polytetrafluoroethylene portion, and exposure of the implant generally requires removal. Furthermore, the product is expensive, and extreme care must be taken during placement so that the polypropylene portion does not contact the bowel at the periphery. There are reports of increased complications with combination mesh that might be attributable to technical error rather than mesh failure [46, 80–82].

When a true fascial substitute is needed, we prefer to use a combination of acellular dermal matrix and polypropylene mesh. The acellular dermal matrix is placed as an interpositional graft when there is direct contact with the bowel

because there is less adhesion and fistula formation. Polypropylene is used as an onlay because of its ability to incorporate into surrounding tissue. It should be emphasized that synthetic material should only be used when there is a low risk of infection [48].

Finally, tensor fasciae latae is another biological option; however, this is less dynamic and thus is associated with a higher incidence of recurrence. Free tissue transfer is a sophisticated operation. A myocutaneous flap can be harvested. The vascular supply comes from the gastroepiploic vessels.

There are several methods of recruiting innervated, well-vascularized skin and soft tissue. Local rotational flaps are ideal for smaller soft tissue defects. For large defects, local options may not provide sufficient soft tissue, and the blood supply to the flap might be unreliable, particularly in the setting of multiple previous operations. Distant tissue transfer, such as the pedicled anterolateral thigh flap or the pedicled extended deep inferior epigastric artery perforator flap, can be used when the defect is too large for local flaps (Fig. 13.9a–e) [39, 84, 85].

We generally use fasciocutaneous flaps rather than muscle flaps to minimize donor site morbidity and prevent tissue atrophy. The loss of a muscle in an already-debilitated patient is not inconsequential. Pedicled distant flaps provide outstanding functional and cosmetic results but are associated

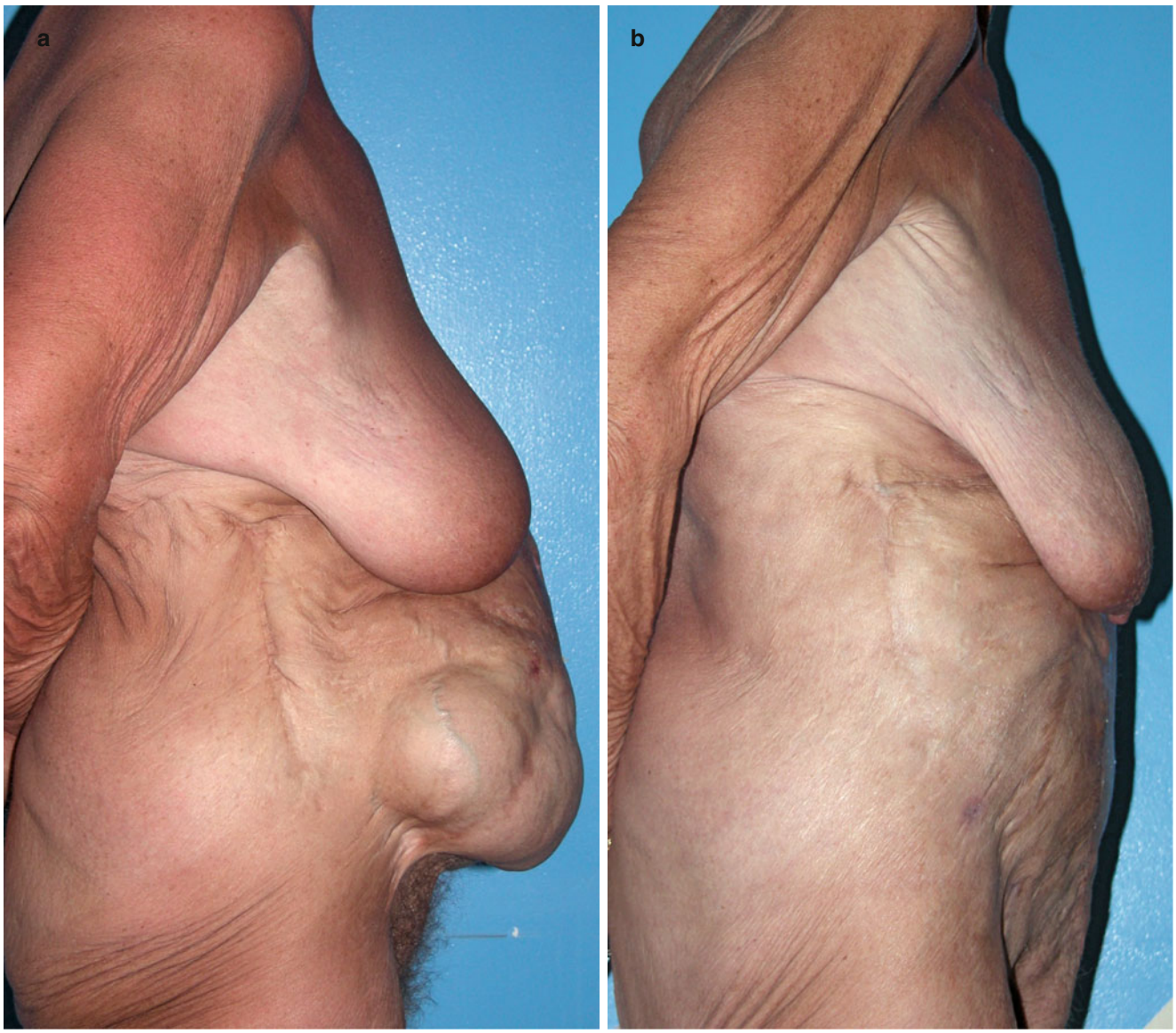


Fig. 13.7 (**a**) Preoperative clinical photographs of massive ventral hernia with severe loss of domain. (**b**) Two-month postoperative clinical photograph following component separation and interpositional AlloDerm. (**c**) Clinical photograph of abdominal wall laxity 11 months following hernia repair (**a–c**: Reprinted with permission from Bluebond-Langner et al. [41])

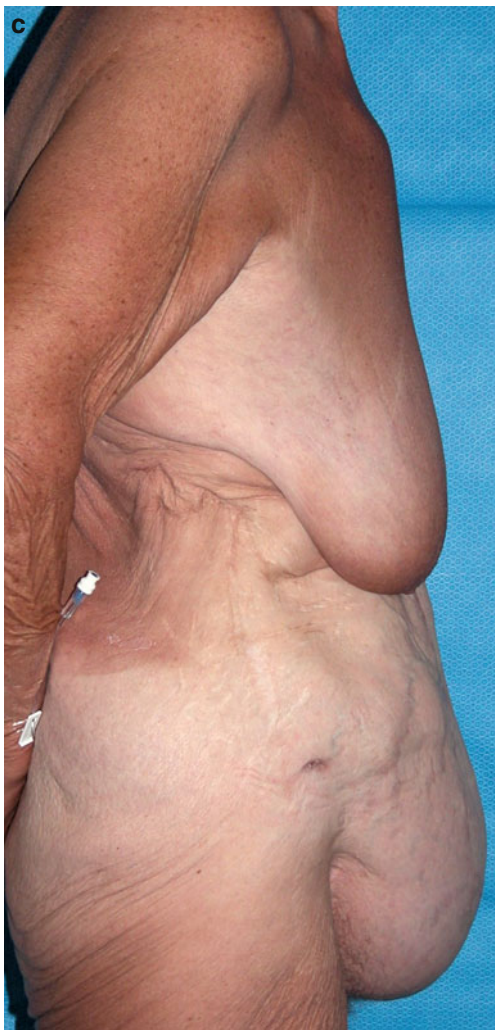


Fig. 13.7 (continued)

with some donor site morbidity and increased operating room time.

In our hands, the anterolateral thigh flap (ALT) has become a reliable donor tissue for the reconstruction of post-traumatic defects. The anterolateral thigh has many advantages, including a predictably long pedicle, a variety of tissue types, and the fact that it facilitates a two-team approach [80, 85].

Tissue expansion is another method of recruiting soft tissue for large defects. Rohrich et al. suggest tissue expansion as an option for defects larger than 15 cm. We generally use tissue expanders for defects wider than 20 cm. Placement of tissue expanders around the fascial defect is an easy surgical option that can be performed on an outpatient basis. The tissue expanders can be placed suprafascially, expanding only the skin, or beneath the external oblique aponeurosis, expanding the fascia. We prefer suprafascial placement because of the concern that the fascia may become attenuated by the expansion if placed subfascially [46, 48, 82].

There are clear disadvantages to using tissue expanders, and the expanders should be considered only in patients with severe skin deficiencies. Expansion is an intensive and sometimes-painful process for the patient, who must return to the clinic for weekly expansion. In addition, the risk of infection, extrusion, and hematoma formation has been reported to be as high as 20%.

Complications of Abdominal Wall Reconstruction

The complications associated with complex abdominal hernia repair have a wide continuum of severity and, if not recognized early, can be devastating. Because abdominal wall reconstruction occurs following multiple abdominal procedures, the vascular supply of the surrounding soft tissue envelope has been interrupted and can predispose the patient to wound infections and partial skin flap necrosis. The extensive undermining that is often necessary for proper fascial closure can predispose patients to seroma formation. The incidence of seroma formation is reported to be as high as 27%. Seromas are associated with an increased risk of infection, wound dehiscence, flap necrosis, and reoperation. Placement of multiple suprafascial drains can decrease the rate of seroma formation. It is important that these drains be left in until the skin flap has adhered to the fascia. Fibrin sealants have been used to decrease the incidence of seroma formation following breast surgery and may have an application in abdominal wall surgery, but these have been met with mixed success [86–89].

When defining the fascial extent, a large portion of the bowel is often temporarily placed outside the abdominal cavity and then carefully packed back into the peritoneum. As a result, patients are at increased risk for ACS and respiratory failure, particularly in the postoperative period. Routine bladder and peak airway pressure measurements are useful adjuncts to clinical examination when monitoring for ACS. After major abdominal surgery, diaphragm movement can be restricted with subsequent lung volume reduction and atelectasis, resulting in hypoxemia. Careful assessment of volume status and pulmonary mechanics before extubation and aggressive pulmonary toilet following extubation can minimize respiratory failure and reintubation [38, 68, 90].

Conclusion

The Shock Trauma Center approach described in Fig. 13.3 incorporates the majority of the reconstructive options and presents them in a systematic fashion. This approach provides a reliable and reproducible method for assessing patients with complex abdominal hernias and determining a logical course of treatment. We advocate aggressive

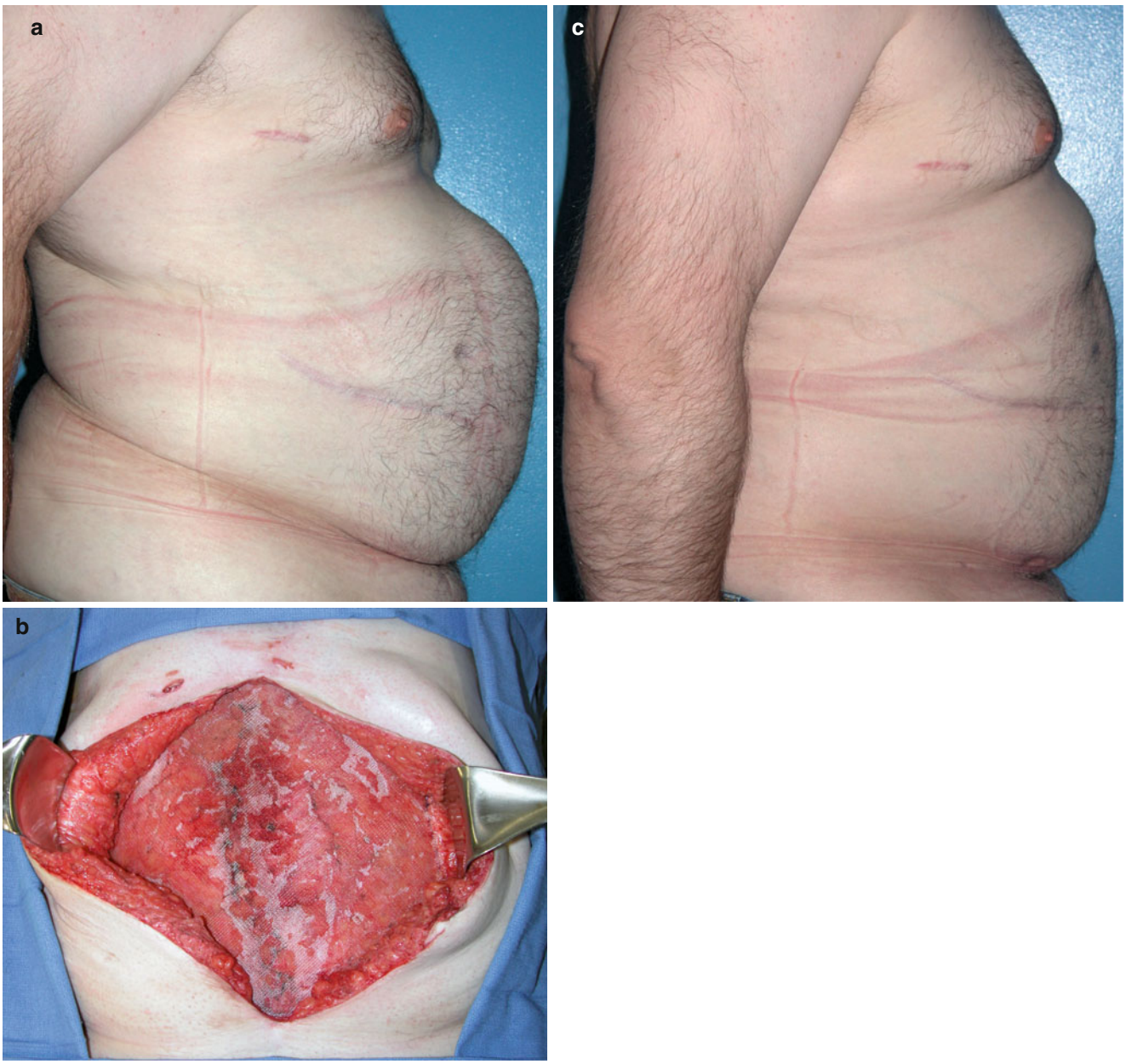


Fig. 13.8 (**a**) Clinical photograph obtained 1 year after reconstruction demonstrating superior bulge. (**b**) Polypropylene mesh onlay following excision of attenuated AlloDerm. (**c**) Clinical photograph 6 months after placement of polypropylene mesh (**a–c**: Reprinted with permission from Rodriguez et al. [48])

early closure in that subset of patients who can aggressively be returned to normal hemodynamics and whose fascial edges have not retracted laterally, resulting in loss of abdominal domain. Early closure, especially within the first 2 weeks, prevents the difficult complications of fistula formation and nutritional support. In those select trauma patients who remain critically ill and whose fascia has retracted laterally, our algorithm (Fig. 13.3) highlights our approach to the reconstruction of the abdominal wall.

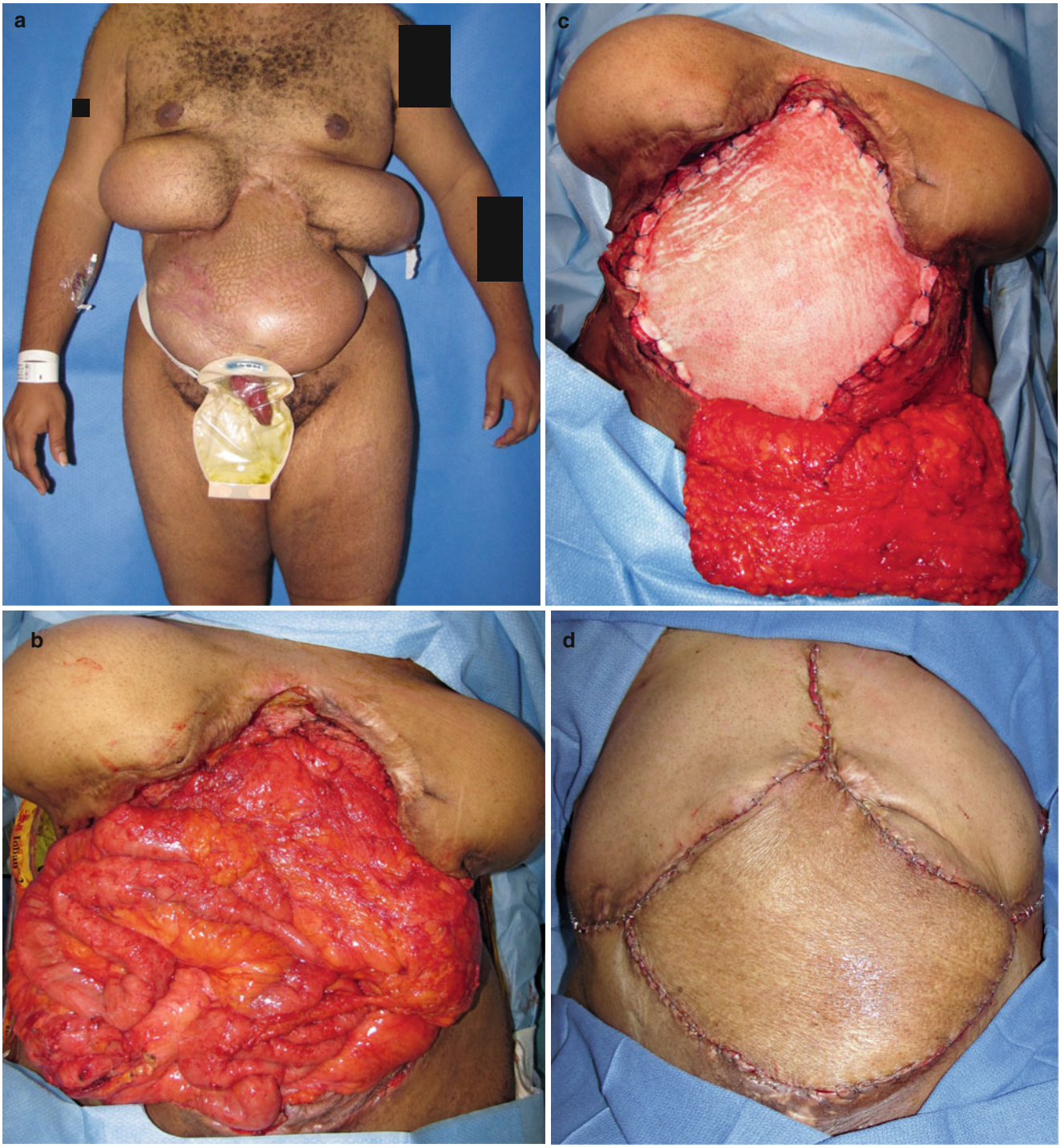


Fig. 13.9 (**a**) A 32-year-old man suffered a large ventral hernia following necrotizing fasciitis secondary to bowel perforation following a liposuction procedure. A staged procedure was required, including temporary closure with a skin graft followed by subsequent bilateral abdominal tissue expanders. (**b**) The definitive procedure began by elevating bilateral adipocutaneous flaps and complete removal of the split-thickness graft exposing the ventral defect in its entirety, which measured 40 × 30 cm. (**c**) A sheet of Strattice biologic mesh was placed into the hernia defect as an inlay. (**d**) After the pedicled ALT was passed medially to the rectus femoris muscle in combination with the upper abdominal repair, the flap was secured over the fascial reconstruction and approximated to the dermis. (**e**) Postoperatively, patient course was complicated by wound breakdown along the incision line at the juncture of the ALT flap and native tissue in the lateral right and left upper quadrants. Follow-up at 1 year after his initial reconstruction showed that the wounds healed with excellent take of the flap (**a**–**e**: Reprinted with permission from Maxhimer et al. [83])

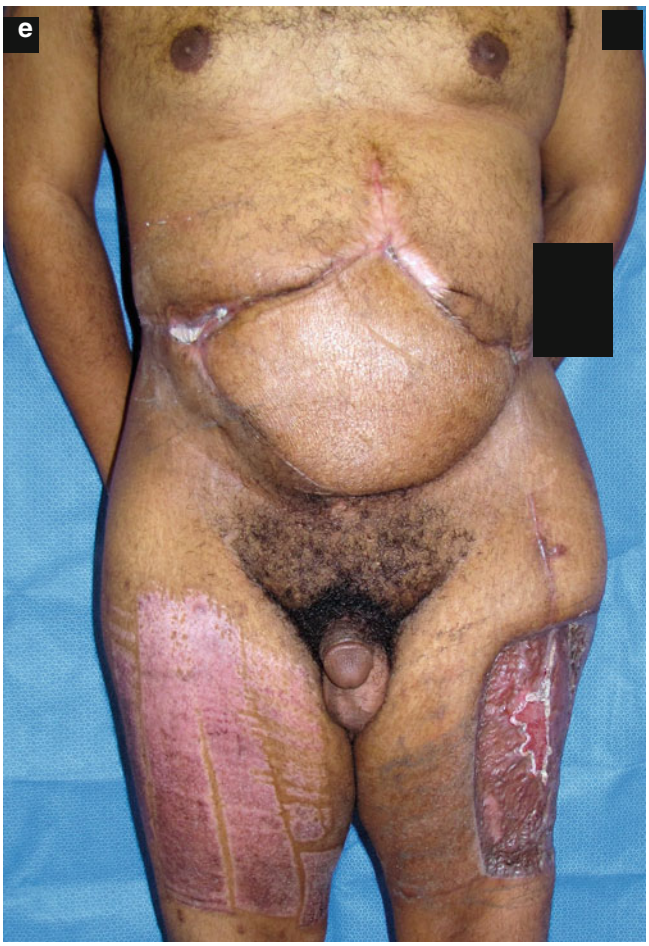


Fig. 13.9 (continued)

References

1. Rotondo MF, Schwab CW, McGonigal MD, et al. Damage control—an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma. 1993;35:375–82.
2. Rotonda MF, Zonies DH. The damage control sequence and underlying logic. Surg Clin North Am. 1997;77:761–77.
3. Hirshberg A, Mattox KL. Damage control in trauma surgery. Br J Surg. 1993;80(12):1501–2.
4. Feliciano DV, Mattox KL, Burch JM, et al. Packing for control of hepatic hemorrhage. J Trauma. 1986;26(8):738–43.
5. Asensio JA, McDuffie L, Petrone P. Reliable variables in the exsanguinated patient which indicate damage control and predict outcome. Am J Surg. 2001;182(6):743–51.
6. Hirshberg A, Mattox KL. Planned reoperation for severe trauma. Ann Surg. 1995;222:3–8.
7. Hirshberg A, Wall Jr MJ, Ramchandani MK, et al. Reoperation for bleeding in trauma. Arch Surg. 1993;128(10):1163–7.
8. Moore FA et al. Incommensurate oxygen consumption in response to maximal oxygen availability predicts postinjury multiple organ failure. J Trauma. 1992;33:58–67.
9. Holcomb JB, Jenkins D, Rhee P, et al. Damage control resuscitation: directly addressing the early coagulopathy of trauma. J Trauma. 2007;62:307–10.
10. Brasel KJ, Ku J, Baker CC, Rutherford EJ. Damage control in the critically ill and injured patient. New Horiz. 1999;7:73.
11. Kreimeier U, Pruecker S, Peter K. Permissive hypotension. J Suisse Med. 2000;130:1516–24.
12. Shoemaker WC, Appel PL, Kram HB, et al. Prospective trial of supranormal values of survivors as therapeutic goal in high risk surgical patients. Chest. 1988;94:1176–86.
13. An G, West M. Abdominal compartment syndrome: a concise clinical review. Crit Care Med. 2008;36(4):1304–10.
14. Ertel W, Oberholzer A, Platz A, et al. Incidence and clinical pattern of the abdominal compartment syndrome after "damage control" laparotomy in 311 patients with severe abdominal and/or pelvic trauma. Crit Care Med. 2000;28:1747–53.
15. Feliciano DV, Moore EE, Mattox KL. Trauma damage control. In: Moore EE, Feliciano DV, Mattox KL, editors. Trauma. 5th ed. New York: McGraw-Hill; 2004. p. 877–99.
16. Ferrada P, Murthi S, Anand J, et al. Transthoracic focused rapid echocardiographic examination: real-time evaluation of fluid status in critically ill trauma patients. J Trauma. 2011;70(1):56–62; discussion 62–4.
17. Broomé A, Hansson L, Lundgren F, et al. Open treatment of abdominal septic catastrophies. World J Surg. 1983;7(6):792–6.
18. Shapiro MB, Jenkins DH, Schwab CW, et al. Damage control: collective review. J Trauma. 2000;49(5):969–78.
19. Balogh Z, McKinley BA, Holcomb JB, et al. Both primary and secondary abdominal compartment syndrome can be predicted early and are harbingers of multiple organ failure. J Trauma. 2003; 54(5):848–59; discussion 859–61.
20. Ivatury RR, Nallathambi M, Rao PM, et al. Open management of the septic abdomen: therapeutic and prognostic considerations based on APACHE II. Crit Care Med. 1989;17(6):511–7.
21. Malhotra A. Low-tidal-volume ventilation in the acute respiratory distress syndrome. N Engl J Med. 2007;357(11):1113–20.
22. Habashi N. Other approaches to open-lung ventilation: airway pressure release ventilation. Crit Care Med. 2005;33(3):228–40.
23. Barker DE, Kaufman HJ, Smith LA, et al. Vacuum pack technique of temporary abdominal closure: 7-year experience with 112 patients. J Trauma. 2000;48(2):201–6; discussion 206–7.
24. Collier B, Guillamondegui O, Cotton B, et al. Feeding the open abdomen. JPEN J Parenter Enteral Nutr. 2007;31(5):410–5.
25. McClave SA, Martinsdale RG, Vanek VW. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition. JPEN J Parenter Enteral Nutr. 2009;33(3):277–316.
26. Teixeira PG, Salim A, Inaba K, et al. A prospective look at the current state of open abdomens. Am Surg. 2008;74(10):891–7.
27. Vogel TR, Diaz JJ, Miller RS, et al. The open abdomen in trauma: do infectious complications affect primary abdominal closure? Surg Infect. 2006;7(5):433–41.
28. Dutton WD, Diaz JJ, Miller RS. Critical care issues in managing complex open abdominal wound. J Intensive Care Med. 2011;1: 1–10.
29. Rutherford EJ, Skeete DA, Brasel KJ. Management of the patient with an open abdomen: techniques in temporary definitive closure. Curr Probl Surg. 2004;41:811–76.
30. Bender JS, Bailey CE, Saxe JM, et al. The technique of visceral packing: recommended management of difficult fascial closure in trauma patients. J Trauma. 1994;36(2):182–5.
31. Fernandez L, Norwood S, Roettger R, et al. Temporary intravenous bag silo closure in severe abdominal trauma. J Trauma. 1996;40(2): 258–60.
32. Fernandez L, Norwood S, Wilkins 3rd H, et al. Intraperitoneal silo: a form of temporary abdominal closure. Surg Rounds. 1999;22: 467–78.

33. Allen RG, Wrenn Jr EL. Silon as a sac in the treatment of omphalocele and gastroschisis. J Pediatr Surg. 1969;4(1):3–8.
34. Fox VJ, Miller J, Nix AM. Temporary abdominal closure using an i.v. bag silo for severe trauma. AORN J. 1999;69(3):530–5, 537, 539–41.
35. Cro C, George KJ, Donnelly J, et al. Vacuum assisted closure system in the management of enterocutaneous fistulae. Postgrad Med J. 2002;78(920):364–5.
36. Erdmann D, Drye C, Heller L, et al. Abdominal wall defect and enterocutaneous fistula treatment with the Vacuum-Assisted Closure (V.A.C.) system. Plast Reconstr Surg. 2001;108(7): 2066–8.
37. Stevens P. Vacuum-assisted closure of laparostomy wounds: a critical review of the literature. Int Wound J. 2009;6:259–266.
38. Garner GB, Ware DN, Cocanour CS, et al. Vacuum-assisted wound closure provides early fascial reapproximation in trauma patients with open abdomens. Am J Surg. 2001;182(6):630–8.
39. Koshima I, Soeda S. Inferior epigastric artery skin flaps without rectus abdominis muscle. Br J Plast Surg. 1989;42:645.
40. Sherck J, Seiver A, Shatney C, et al. Covering the "open abdomen": a better technique. Am Surg. 1998;64(9):854–9.
41. Bluebond-Langner R, Keifa ES, Mithani S, et al. Recurrent abdominal laxity following interpositional human acellular dermal matrix. Ann Plast Surg. 2008;60(1):76–80.
42. Scott BG, Feanny MA, Hirshberg A. Early definitive closure of the open abdomen: a quiet revolution. Scand J Surg. 2005;94:9.
43. Eppley B. Experimental assessment of the revascularization of acellular human dermis for soft-tissue augmentation. Plast Reconstr Surg. 2001;107:757.
44. Voyles CR, Richardson JD, Bland KI, et al. Emergency abdominal wall reconstruction with polypropylene mesh: short-term benefits versus long-term complications. Ann Surg. 1981;194:219.
45. Puente II, Sleeman D, et al. Management of fascial dehiscence in the critically ill. Med Intensiva. 1993;17:S62.
46. Fabian TC, Croce MA, Pritchard F, et al. Planned ventral hernia: staged management for acute abdominal defects. Ann Surg. 1994;219:643.
47. Saxe JM, Ledgerwood AM, Lucas CE. Management of the difficult abdominal closure. Surg Clin North Am. 1993;73:243–51.
48. Rodriguez ED, Bluebond-Langner R, Silverman RP, et al. Abdominal wall reconstruction following severe loss of domain. The R. Adams Cowley Shock Trauma Center algorithm. Plast Reconstr Surg. 2007;120:669–80.
49. Cothren CC, Moore EE, Johhnson JL, et al. One hundred percent fascial approximation with sequential abdominal closure of the abdomen. Am J Surg. 2006;192:238–42.
50. Weinberg JA, George RL, Griffin RL, et al. Closing the open abdomen: improved success with Wittman Patch staged closure. J Trauma. 2008;65:345–8.
51. Teichmann W, Wittmann DH, Andreone PA. Scheduled reoperations (etappenlavage) for diffuse peritonitis. Arch Surg. 1986;121(2): 147–52.
52. Wittmann DH, Aprahamian C, Bergstein JM. Etappenlavage: advanced diffuse peritonitis managed by planned multiple laparotomies utilizing zippers, slide fastener, and Velcro analogue for temporary abdominal closure. World J Surg. 1990;14(2): 218–26.
53. Wittmann DH, Aprahamian C, Bergstein JM, et al. A burr-like device to facilitate temporary abdominal closure in planned multiple laparotomies. Eur J Surg. 1993;159:75–9.
54. Boele van Hensbroek PB, Wind J, Dijkgraaf MG, et al. Temporary closure of the open abdomen: a systematic review on delayed primary fascial closure in patients with an open abdomen. World J Surg. 2009;33:199–207.
55. Miller PR, Meredith JW, Johnson JC, et al. Prospective evaluation of vacuum-assisted fascial closure after open abdomen: planned ventral hernia rate is substantially reduced. Ann Surg. 2004; 239:608–16.
56. Bleichrodt RP, de Vries Reilingh TS, Maylar A, et al. Component separation technique to repair large midline hernias. Oper Tech Gen Surg. 2004;6:179–88.
57. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86:519.
58. Williams JK, Carlson GW, de Chalain T, et al. Role of tensor fasciae latae in abdominal wall reconstruction. Plast Reconstr Surg. 1998;101:713–8.
59. Sukkar SM, Dumanian GA, Szczerba SM, et al. Challenging abdominal wall defects. Am J Surg. 2001;181:115.
60. Luijendijk RW, Hop WC, van den Tol MP, et al. A comparison of suture repair with mesh repair for incisional hernia. N Engl J Med. 2000;343:392.
61. Tobias AM, Low DW. The use of subfascial Vicryl mesh buttress to aid in the closure of massive ventral hernias following damage control laparotomy. Plast Reconstr Surg. 2003;112:766.
62. Menon NF, Rodriguez ED, Byrnes CK, et al. Revascularization of human acellular dermis in full thickness abdominal wall reconstruction in the rabbit model. Ann Plast Surg. 2003;50:523.
63. Butler CE, Prieto VG. Reduction of adhesions with composite AlloDerm/polypropylene mesh implants for abdominal wall reconstruction. Plast Reconstr Surg. 2004;114:464.
64. Leber GE, Garb JL, Alexander AI, et al. Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg. 1998;133:378.
65. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal re-exploration. Ann Surg. 1984;199(1):28–30.
66. Lacey SR, Bruce J, Brooks SP, et al. The relative merits of various methods of indirect measurement of intraabdominal pressure as a guide to closure of abdominal wall defects. J Pediatr Surg. 1987;22(12):1207–11.
67. Mayberry JC, Mullins RJ, Crass RA, et al. Prevention of abdominal compartment syndrome by absorbable mesh prosthesis closure. Arch Surg. 1997;132(9):957–61; discussion 961–2.
68. Saggi BH, Sugerman HJ, Ivatury RR, et al. Abdominal compartment syndrome. J Trauma. 1998;45(3):597–609.
69. Schein M, Wittmann DH, Aprahamian CC, et al. The abdominal compartment syndrome: the physiological and clinical consequences of elevated intra-abdominal pressure. J Am Coll Surg. 1995;180(6):745–53.
70. Nagy KK, Fildes JJ, Mahr C, et al. Experience with three prosthetic materials in temporary abdominal wall closure. Am Surg. 1996;62:331.
71. Scott BG, Welsh FJ, Pham HQ, et al. Early aggressive closure of the open abdomen. J Trauma. 2006;60:17.
72. Morris Jr JA, Eddy VA, Blinman TA, et al. The staged celiotomy for trauma. Issues in unpacking and reconstruction. Ann Surg. 1993;21(5):576–84. discussion 584–6.
73. Silverman RP, Li EN, Holton LH, et al. Ventral hernia repair using allogenic acellular dermal matrix. Hernia. 2004;8:336.
74. Rohrich RJ, Lowe JB, Hackney FL, et al. An algorithm for abdominal wall reconstruction. Plast Reconstr Surg. 2000;105:202.
75. Fansler RF, Taheri P, Cullinane C, et al. Polypropylene mesh closure of the complicated abdominal wound. Am J Surg. 1995; 170:15.
76. Morris-Stiff GJ, Hughes LE. The outcomes of non-absorbable mesh placed within the abdominal cavity: literature review and clinical experience. J Am Coll Surg. 1998;186:352.
77. Scripcariu V, Carlson G, Bancewicz J, et al. Reconstructive abdominal operations after laparostomy and multiple repeat laparotomies for severe intra-abdominal infection. Br J Surg. 1994;81(10): 1475–8.

78. Stone HH, Fabian TC, Turkleson ML, et al. Management of acute full-thickness losses of the abdominal wall. Ann Surg. 1981; 193(5):612–8.
79. Tyrell J, Silberman H, Chandrasoma P, et al. Absorbable versus permanent mesh in abdominal operations. Surg Gynecol Obstet. 1989;168(3):227–32.
80. Jacobsen WM, Petty PM, Bite U, et al. Massive abdominal-wall hernia reconstruction with expanded external/internal oblique and transversalis musculofascia. Plast Reconstr Surg. 1997; 100:326.
81. Bloomfield GL, Ridings PC, Blocher CR, et al. Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion. J Trauma. 1996;40(6): 936–41; discussion 941–3.
82. Hirshowitz B, Linden Baum E, Har-Shai Y. A skin-stretching device for harnessing of the visco-elastic properties of skin. Plast Reconstr Surg. 1994;92:260–70.
83. Maxhimer JB, Hui-Chou HG, Rodriguez ED. Clinical applications of the pedicled anterolateral thigh flap in complex abdominal-pelvic reconstruction. Ann Plast Surg. 2011;66(3): 285–91.
84. Classen D. The extended deep inferior epigastric flap: a case series. Ann Plast Surg. 1999;42:137.
85. Rodriguez ED, Rosson GD, Bluebond-Langner R, et al. The utility of the anterolateral thigh donor site in reconstructing the United States trauma patient. J Trauma. 2007;62:892.
86. Robinson TN, Clarke JH, Walsh MD. Major mesh related complications following hernia repair: events reported to the Food and Drug Administration. Surg Endosc. 2005;19:1556.
87. Paul A, Korenkov M, Peters S, et al. Unacceptable results of the Mayo procedure for repair of abdominal incisional hernias. Eur J Surg. 1998;164:361.
88. Read RC. Repair of incisional hernia. Curr Surg. 1990;47:278.
89. George CD, Ellis H. The results of incisional hernia repair: a twelve-year review. Ann R Coll Surg Engl. 1986;68:185.
90. Mayberry JC, Goldman RK, Mullins RJ, et al. Surveyed opinion of American trauma surgeons on the prevention of the abdominal compartment syndrome. J Trauma. 1999;47:509–13.

14 Complex Tissue Transfer in the Management of Abdominal Wall Defects

Shigeki Kushimoto

Introduction

The concepts of damage control and improved understanding of the pathophysiology of abdominal compartment syndrome have proven to be great advances in trauma care [1–3]. Furthermore, these insights have been incorporated into the care of nontraumatic surgical conditions [4, 5]. Massive fluid resuscitation for hemorrhagic and septic shock results in significant tissue edema, which does not spare the bowel. The consequent visceral edema can preclude abdominal wall closure after laparotomy because the fascia cannot be reapproximated without excessive tension. Abdominal wall closure under excessive tension often leads to abdominal compartment syndrome and fascial necrosis. Clear recognition of these complications has led to the widespread practice of leaving the abdominal cavity open after either damage control surgery or decompressive laparotomy for abdominal compartment syndrome. However, these approaches require prolonged open abdomen management. During this interval, the musculofascial structure of the abdominal wall contracts laterally, leaving patients with a large midline defect if standard fascial closure is not possible. Although abdominal wall defects result from multiple etiologies, including trauma, previous abdominal surgeries, congenital abnormalities, and infection [6], the concept of leaving the abdominal cavity open after damage control and abdominal compartment syndrome as a therapeutic strategy has markedly contributed to the increased frequency of abdominal wall defects.

S. Kushimoto, MD, PhD
Division of Emergency Medicine,
Tohoku University Graduate School of Medicine,
1-1 Seiryo-machi, Aoba-ku, Sendai 980-8574, Japan
e-mail: kussie@emergency-medicine.med.tohoku.ac.jp

Temporary Abdominal Wall Closure for Acute Abdominal Wall Defect and During Open Abdomen Management

To reduce the need for an intermediate period with a large ventral hernia requiring later abdominal wall reconstruction, several techniques, such as vacuum-assisted wound closure and application of a Wittmann Patch® (Starsurgical, Burlington, WI), have been employed [7–9]. Recently, several studies have shown that delayed abdominal fascial closure is safe and effective for achieving successful closure in 65–100% of patients with an open abdomen [10–12]. There is evidence that vacuum-assisted closure devices facilitate delayed primary fascial closure, with high success rates and low morbidity [8, 12–14] by both commercially available devices (V.A.C.® Therapy, KCI, San Antonio, TX) and noncommercial "vacuum-packed dressing," although the effectiveness of vacuum-assisted closure devices to achieve delayed fascial closure in patients with abdominal sepsis has not been as high as in trauma patients [15]. In the setting of ongoing intraabdominal infection or the formation of an enterocutaneous fistula, abdominal fascial closure is often not possible [16], because of ongoing visceral edema, with loss of the abdominal domain or loss of fascia secondary to infection. Although Miller et al. demonstrated that early abdominal fascial closure can be achieved in the majority (63%) of damage control cases during the initial relaparotomy, delayed abdominal fascial closure before 8 days was associated with fewer complications (with rates of 12% in those closed before 8 days and 52% with closure after 8 days), suggesting that early fascial closure might be crucial for minimizing complications associated with open abdomen/abdominal wall defects [10].

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_14,

Abdominal Wall Reconstruction Following Temporary Closure in the Management of Abdominal Wall Defects

Surgical options available for abdominal wall defects, if primary suture is not possible, are limited to (1) bridge repair of the fascial defect using a mesh to create a bridge closure; (2) acute abdominal wall reconstruction, most commonly using component separation and its modifications; or (3) a planned ventral hernia [12]. Although acute abdominal wall reconstruction using tissue transfer techniques has been reported [12, 17, 18], in the typical care of patients requiring open abdomen management who are not candidates for early standard fascial closure, many still require a period with a large ventral hernia in which granulated abdominal contents are covered with only a skin graft, necessitating subsequent complex abdominal wall reconstruction. Moreover, the risk of enterocutaneous fistula may increase as the duration of open abdomen management is prolonged [19].

To accomplish late reconstruction of the abdominal wall for patients after a period with a planned ventral hernia following open abdomen management, several tissue transfer methods have been proposed, such as component separation [20], rectus turnover flap [21], and modified component separation techniques [19, 22]. Although these methods have been reported for abdominal wall reconstruction at 6–12 months or even later after the initial operation, application of these techniques in the early phase of the open abdomen has not been adequately evaluated. Even the techniques used in abdominal wall reconstructions are constantly changing, the goals of treatment remain the same: protection of abdominal contents and restoration of functional support. Vascularized autologous tissue repair is extremely useful in cases at high risk of infection, such as those with abdominal sepsis and those requiring prolonged open abdomen management.

Complex Tissue Transfer in the Management of Abdominal Wall Defects

Although several flap techniques for abdominal wall reconstruction have been demonstrated, including free tensor fascia lata flap, anterolateral thigh flap, latissimus dorsi muscle free flap, and rectus femoris musculocutaneous free flap [23], here we focus on the component separation technique, including its modifications and the anterior rectus abdominis sheath turnover flap method of complex tissue transfer.

Basic Musculoskeletal and Neurovascular Anatomy of Anterior Abdominal Wall

The anterior abdominal wall consists of paired rectus and oblique muscles that coalesce in the midline to create a myofascial sling that resists internal pressure, provides a stable platform for movement and assistance with respiratory excursion. Flexion of the abdominal wall is mainly facilitated by the midline rectus abdominis muscles, with their origin at the pubic symphysis and the insertion at the xiphoid process and the fifth to seventh costal cartilages. Lateral support of the abdomen is provided by three layers: external oblique, internal oblique, and transverse abdominis muscles. These muscles interdigitate toward the midline bilaterally to form the anterior and posterior rectus sheaths, with their corresponding medial insertions into the linea alba. Above the arcuate line, the aponeuroses of these muscles divide, with the external oblique providing fibers to the anterior rectus sheath, the transversalis muscle donating its fibers posteriorly, and the internal oblique splitting to contribute fibers to both the anterior and the posterior sheath. However, below the arcuate line, all three aponeuroses run anterior to the rectus muscle, with only the transversalis fascia providing posterior support.

A neurovascular plane exists within the anterolateral abdominal wall, traversing between the internal oblique and transversalis muscles. Coursing within this plane is the innervation to the oblique and rectus muscles, provided by the inferior six thoracic nerves (T7–T11 and the subcostal nerve T12), and the iliohypogastric and ilioinguinal nerve branches of L1. Huger classified the vascular supply of the anterolateral abdomen into three zones [24]. Zones I and II, the midabdomen and lower abdomen, respectively, are supplied by the vascular arcade of the superior and inferior deep epigastric arteries, with contributions from the superficial inferior epigastric, superficial circumflex iliac, and deep circumflex iliac arteries to the lower abdominal wall. Laterally, in zone III, the intercostal, subcostal, and lumbar arteries course toward the midline with their corresponding nerve branches. This anterolateral configuration allows for a relatively avascular and nerve-sparing plane to exist between the external and internal oblique muscles on either side of the midline, which is the site of muscle splitting for the component separation method as described in the next section.

Component Separation Method

Ramirez et al. popularized the component separation technique for reconstruction of large abdominal wall fascial defects without the use of prosthetic mesh [20]. In its basic form, the technique is as follows (Fig. 14.1a–f):

1. Anterior abdominal wall skin flaps are developed and dissected to the anterior superior iliac spine and the chest wall. The procedure is initiated by elevating the skin and subcutaneous flaps off the underlying abdominal musculature in a lateral direction toward the anterior axillary line to explore the anterior surface of the external oblique aponeurosis 2–3 cm lateral to the linea semilunaris. The linea semilunaris is dissected along with the insertion of the external oblique fascia.

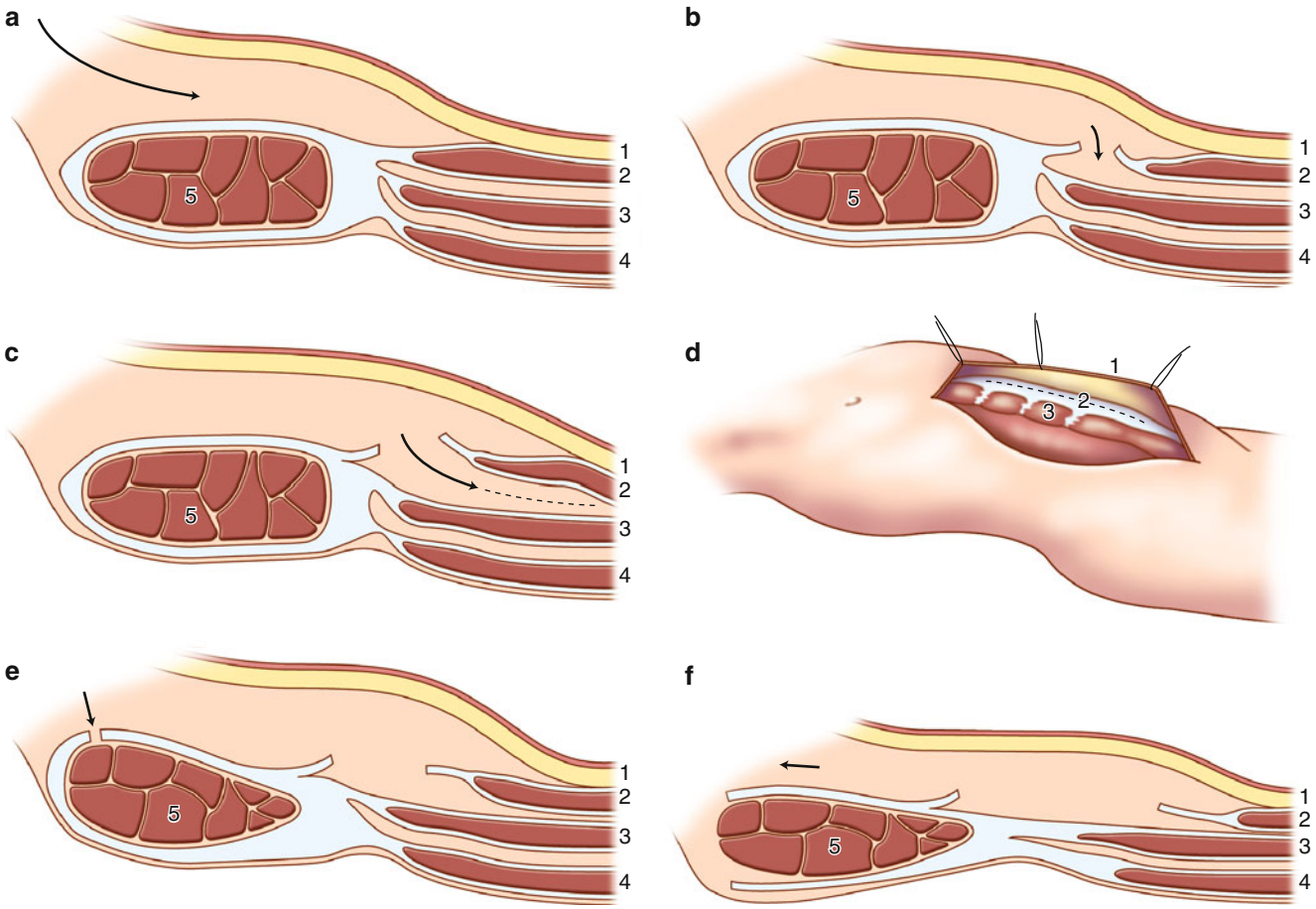


Fig. 14.1 The component separation technique. After abdominal cavity entry, the bowels are dissected free from the ventral abdominal wall. (**a**) The skin and subcutaneous fat (*1*) are dissected free from the anterior sheath of the rectus abdominis muscle (*5*) and the aponeurosis of the external oblique muscle (*2*). (**b**, **c**) The aponeurosis of the external oblique muscle (*2*) is transected longitudinally about 2 cm lateral to the rectus sheath, including the muscular part on the thoracic wall, which extends at least 5–7 cm cranially from the costal margin. (**d**) The external oblique muscle (*2*) is separated from the internal oblique muscle (*3*) as far as possible laterally. (**e**, **f**) If primary closure is impossible because of tension, a further gain of 2–4 cm can be obtained by separation of the posterior rectal sheath from the rectus abdominis muscle (*5*). The rectus muscle and the anterior rectal sheath can be advanced to the midline over a distance of about 10 cm at the waistline. Care must be taken not to damage the blood vessels and nerves that run between the internal oblique and transverse (*4*) muscles and enter the rectus abdominis muscle at the posterior side (Adapted with permission of Elsevier from de Vries Reilingh et al. [25])

2. The aponeurosis of the external oblique muscle is divided lateral to the semilunar line on to the chest wall to the level of the xiphoid. A vertically oriented incision parallel to the linea semilunaris is made 2–3 cm lateral to it, extending from the inguinal ligament to the level of the costal margin and above it. This superior extension is important in cases with defects extending up to the xiphoid process to obtain adequate release of tissues for these superior closures. The incision should be made well lateral to the linea semilunaris, just medial to the musculofascial junction of the external oblique muscle itself. Figure 14.1 is a diagrammatic illustration showing elevation of the skin flap laterally and development of the plane between the external oblique and internal oblique muscles. This plane was opened all the way to the posterior axillary line.
3. Free the external oblique to allow the rectus myofascial component to be mobilized medially. After division of the external oblique fascia, the deep surface of the external oblique muscle is identified, and the plane between the external and internal oblique muscles is developed. When making the initial incision in the oblique fascia, the surgeon must be careful not to dissect deep into this layer of the external oblique fascia to avoid injuring the internal oblique fascia or muscle.
4. The midline is sutured together.

Degree of Tissue Advancement at Various Locations on the Abdominal Wall for the Innervated Rectus Abdominis, Internal Oblique, Transversus Abdominis Muscle Complex

The dissection proceeds in this relatively avascular intermuscular plane and is continued in a lateral direction to at least the level of the midaxillary line. At this point, the mobility of the innervated rectus abdominis–internal oblique–transversus

abdominis muscle complex is determined. If additional mobility of these structures on either side of the midline is desired, then the dissection in the intermuscular plane can be continued to the posterior axillary line. Each ipsilateral complex can be expected to advance toward the midline 4 cm in the upper abdomen, 8 cm at the umbilicus, and 3 cm in the lower abdomen.

Modifications of the Component Separation Method

The original component separation method has several disadvantages, as suggested previously. Mass et al. described three disadvantages [26]. First, the skin and subcutaneous tissue must be mobilized laterally over a large distance to reach the aponeurosis of the external oblique muscle lateral into the flank. This creates a large wound surface that covers the entire abdominal wall, from costal margin to pubic bone. Second, mobilization of the skin endangers its blood supply, which may lead to skin necrosis at the midline if circulation through the intercostal arteries is interrupted. Third, the technique is difficult to use in patients with an enterostomy or when a new enterostomy must be made.

The purposes of modifying the original component separation method are as follows: (1) additional advancement of components; (2) preservation of the blood supply to the skin and subcutaneous tissue; (3) overcoming the problem of stoma reconstruction; and (4) reduction of the subcutaneous tissue mobilization area. The first and second goals are especially important.

For additional advancement of components to the midline, separation of the rectus muscle from the posterior rectal sheath has been used in almost all reported techniques [19, 26–29]. With this modification, the rectus muscle and the anterior rectal sheath can be expected to advance to the midline over a distance of about 10 cm at the level of the umbilicus (Figs. 14.1a–f and 14.2a, b).

Maas and colleagues described a modification of the original technique of component separation, designed to preserve the blood supply to the skin and subcutaneous tissue and to overcome the problem of stoma reconstruction in these patients [26]. Using their technical modification, the aponeurosis of the external oblique muscle is dissected free through a separate, longitudinal skin incision at a distance of about 15 cm from the median skin border (Fig. 14.2a, b). The aponeurosis is transected just lateral to its insertion in the rectal sheath, from the costal margin to 5 cm above the pubic bone. The external oblique muscle is separated from the internal oblique muscle. A well-vascularized compound flap is created and can be advanced to the midline. The rectus muscle is separated from the posterior sheath to further mobilize this flap.

Component separation has become the most commonly used surgical technique for closure of large "planned" ventral

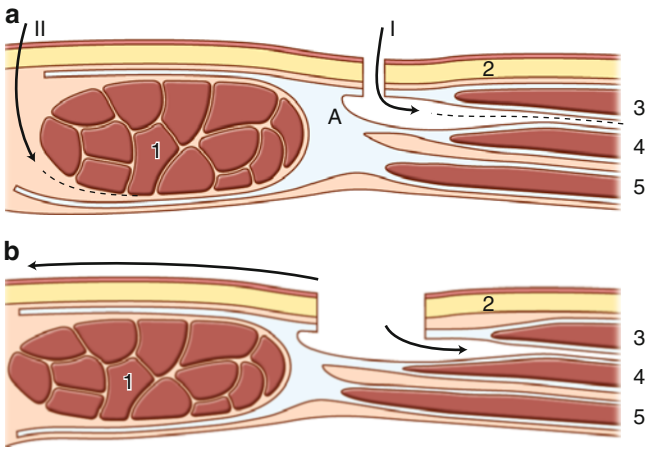


Fig. 14.2 Modified component separation technique. (**a**) I, the external oblique muscle is transected through a separate incision, just lateral to the rectal sheath; II, separation of the rectus abdominis muscle from the posterior rectal sheath. (**b**) The compound flap can be advanced to the midline. The skin is vascularized through the perforating branches of the epigastric arteries. *1* Rectus abdominis muscle; *2* skin and subcutaneous tissue; *3* external oblique muscle; *4* internal oblique muscle; *5* transverse muscle (Adapted with permission of Elsevier from Maas et al. [26])

hernias covered with a skin graft during the elective reconstruction phase [30–32]. Its use for acute definitive closure in the setting of an open abdomen has not been fully evaluated. Formal component separation is generally considered to be an "elective" reconstruction technique. Although its use in the acute setting aimed for resolving intra-abdominal sepsis, visceral and abdominal wall edema as a result of systemic inflammatory responses, and ongoing sepsis has not yet been recommended [12], early definitive abdominal wall closure can reduce the need for skin grafting and later abdominal wall reconstruction and may decrease risks associated with open abdomen/abdominal wall defects, especially enteric fistula.

Anterior Rectus Abdominis Sheath Turnover Flap Method

We recently demonstrated the usefulness of this method for early fascial closure in patients requiring open abdomen management [17]. This technique may reduce the need for skin grafting and later abdominal wall reconstruction. It can also be used for later reconstruction, as previously reported [21].

During open abdomen management, care must be taken to prevent damage to the fascia, including the linea alba, to allow a definitive turnover flap of the anterior rectus sheath. If the abdominal fascia could be fully approximated without tension, standard fascial closure was performed. At 10–14 days after the initial laparotomy, a turnover flap of the anterior rectus abdominis sheath was considered instead if

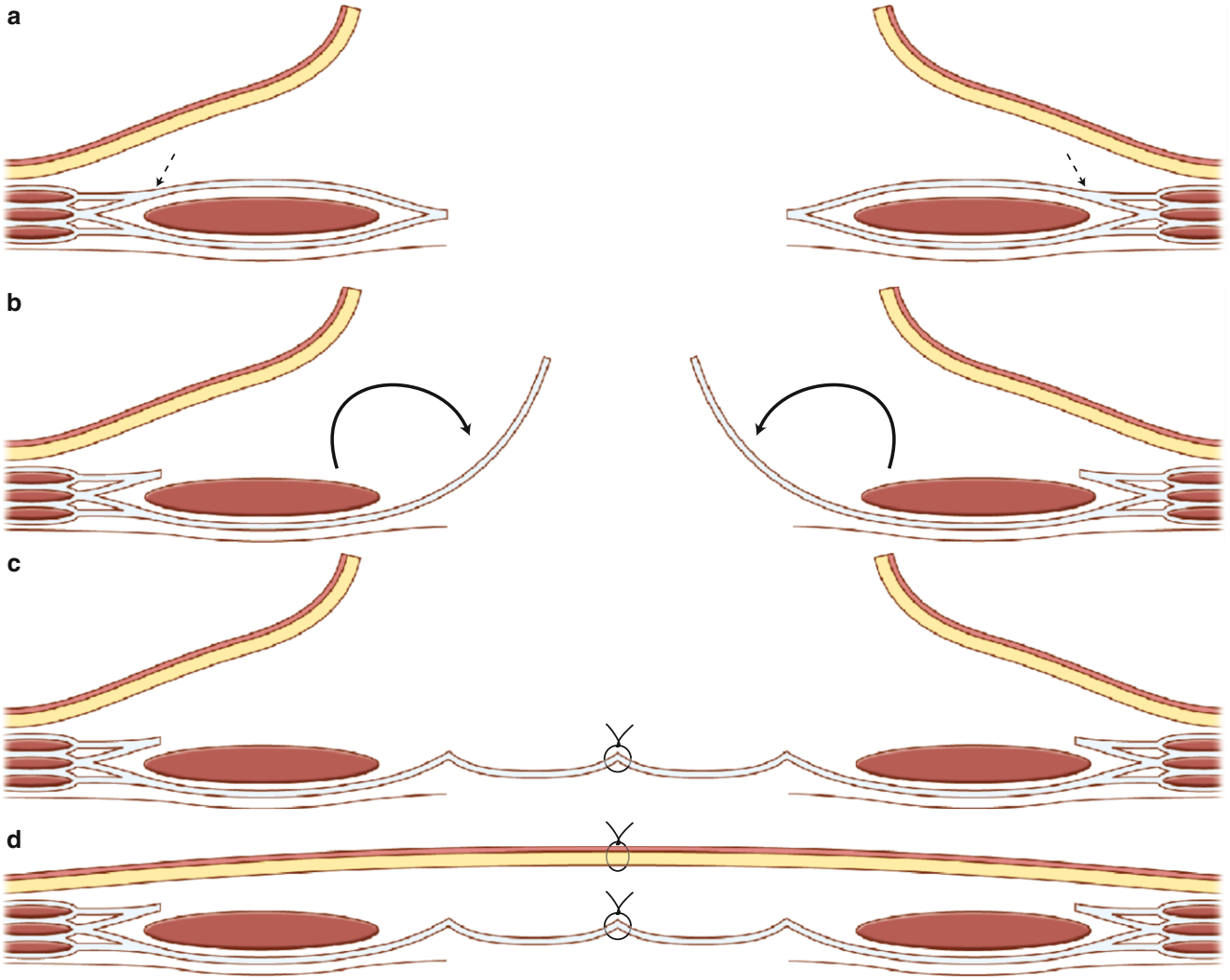


Fig. 14.3 Cross-sectional schematic diagram of the technique for turnover flap creation from the anterior rectus abdominis sheath. The procedure is started by separating the skin and underlying adipose tissue from the anterior rectus sheath as a flap, with a base several centimeters beyond the lateral border of the rectus sheath (**a**). The turnover flap is then fashioned from the anterior sheath by longitudinally incising the sheath along the entire length of its lateral border. The site of this incision must be chosen carefully to avoid entry at the conjoined point of the internal oblique aponeurosis and the external oblique aponeurosis (**b**). The anterior sheath is then dissected laterally to medially, freeing it from the rectus muscle. The linea alba is kept intact to serve as a medial hinge. The turnover flap of the anterior rectus sheath is approximated by interrupted sutures (**c**), and the skin is closed primarily (**d**) (Adapted with kind permission of Springer from Kushimoto et al. [17])

the distance to be closed with fascia was less than 15 cm in patients who were not candidates for standard fascial closure because of prolonged visceral edema. Formation of a planned ventral hernia using a skin graft over granulated abdominal contents was employed in patients without edema resolution 3 weeks or more after the initial laparotomy who were not candidates for either method of fascial closure.

Surgical Procedure

The surgical procedure starts with separation of the skin and underlying adipose tissue from the anterior rectus sheath as a flap, with a base several centimeters beyond the lateral border of the rectus sheath. Next, turnover flap creation from the anterior sheath is initiated by incising the anterior sheath along the entire length of its lateral border. When making this longitudinal incision, the specific incision site must be chosen carefully to avoid entry at the conjoined point of the internal and external oblique aponeuroses, which could weaken the anterior sheath and predispose the patient to subsequent hernia formation. Because the largest fascial gap is in the midabdomen, where a wide flap is needed to approximate the fascia in most patients, longitudinal incision of the anterior rectus sheath should be started at the upper or lower abdominal surface of the anterior sheath to avoid entry at the conjoined point. The anterior sheath is then dissected laterally to medially, freeing it from the rectus muscle. Kept intact, the linea alba serves as a medial hinge to mobilize the flap (Fig. 14.3a–d). If the linea alba is no longer intact, suture

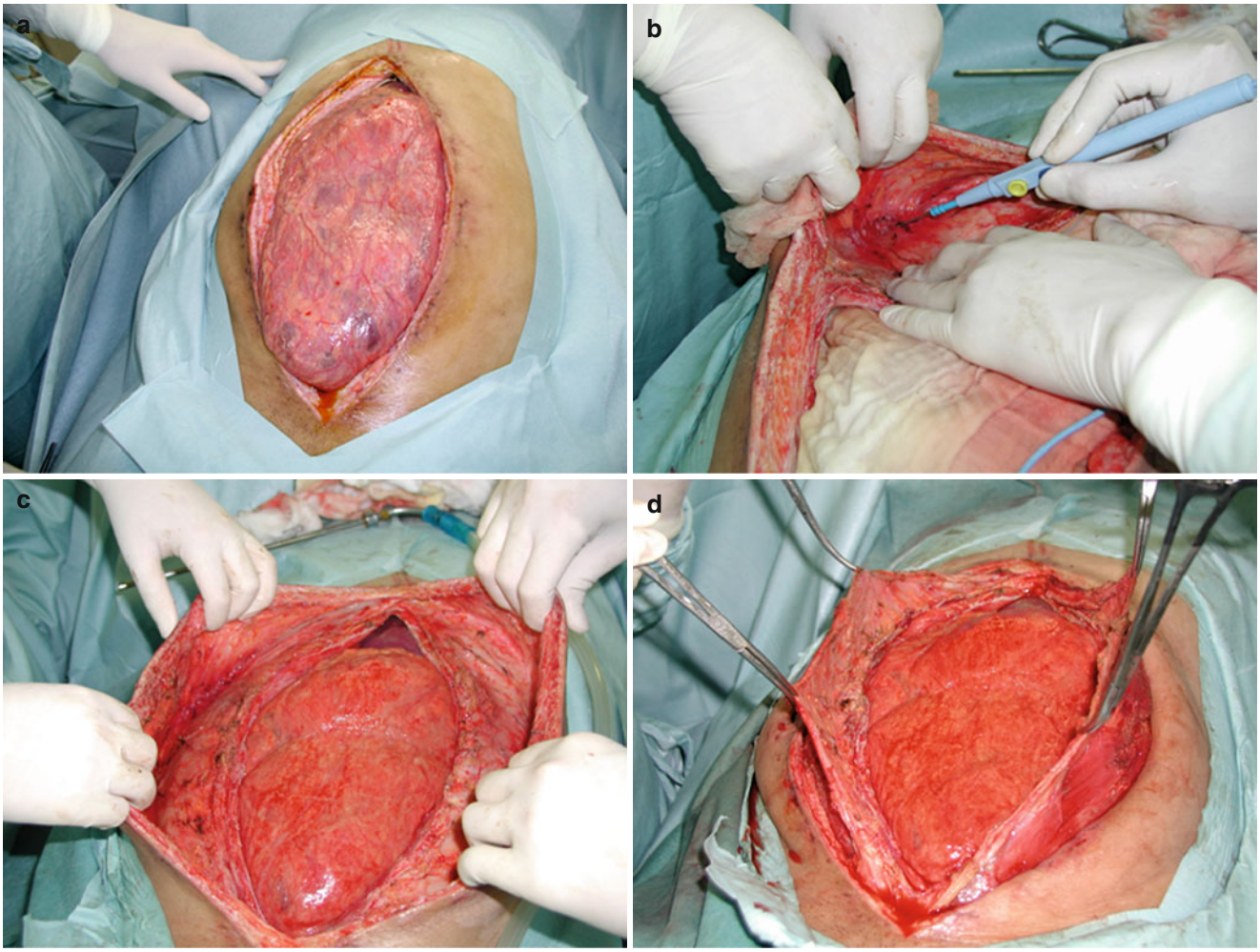


Fig. 14.4 Intraoperative view of the anterior rectus abdominis sheath turnover flap method (initial steps). (**a**) View just after vacuum packing removal (11 days of open abdomen). (**b**) Skin and underlying adipose tissue are first separated from the anterior rectus sheath as a flap. (**c**) Skin and adipose tissue have been completely dissected from the anterior sheath bilaterally beyond the lateral border of the rectus sheath. (**d**) The anterior rectus sheath flap is reflected medially by dissecting laterally to medially, freeing it from the rectus muscle. The linea alba is kept intact as a medial hinge (Reprinted with kind permission of Springer from Kushimoto et al. [17])

repair must be performed. The fascial flap is then reflected medially, with careful attention not to damage the anterior sheath.

After creating bilateral turnover flaps, we approximate the flaps to cover the abdominal contents using interrupted sutures (3–0 polyglactin 910). We never use prosthetic materials to reinforce the turnover flaps or to repair exceptionally large fascial defects. Thereafter, the skin and underlying adipose tissue are approximated with drainage to the base of the adipose tissue dissection (Figs. 14.4a–d, 14.5a–d, 14.6a–d, and 14.7a–d).

Blood Supply to the Anterior Rectus Turnover Flap

Blood supply to the anterior rectus turnover flap is an issue awaiting clarification. Ennis et al. stated that "the flap is vascularized autogenous tissue"; "small anterior venules at the medial portion of the flap" were described as constituting a major vascular element of the flap in Ochsner's comment at a conference discussion session [22]. However, the blood supply to the anterior rectus sheath has been suggested to arise primarily from perforating intramuscular branches of the deep superior and inferior epigastric arteries [33–35], and some of these perforators to the anterior fascia are inevitably transected

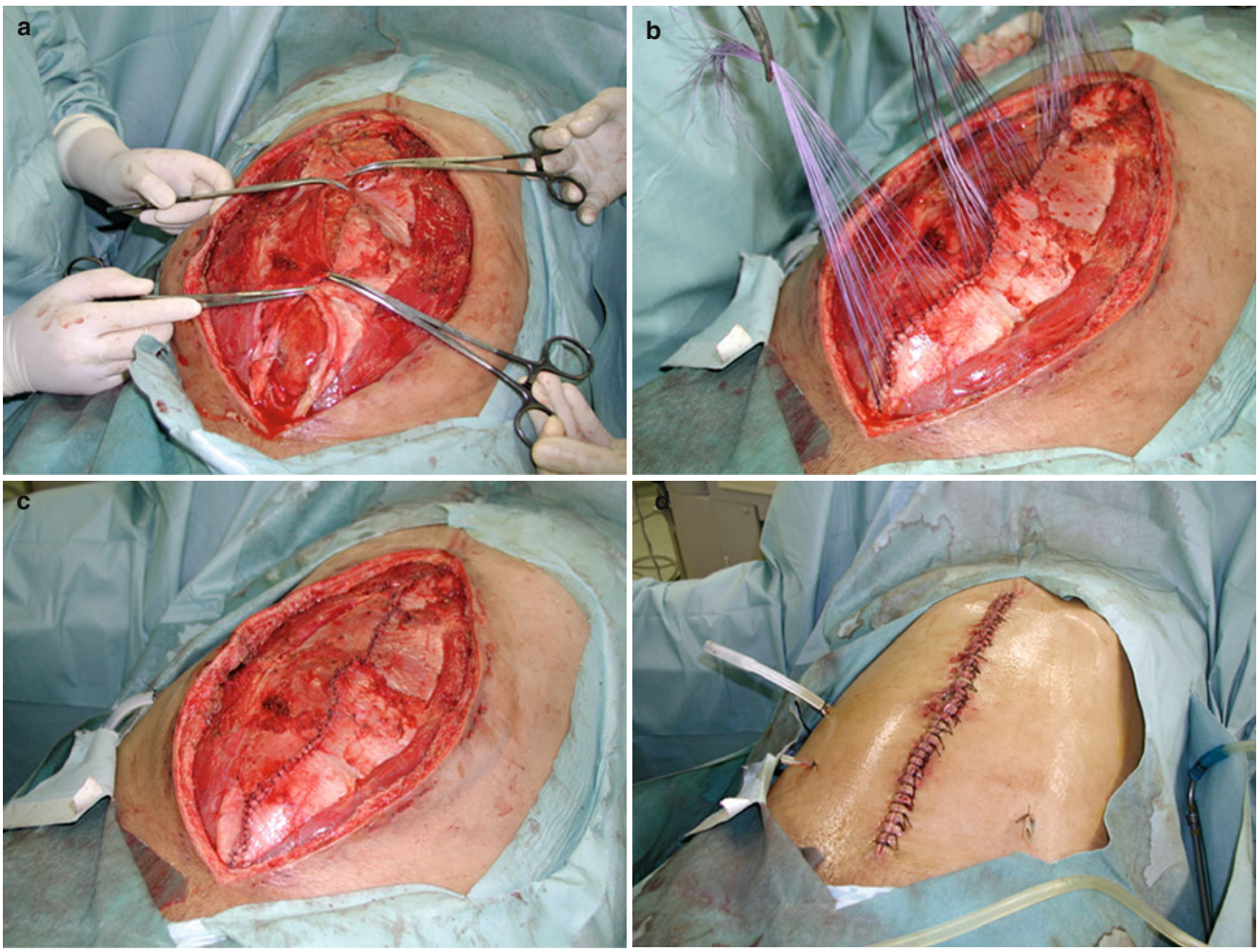


Fig. 14.5 Intraoperative view of the anterior rectus abdominis sheath turnover flap method (later steps). (**a**) Approximating the bilateral turnover flaps. (**b** and **c**) Turnover flaps from the anterior rectus sheaths are approximated by interrupted sutures. (**d**) Skin and subcutaneous tissue are sutured primarily (Reprinted with kind permission of Springer from Kushimoto et al. [17])

during reflection of the anterior rectus sheath flap as it is freed from the rectus muscle. Numerous small arteries on the anterior surface of the anterior rectus sheath may be supplied by branches of deep epigastric arteries along the linea alba, complementing the blood supply to the anterior rectus sheath [33]. Although the blood supply to the anterior rectus sheath turnover flap remains uncertain, we observed the flap to be completely intact even in a patient with major wound infection whose entire midline skin closure had dehisced.

In our series, the duration of open abdomen was 17.6 ± 24.6 days for all study patients. Twelve of 18 nontrauma patients survived, as did 8 of 11 trauma patients. Turnover flap closure was performed in 9 nontrauma patients (1–31 days after the initial surgery [9.4 ± 9.2 days]). Among trauma patients, turnover flap closure was used at 6 days in one and at 30 days in another. None of our patients developed enterocutaneous fistula or abdominal abscess. Although midabdominal bulging is observed in more than half of patients with anterior rectus abdominis sheath turnover flap closure, no abdominal wall hernias requiring secondary reconstruction developed during follow-up periods of up to 65 months.

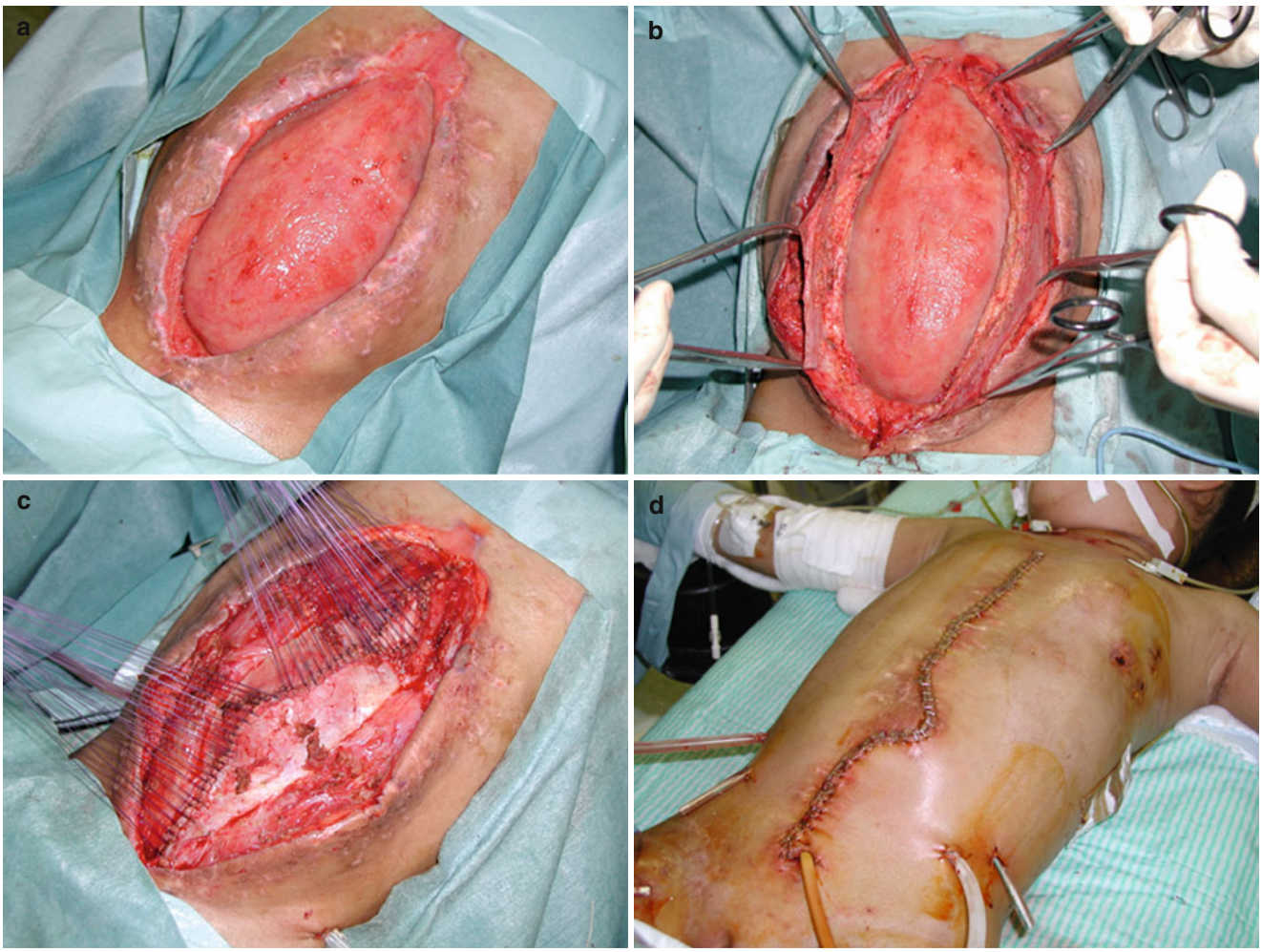


Fig. 14.6 Intraoperative view of the turnover flap method using the anterior rectus abdominis sheath carried out 30 days after initial laparotomy. (**a**) View just after vacuum packing removal (30 days of open abdomen) showing granulated abdominal contents and retracted musculofascial structures of the anterior abdomen. (**b**) The anterior rectus sheath flap is reflected medially, dissecting laterally to medially to free it from the rectus muscle. (**c**) Bilateral turnover flaps from the anterior rectus sheaths are approximated using interrupted sutures. (**d**) Skin and subcutaneous tissue are sutured primarily (Reprinted with kind permission of Springer from Kushimoto et al. [17])

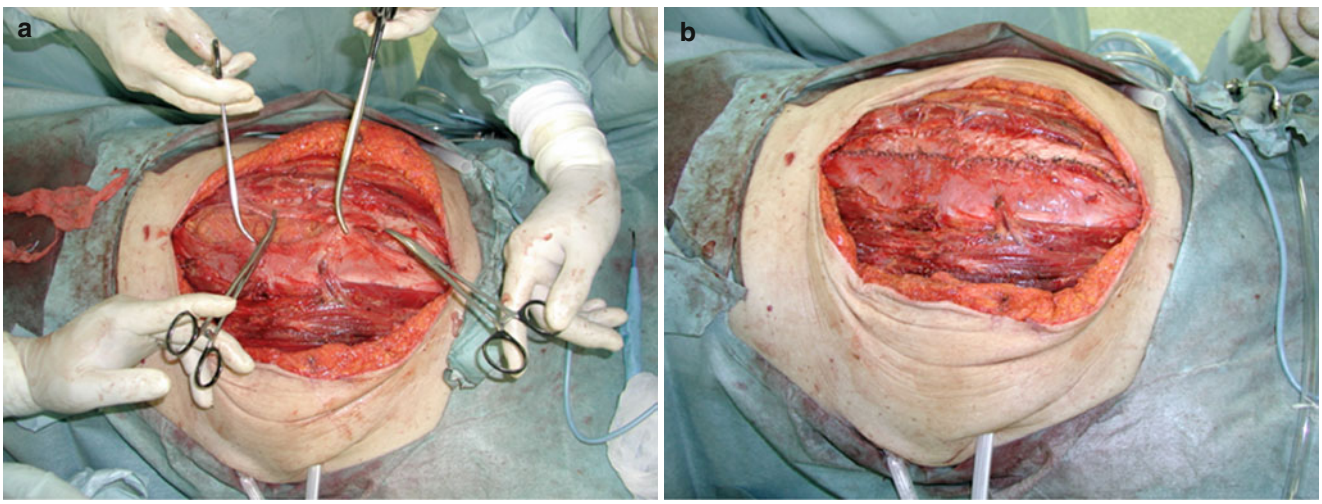


Fig. 14.7 Intraoperative view of the turnover flap created by the anterior rectus abdominis sheath method in a patient with major infection. (**a**) After 2 days of open abdomen, the anterior rectus sheath flap is reflected medially by dissecting laterally to medially, freeing it from the rectus muscle. (**b**) Bilateral turnover flaps of the anterior rectus sheath are approximated by interrupted sutures. (**c**) Major wound infection was evident on postoperative day 5. All skin over the wound has dehisced, but the fascial flap remained intact. This photograph shows the fascial flap covered with granulation extending from the lateral side on postoperative day 20. The skin was closed secondarily. (**d**) View of the anterior abdominal wall in the sitting position (postoperative day 60 after fascial closure) (Reprinted with kind permission of Springer from Kushimoto et al. [17])

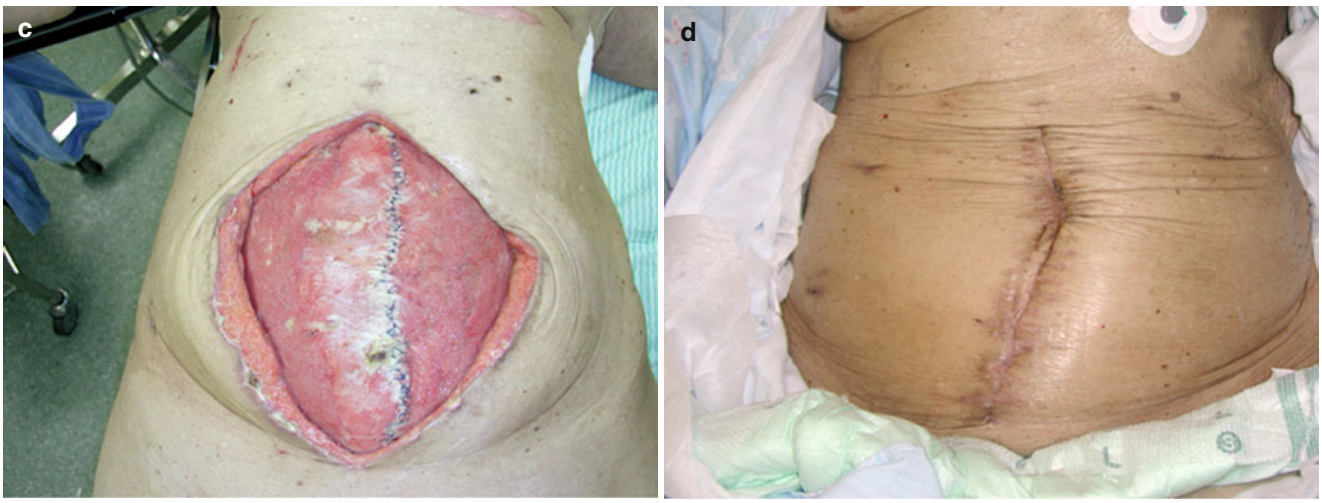


Fig. 14.7 (continued)

Conclusion

In caring for patients requiring open abdominal management/abdominal wall defects, vacuum-assisted wound closure reportedly raises the likelihood of early fascial reapproximation and decreases the need for later complex abdominal wall reconstruction [7, 36]. However, in the typical scenario necessitating open abdomen management for cases unable to undergo early standard fascial closure, many patients require prolonged open abdomen because of visceral edema. During this period, the laterally displaced muscles of the abdominal wall retract, shorten, and scar in their altered positions. Next is an interval with an intentional large ventral hernia, during which granulated abdominal contents are covered only by a skin graft. This abdominal wall defect requires late reconstruction 6–12 months after the initial surgery. Enterocutaneous fistula formation is a devastating complication of open abdomen. This reportedly occurs in 5–25% of cases [8, 19, 37, 38], although lower fistula rates have been reported using vacuum-assisted wound closure [7, 8, 36]. Enterocutaneous fistula formation can develop even after skin grafting of the granurated open abdominal wound. Early definitive wound closure is essential to prevent fistula formation. From this perspective, the complex tissue transfer method for early fascial closurecan benefit patients by reducing the risk of this devastating complication.

References

1. Rotondo MF, Schwab CW, McGonigal MD, et al. "Damage control": an approach for improved survival in exsanguinating penetrating abdominal injury. J Trauma. 1993;35:375–83.
2. Moore EE, Burch JM, Franciose RJ, et al. Staged physiologic restoration and damage control surgery. World J Surg. 1998;22:1184–91.
3. Shapiro MB, Jenkins DH, Schwab CW, et al. Damage control: collective review. J Trauma. 2000;49:969–78.
4. Mcnelis J, Soffer S, Marini CP, et al. Abdominal compartment syndrome in the surgical intensive care unit. Am Surg. 2002;68:18–23.
5. Rasmussen TE, Hallett Jr JW, Noel AA, et al. Early abdominal closure with mesh reduces multiple organ failure after ruptured abdominal aortic aneurysm repair: guidelines from a 10-year case–control study. J Vasc Surg. 2002;35:246–53.
6. Lowe JB, Lowe JB, Baty JD, et al. Risks associated with "components separation" for closure of complex abdominal wall defects. Plast Reconstr Surg. 2002;111:1276–83.
7. Garner GB, Ware DN, Cocanour CS, et al. Vacuum-assisted wound closure provides early fascial reapproximation in trauma patients with open abdomen. Am J Surg. 2001;182:630–8.
8. Miller PR, Thompson JT, Faler BJ, Meredith JW, Chang MC. Late fascial closure in lieu of ventral hernia: the next step in open abdomen management. J Trauma. 2002;53:843–9.
9. Wittmann DH, Aprahamian C, Bergstein JM. Etappenlavage: advanced peritonitis managed by planned multiple laparotomies utilizing zippers, slide fastener, and Velcro analogue for temporary abdominal closure. World J Surg. 1990;14:218–26.
10. Miller RS, Morris Jr JA, Diaz Jr JJ, Herring MB, May AK. Complications after 344 damage-control open celiotomies. J Trauma. 2005;59:1365–71.
11. Cothren CC, Moore EE, Johnson JL, Moore JB, Burch JM. One hundred percent fascial approximation with sequential abdominal closure of the open abdomen. Am J Surg. 2006;192:238–42.
12. Diaz Jr JJ, Dutton WD, Ott MM, et al. Eastern Association for the Surgery of Trauma: a review of the management of the open abdomen—Part 2 "Management of the Open Abdomen". J Trauma. 2011;71(2):502–12.
13. Defranzo AJ, Pitzer K, Molnar JA, et al. Vacuum-assisted closure for defects of the abdominal wall. Plast Reconstr Surg. 2008;121:832–9.
14. Petersson U, Acosta S, Bjorck M. Vacuum-assisted wound closure and mesh-mediated fascial traction—a novel technique for late closure of the open abdomen. World J Surg. 2007;31:2133–7.
15. Tsuei BJ, Skinner JC, Bernard AC, Kearney PA, Boulanger BR. The open peritoneal cavity: etiology correlates with the likelihood of fascial closure. Am Surg. 2004;70:652–6.

16. Teixeira PG, Salim A, Inaba K, et al. A prospective look at the current state of open abdomens. Am Surg. 2008;74:891–7.
17. Kushimoto S, Yamamoto Y, Aiboshi J, et al. Usefulness of the bilateral anterior rectus abdominis sheath turnover flap method for early fascial closure in patients requiring open abdominal management. World J Surg. 2007;31:2–8.
18. Geffen HJAA, Simmermacher RKJ, Vroonhoven TJMV, et al. Surgical treatment of large contaminated abdominal wall defects. J Am Coll Surg. 2005;201:206–12.
19. Jernigan TW, Fabian TC, Croce MA, et al. Staged management of giant abdominal wall defect. Acute and long-term results. Ann Surg. 2003;238:349–57.
20. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86:519–26.
21. DeFranzo AJ, Kingman GJ, Sterchi JM, Marks MW, Thorne M. Rectus turnover flaps for the reconstruction of large midline abdominal wall defects. Ann Plast Surg. 1996;37:18–23.
22. Ennis LS, Young JS, Gampper TJ, Drake DB. The "open-book" variation of component separation for repair of massive midline abdominal wall hernia. Am Surg. 2003;69:733–43.
23. Porshinsky B, Ramasastry S. Abdominal wall reconstruction with free flaps. Clin Plast Surg. 2008;33:269–80.
24. Huger WE. The anatomic rationale for abdominal lipectomy. Am Surg. 1979;45:612–7.
25. de Vries Reilingh TS, van Goor H, Rosman C, et al. "Components separation technique" for the repair of large abdominal wall hernias. J Am Coll Surg. 2003;196:32–7.
26. Maas SM, van Engeland M, Leeksma NG, et al. A modification of the "components separation" technique for closure of abdominal wall defects in the presence of an enterostomy. J Am Coll Surg. 1999;189:138–40.
27. Nguyen V, Shestak KC. "Separation of anatomic components" method of abdominal wall reconstruction—clinical outcome analysis and an update of surgical modifications using the technique. Clin Plast Surg. 2006;33:247–57.
28. Geffen HJAA, Simmermacher RKJ, Vroonhoven TJMV, et al. Surgical treatment of large contaminated abdominal wall defects. J Am Coll Surg. 2005;201:206–12.
29. Ghazi B, Deigni O, Yezhelyev M, et al. Current options in the management of complex abdominal wall defects. Ann Plast Surg. 2011;66:488–92.
30. Howdieshell TR, Proctor CD, Sternberg E, Cue JI, Mondy JS, Hawkins ML. Temporary abdominal closure followed by definitive abdominal wall reconstruction of the open abdomen. Am J Surg. 2004;188:301–6.
31. Rodriguez ED, Bluebond-Langner R, Silverman RP, et al. Abdominal wall reconstruction following severe loss of domain: the R. Adams Cowley Shock Trauma Center algorithm. Plast Reconstr Surg. 2007;120:669–80.
32. Connolly PT, Teubner A, Lees NP, Anderson ID, Scott NA, Carlson GL. Outcome of reconstructive surgery for intestinal fistula in the open abdomen. Ann Surg. 2008;247:440–4.
33. Moon HK, Taylor GI. The vascular anatomy of rectus abdominis musculocutaneous flaps based on the deep superior epigastric system. Plast Reconstr Surg. 1988;82:815–32.
34. El-Mrakby HH, Milner RH. The vascular anatomy of the lower anterior abdominal wall: a microdissection study on the deep inferior epigastric vessels and the perforator branches. Plast Reconstr Surg. 2002;109:539–43.
35. Boyd JB, Taylor GI, Corlett R. The vascular territories of the superior epigastric and the deep inferior epigastric systems. Plast Reconstr Surg. 1984;73:1–16.
36. Suliburk JW, Ware DN, Balogh Z, et al. Vacuum-assisted wound closure achieves early facial closure of open abdomens after severe trauma. J Trauma. 2003;55:1155–60.
37. Fabian TC, Croce MA, Pritchard FE, et al. Planned ventral hernia: staged management for acute abdominal wall defects. Ann Surg. 1994;219:643–53.
38. Nagy KK, Filder JJ, Mahr C, et al. Experience with three prosthetic materials in temporary abdominal wall closure. Am Surg. 1996;62:331–8.

Abdominoplasty and Panniculectomy in the Presence of Abdominal Wall Hernias

15

Maurice Y. Nahabedian

Introduction

The combination of abdominal panniculectomy and hernia repair is becoming an increasingly accepted technique. The issue of what to do with the excess skin has always been a consideration. Advocates of immediate panniculectomy will cite advantages such as less infection, better healing, and improved patient satisfaction. Advocates for delayed panniculectomy will cite the added length of the operation when combined with hernia repair, compromised vascularity of the abdominal skin and fat, and delayed healing, all of which may have a negative impact on outcomes.

There has been a variety of publications in the scientific literature that have addressed this issue [1–7]. A known comorbidity in many patients with a complex abdominal hernia is obesity. It is not unusual to operate on patients with abdominal hernias that have body mass index (BMI) measurements in excess of 30 [4]. These patients are at increased risk for delayed healing, infection, and skin necrosis. A significant advancement in support of immediate panniculectomy is the concept of perforator sparing [2]. These perforating vessels emanate from the primary regional source vessels that traverse through the abdominal musculature and perfuse the overlying skin and fat via perforating vessels. Preservation of these perforators will better preserve the vascularity to the adipocutaneous component and minimize wound-healing complications. This technique is further discussed in this chapter.

The benefits of immediate panniculectomy have been established. Hughes and colleagues have demonstrated that the incidence of wound-related problems such as dehiscence, infection, and necrosis are increased in the setting of obesity [4]. Performance of a panniculectomy at time of hernia repair not only reduces the incidence of wound complications but also can reduce the incidence of hernia recurrence [4]. Reid and Dumanian demonstrated a reduction in the incidence of wound infection from 18 to 50 % down to 8 % when the hernia repair was performed in conjunction with a panniculectomy [7]. Other studies have demonstrated that a panniculectomy in the setting of a hernia repair can improve patient satisfaction with regard to overall appearance, hygiene, and self-confidence. Cooper et al. reviewed a series of 92 patients following abdominal panniculectomy; 47 % had a concomitant abdominal wall hernia [1]. The panniculectomy was performed in one of three ways: minimal undermining, extensive undermining, and using a fleur-de-lis approach. Complication and reoperation rates were similar in all groups at approximately 43 and 14 %, respectively. Overall results demonstrated high satisfaction in 57 % and good satisfaction in 24 %. Hygiene was much improved in 74 % and somewhat improved in 13 %. Self-confidence was much improved in 53 % and somewhat improved in 33 % of patients. Finally, physical appearance was much improved in 56 % and somewhat improved in 33 % of patients.

Performing a panniculectomy may appear simple at first glance; however, there are several important factors that must be appreciated to perform it safely and effectively. The goal of this chapter is to review some of the techniques of abdominal panniculectomy and to provide the operating surgeon with a template for proceeding in these patients.

Clinical Anatomy

The relevant anatomy of the anterior abdominal wall can be subdivided into four components: the skin and subcutaneous tissue, the superficial fascial system (SFS), the anterior rectus sheath, and the muscle layers, which include the rectus abdominis muscle as well as the internal, external, and transverse oblique musculature. The collagen fibers of the rectus sheath and linea alba are principally involved in the stabilization and support of the anterior abdominal wall. The SFS

M.Y. Nahabedian, MD
Department of Plastic Surgery, Georgetown University,
3800 Reservoir Road NW, Washington, DC 20007, USA
e-mail: drnahabedian@aol.com, www.mauricenahabedian.com

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_15,

consists of fibrous septa that connect the skin to the underlying muscle fascia to create zones of adherence [8]. Inherent to these layers are the blood supply and innervation. There is an inherent interrelationship between these factors, all of which are important when performing a panniculectomy.

Panniculectomy is defined as the excision of the redundant skin and subcutaneous fat to improve the abdominal contour. It is differentiated from abdominoplasty in that there is a functional component in patients with a pannus requiring a panniculectomy. Patients with an abdominal pannus may have other associated morbidities, such as diabetes mellitus, hypertension, or chronic obstructive pulmonary disease (COPD). They often have changes associated with the skin and soft tissue and might be predisposed to infection and ulceration. In its simplest form, a panniculectomy involves removal of excess skin and fat. However, for a panniculectomy to be effective, it must also address the underlying anatomy. This includes the supportive layer of the anterior abdominal wall as well as the underlying muscles. Failure to address all components can lead to failure of the operation, poor outcomes, and unhappy patients.

Skin and Subcutaneous Fat

The fat or subcutaneous layer will vary in thickness from patient to patient. In some patients, the excess skin and fat may form a pannus in which there is an overhanging apron of tissue. In others, there might be a convexity without true pannus formation. This is what physically differentiates a panniculectomy from an abdominoplasty.

The vascularity of the fat is generally sparse and composed of thin arterioles, venules, and capillaries. The neural structures are rarely visible but extend from the fascial layer to the skin. They are sensory in nature and oriented perpendicularly. Motor nerves are usually not seen coursing through the subcutaneous layer of the anterior abdominal wall.

Anterior Rectus Sheath and Linea Alba

The anterior rectus sheath and linea alba are important structures that are sometimes overlooked when assessing abdominal contour [9–12]. They, along with the abdominal muscles, are responsible for maintaining support and strength of the anterior abdominal wall. The anterior rectus sheath and the linea alba also are the fibrous support system of the anterior abdominal wall. The anterior rectus sheath is the convergence of the aponeurotic extensions of the external and internal oblique musculature. The linea alba is the midline confluence of these aponeurotic extensions. The anterior rectus sheath and linea alba are composed of collagen fibers arranged in an interwoven lattice. The width and thickness of these structures will vary along the surface of the anterior abdominal wall. These measurements will fluctuate at various regions of the anterior abdominal wall and are related to the distance from the umbilicus. With respect to the linea alba, its width ranges from 11 to 21 mm between the xiphoid process and the umbilicus and then decreases from 11 to 2 mm from the umbilicus to the pubic symphysis. The thickness of the linea alba ranges from 900 to 1,200 μm between the xiphoid and the umbilicus and increases from 1,700 to 2,400 μm from the umbilicus to the pubic symphysis. With respect to the anterior rectus sheath, the thickness ranges from 370 to 500 μm from the xiphoid to the umbilicus and then increases from 500 to 700 μm from the umbilicus to the pubic symphysis. The posterior rectus sheath, on the other hand, is slightly thicker than the anterior rectus sheath above the umbilicus, 450–600 μm, but then drops off precipitously from the umbilicus to the arcuate line, 250–100 μm.

Vascularity and Innervation

The vascularity and innervation to the supportive structures of the anterior abdominal wall are important. The blood supply to the adipocutaneous layer is derived from the superficial inferior epigastric vessels and perforating branches from the deep inferior epigastric vessels. The blood supply to the anterior sheath is derived primarily from the superficial epigastric, inferior epigastric, and intercostal vascular networks. The innervation to the adipocutaneous layer is sensory and derived from the thoracic intercostal nerves. These sensory nerves will pierce the anterior rectus sheath and provide sensation to the skin.

The perforating vascularity supplying the adipocutaneous layer of the anterior abdominal wall has become increasingly appreciated in light of the fact that simultaneous panniculectomies are becoming increasingly commonplace. There are numerous perforating vessels throughout the anterior abdominal wall, the classification of which has been described [13]. These vessels are of variable caliber and randomly interspersed. Along the rectus abdominis musculature, the perforating vessels are most often arranged in two rows: medial and lateral. The caliber of these perforating vessels generally ranges from 1 to 2.5 mm in diameter, with the larger perforators usually located along the lateral row. Lateral row perforators usually emanate in proximity to the point of entry of the intercostal innervation to the rectus abdominis muscle,

which is normally at the junction of the lateral and central segment of the muscle. Along the oblique musculature, the perforators are generally the same size as those emanating from the inferior epigastric artery and veins. Although it is not exactly known how much tissue can be perfused by any given perforator, prior experience performing perforator flaps for breast reconstruction has demonstrated that about 750 g of tissue can be perfused by a 1.5-mm perforating artery and vein.

Preoperative Considerations

As with all operations, proper patient selection is important. A careful assessment of the risks and benefits of panniculectomy must be made, and the patient must be informed of the risks.

Assessment of Risk Factors

It is well known that patients with complex abdominal hernias may have a number of potential risk factors [2–4, 7]. These include but are not limited to diabetes mellitus, obesity, hypertension, pulmonary disease, poor nutritional status, cardiac disease, prior abdominal hernias, connective tissue disorders, abdominal aortic aneurisms, and immunosuppression. Patients considered for panniculectomy present a complicated picture because of other potential risks, which include but are not limited to prior soft tissue or cutaneous infections, fistula, indurated skin, lymphedema, and ulcerations. Before considering panniculectomy, a thorough history and physical examination is required. An assessment of these risks is necessary. Patient optimization might be necessary, especially in the setting of elective repair of the hernia and panniculectomy to lessen the risk of postoperative complications. In patients with an elevated BMI (>35), uncontrolled diabetes mellitus, and poor nutritional status, the incidence of complications, such as delayed healing, incisional dehiscence, soft tissue necrosis, infection, and prolonged drainage, is likely to be increased. The anticipated length of the operation may influence the timing of the panniculectomy, immediate or delayed.

Prior Hernia Surgical History

Patients with a prior history of hernia repair have added risks. Houck et al. demonstrated that the risk of a soft tissue infection is increased in patients with an abdominal hernia [14]. Although many of these operations appear at first glance to be "clean" operations, they are in fact more susceptible to infection. The incidence of infection is approximately tenfold higher in patients with a hernia (16 % vs. 1.5 %). In patients with a prior abdominal hernia repair, the risk of infection continues to increase (42 % vs. 12 %). Given these facts, all elements of prior hernia repairs must be acquired. The other important aspect of this is to evaluate the prior incisions that were used for the initial and subsequent operations. Many of these incisions can compromise the blood supply to the adipocutaneous layer and can have an impact on the optimal design for the panniculectomy. In general, vertical midline incisions are preferred because a fleur-de-lis pattern can be used. With this pattern, excess skin and fat can be excised in the horizontal as well as the vertical plane. When the prior incisions are located in the mid- or upper abdomen and are transverse in orientation, problems related to blood supply can occur. Low transverse incisions are usually fine because this is where the transverse incisions are most often made for panniculectomy. In almost all situations, it is preferred to gain access to the abdominal cavity through a preexisting incision. The panniculectomy can be performed utilizing these incisions, creating additional incisions as needed while maintaining care to preserve the vascularity to the remaining soft tissues.

Operative Steps

Preparing for panniculectomy is in many ways similar to preparing for abdominoplasty. The principles and concepts for the two are similar. Design patterns for skin excision must consider the vascularity of the skin. This will be related to the location of the prior incisions, the thickness of the soft tissues, and the degree of undermining. The degree of soft tissue undermining must include an appreciation for the thickness of the pannus because this can have an impact on the perfusion of the adipocutaneous component.

Design Patterns for Panniculectomy

There are several design patterns for abdominal panniculectomy. These include the horizontal incision, vertical incision, and a horizontal and vertical incision, also known as the fleur-de-lis pattern. A recent modification of the fleur-de-lis pattern is the "Mercedes pattern," which is designed to minimize delayed healing at the trifurcation point [3]. The specific pattern will depend on the location of excess tissue and the location of the prior incisions. Most patients with abdominal

hernias will have a prior vertical midline incision. It is almost always preferred to use these incisions and to incorporate them into the excision pattern to minimize the creation of additional scars.

Technique of Perforator Sparing

In patients with abdominal hernias and excess abdominal skin and fat, some degree of undermining is usually necessary to close and contour the abdominal wall adequately. The undermining is always at the junction of the fascia and fat. All of the perforators supplying the skin and fat will pierce the fascia [2]. In general, the smaller perforators (0.5–1.0 mm) are usually cauterized. The larger perforators (1.5–2.5 mm), when visible, can be preserved. When considering perforator preservation, the presence of a palpable pulse is a good indicator of sufficient vascular caliber. The number of perforators to be spared is usually based on body habitus and the thickness of the abdominal pannus. The number of perforators spared typically ranges from one to three. Personal experience has demonstrated that preservation of a single perforating vessel on each side of the hemiabdomen is useful and can minimize delayed healing. Surgeons should be aware that some patients who have had prior hernia repairs might no longer have perforating vessels. That said, angiogenic factors, in some patients, can result in neovascularization based on the reestablishment of a perforator network.

Technique of Skin and Fat Excision

An important preoperative consideration when performing concurrent hernia repair and panniculectomy is the sequencing of the operation. At my institution, both the hernia and plastic surgeon are usually involved. The role of the plastic surgeon is most often to perform the panniculectomy; however, it is acknowledged that a single surgeon (plastic or general) may complete both portions of the operation. With the patient in the standing position, the amount of excess skin is approximated by grasping and elevating the pannus. The markings typically will include the incision for the hernia repair as well as the incisions for the panniculectomy.

Prior to the operative incisions, measures to control and limit blood loss might be considered. One such maneuver is to place tumescent fluid into the soft tissues of the pannus. The typical tumescent solution consists of 1 mL of 1:1,000 epinephrine solution per liter of lactated Ringer's solution. The operative approach to the abdomen is usually through a midline incision. It is my practice to repair the hernia prior to performing the panniculectomy. This is important to better assess the exact amount of skin and fat to be excised. If an open component separation has been performed, it is important to consider the blood supply to the skin. When the perforating vessels supplying the adipocutaneous component are visualized, preservation is recommended whenever possible. Once the hernia repair is complete, the adipocutaneous component is minimally and carefully separated from the anterior rectus sheath. The degree of undermining will depend on the thickness of the adipocutaneous tissues, location of scars, and assessment of vascularity. In general, it is wise to proceed cautiously when undermining, especially in patients with an extraordinarily thick and indurated pannus, as the degree of undermining is directly related to compromised distal tissue perfusion. Skin excisions involving the vertical dimension are performed by elevating the adipocutaneous flaps and redraping one side over the other. The overlapping areas are marked and excised. The amount of excess skin and fat is determined. Skin excisions involving the vertical and horizontal dimension proceed with the horizontal incision, which is usually located at the inferior base of the pannus. It is important to excise any abnormal or thickened skin. The vascularity of the skin flap is based superiorly. It is important to avoid extensive undermining, which may compromise vascularity. If the hernia was extremely large and associated with a loss of domain in which the hernia sac lined the deep fat layer, this sac or scar is excised because it might be a nidus for infection based on the phenomenon of bacterial translocation. The skin flaps are then elevated and redraped to determine how much will be excised. Skin excision is performed sharply to minimize any thermal damage to the edges.

Butler and colleagues have described the Mercedes pattern for abdominal panniculectomy in the setting of hernia repair [3]. This technique is indicated in patients for whom a vertical and horizontal skin excision is necessary. The advantage of this pattern is that it will minimize the delayed healing and skin necrosis that often occurs at the trifurcation point of the vertical and horizontal incisions. In preparation for this technique, the vertical midline and transverse horizontal patterns are delineated much like the standard techniques. The unique feature of this design is that an equilateral triangular pattern is delineated just below the umbilicus, extending to the horizontal markings. The lengths of these triangular limbs are usually 15–20 cm and will vary based on body habitus and the dimensions of the pannus. This triangular skin is not excised with the panniculectomy. It is preserved as a caudally based flap that is advanced in the cephalad direction following the central and lateral skin excisions.

Closure Techniques

Prior to skin closure, the wounds are copiously irrigated with an antibiotic solution. Closed-suction drains are placed in the lateral gutters and as needed for the hernia repair. The closure is completed in layers using absorbable sutures in the Scarpa's layer and the dermis. The cutaneous closure can be performed using staples or sutures depending on the perceived risks of infection, delayed healing, and incisional dehiscence. In some cases, the incision is not closed completely, and a vacuum-assisted closure (VAC) device is applied. The reasoning for this is to minimize potential fluid collections and soft tissue edema. Once stable, the VAC can be removed and the wound closed secondarily.

Clinical Example

A 50-year-old woman presents with a large recurrent epigastric midline hernia with an abdominal pannus (Figs. 15.1 and 15.2). Complaints include pain and appearance. Comorbidities include controlled diabetes mellitus and hypertension. The hernia defect measures 10×8 cm. Concomitant hernia repair and panniculectomy are planned. A fleur-de-lis pattern is delineated with the patient standing (Fig. 15.3). In the operating room, the incisional approach for the hernia and panniculectomy are redelineated (Fig. 15.4). Following the lysis of adhesions, undermining over the anterior rectus sheath is completed with perforator sparing (Fig. 15.5). Porcine acellular dermal matrix is used as an underlay to reinforce the repair (Fig. 15.6). Unilateral component separation and direct approximation of the fascial edges is achieved, thus completing the hernia portion of the procedure (Fig. 15.7).

The panniculectomy portion begins by extending the vertical incision to the inferior horizontal markings. The lower transverse incision is created, extending to the anterior rectus sheath with undermining that extends to the upper transverse markings (Fig. 15.8). The upper transverse and vertical incisions are created (Fig. 15.9) and based on degree of tissue laxity and mobility. It is recommended to excise slightly less of the adipocutaneous tissue than expected in the event that there might be some postoperative tissue necrosis. Figure 15.10 depicts the excised tissue, which measures approximately 60 cm horizontally and 40 cm vertically. Figure 15.11 demonstrates the appearance of the abdominal wall prior to closure. The perforators have been spared along the vertical border. Closure is completed in a layered fashion using absorbable sutures in Scarpa's layer, the dermis, and skin (Fig. 15.12). Staples may be considered for skin closure. Closed suction drains are placed. Figure 15.13 demonstrates improved abdominal contour at 6-month follow-up.

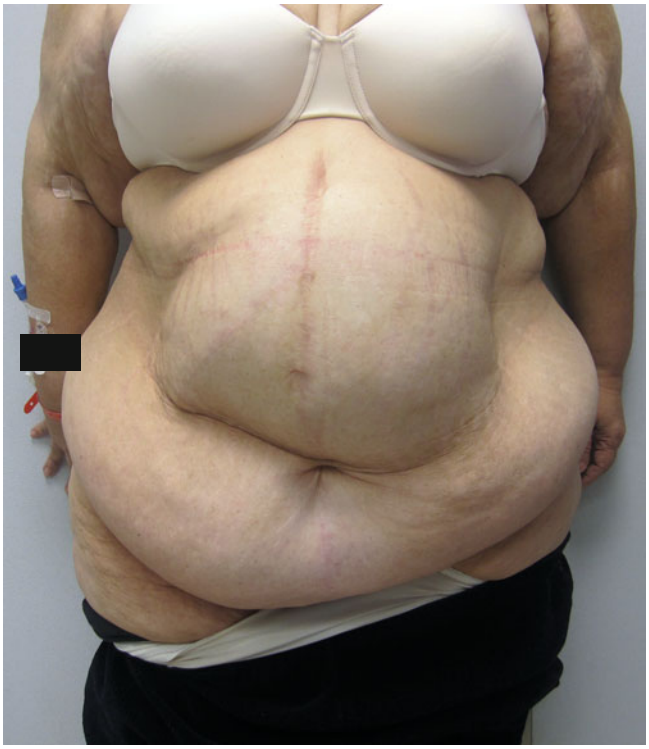


Fig. 15.1 Preoperative view demonstrating a moderate size epigastric hernia and skin redundancy

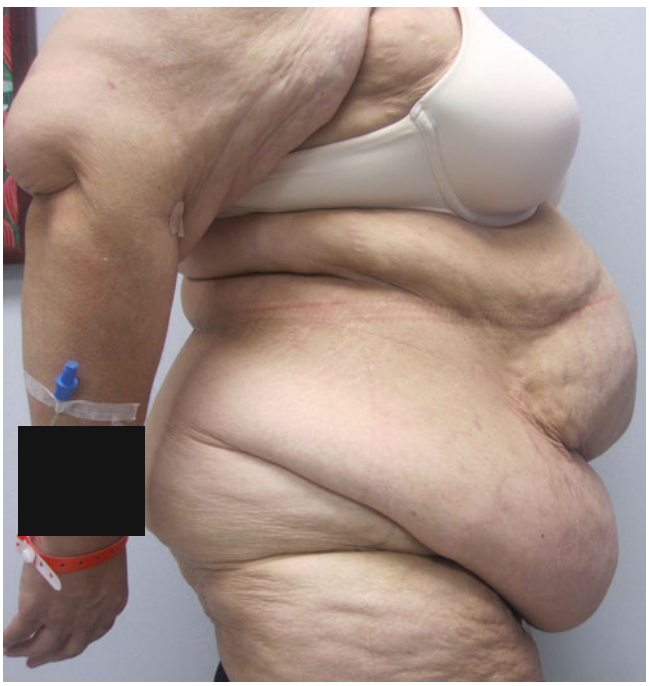


Fig. 15.2 The abdominal pannus and hernia are clearly depicted on this lateral view

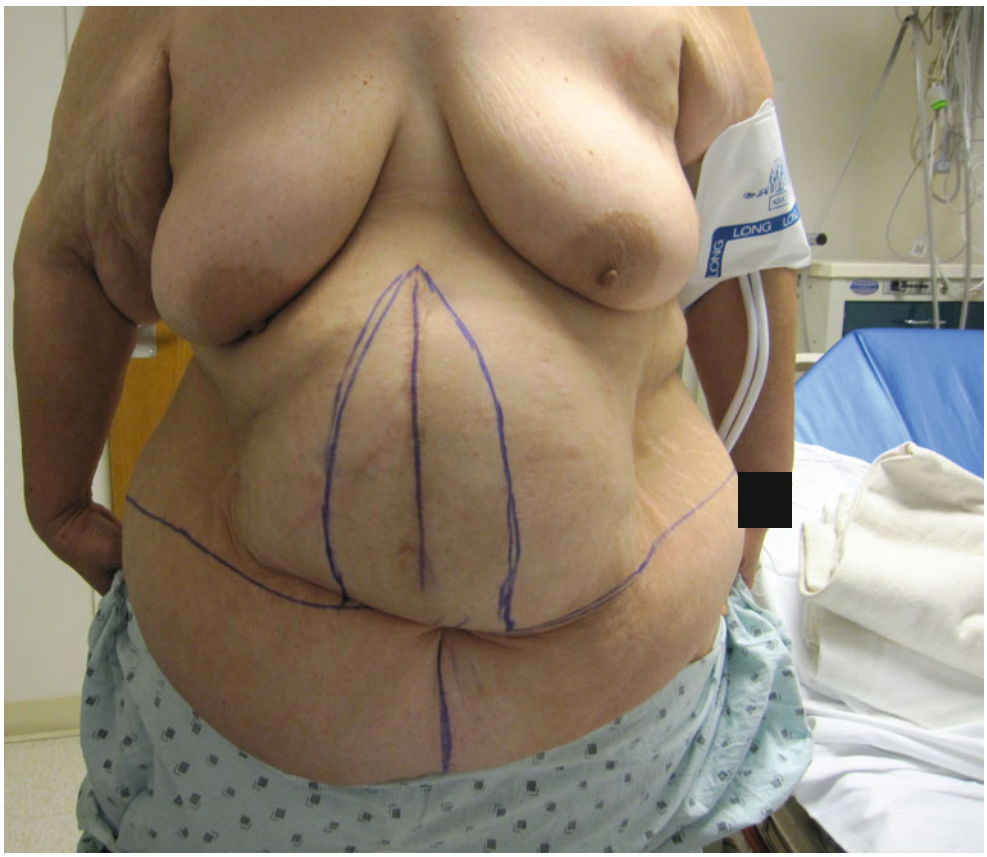


Fig. 15.3 The preoperative markings are delineated using a midline approach for the hernia and a fleur-de-lis pattern for the panniculectomy

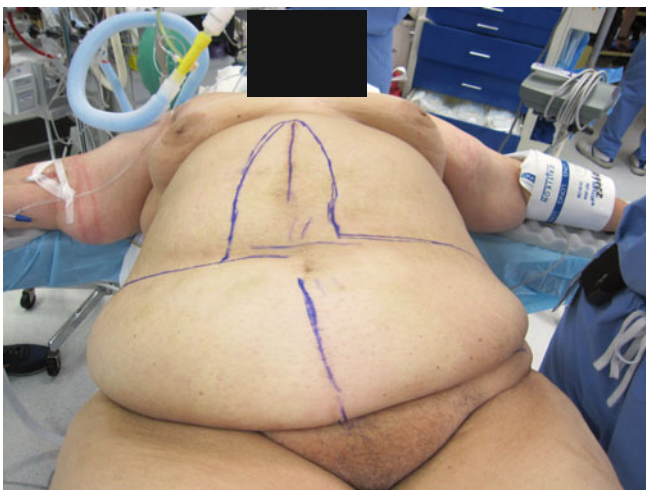


Fig. 15.4 The markings are redelineated with the patient supine on the operating table

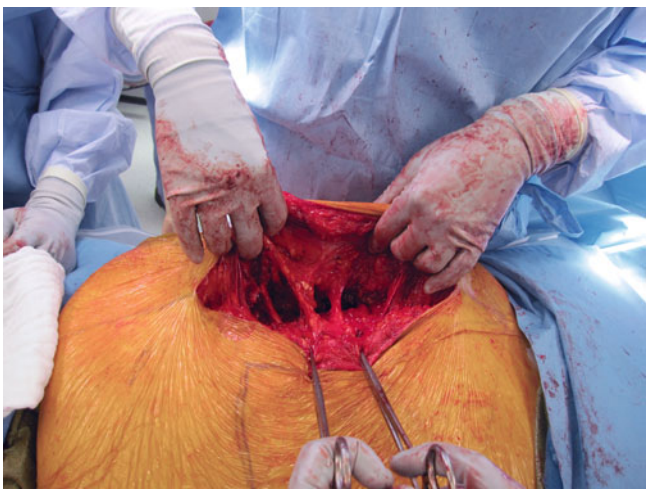


Fig. 15.5 The perforators emanating from the inferior epigastric artery and supplying the adipocutaneous tissues are clearly demonstrated and preserved

Postoperative Care

The postoperative care of the patient having hernia repair and panniculectomy is important. All patients are continued on intravenous antibiotic, usually for about 1 week. This may be prolonged if the patient develops an infection. The duration of the drains is variable and based on quantity of fluid as well as the need for prolonged negative pressure to promote tissue adherence. Pulmonary consideration may be relevant as the added pressure on the diaphragm from the hernia repair and the panniculectomy might increase airway resistance. Incentive spirometry and early ambulation are

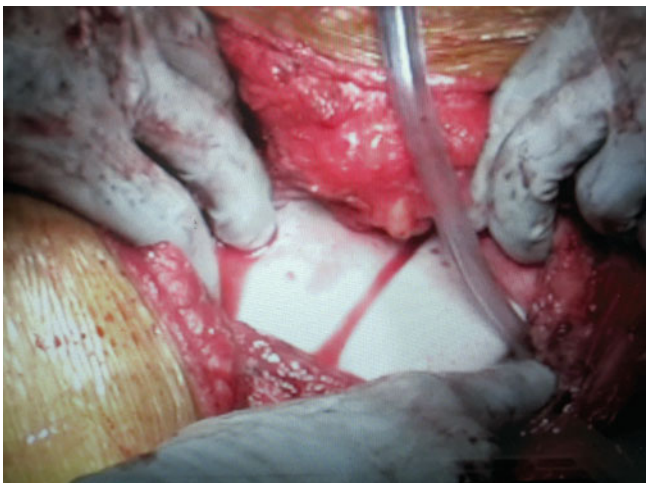


Fig. 15.6 Following mobilization of the abdominal musculature, porcine acellular dermal matrix is used as an underlay for reinforcement

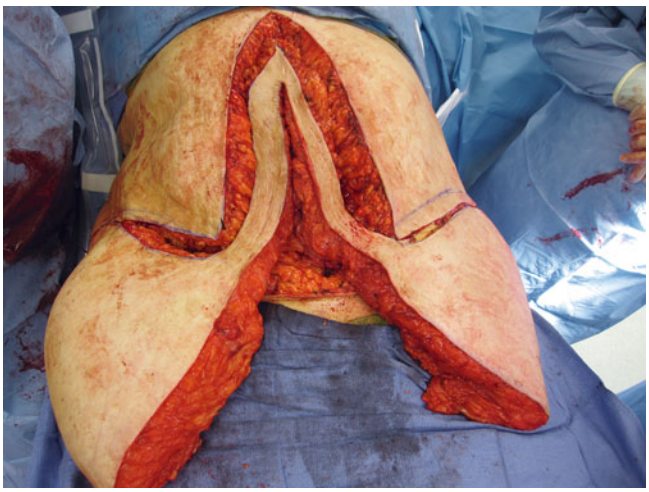


Fig. 15.9 The upper transverse and upper vertical skin is incised

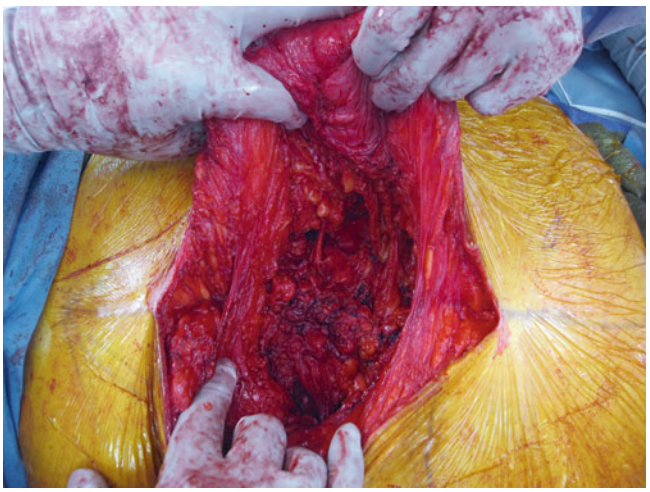


Fig. 15.7 Closure of the fascial edges with perforator sparing

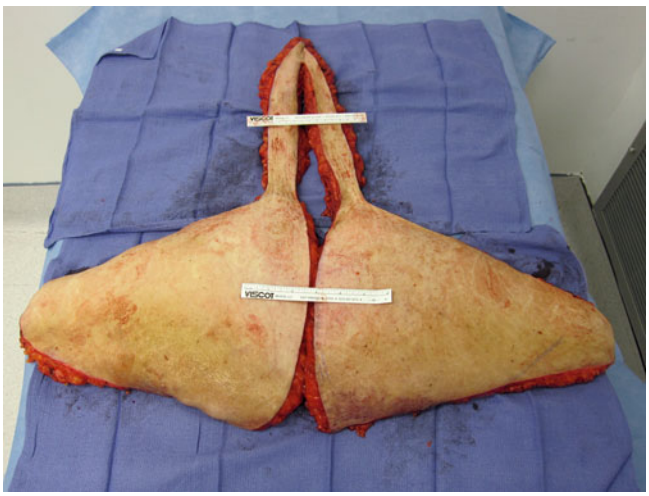


Fig. 15.10 The excised tissues measuring approximately 60×40 cm

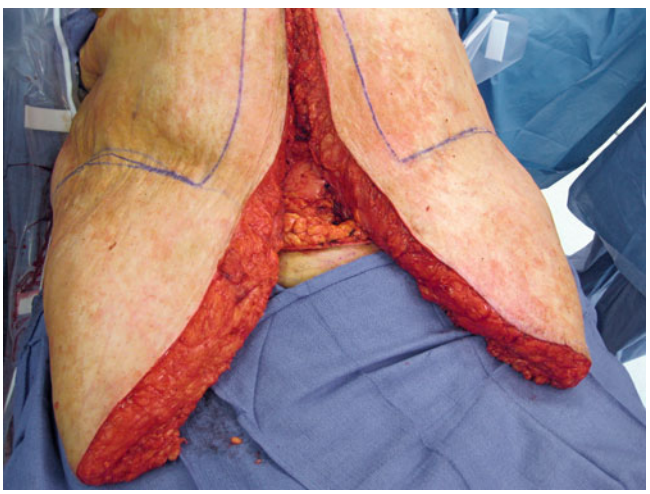


Fig. 15.8 Initiation of the panniculectomy incising the vertical and horizontal components

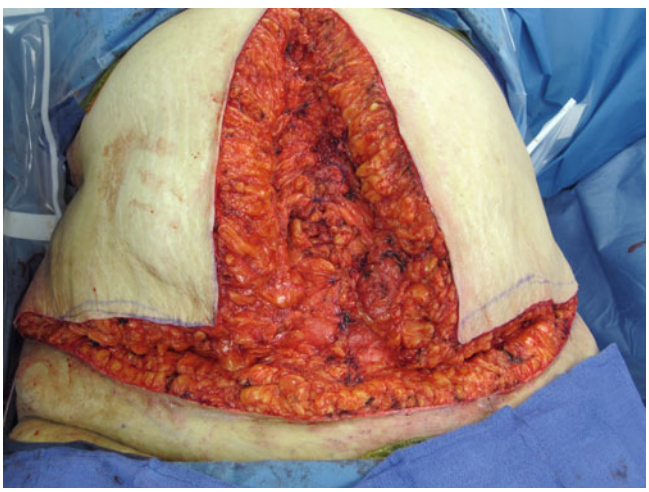


Fig. 15.11 The appearance of the abdominal wall following excision

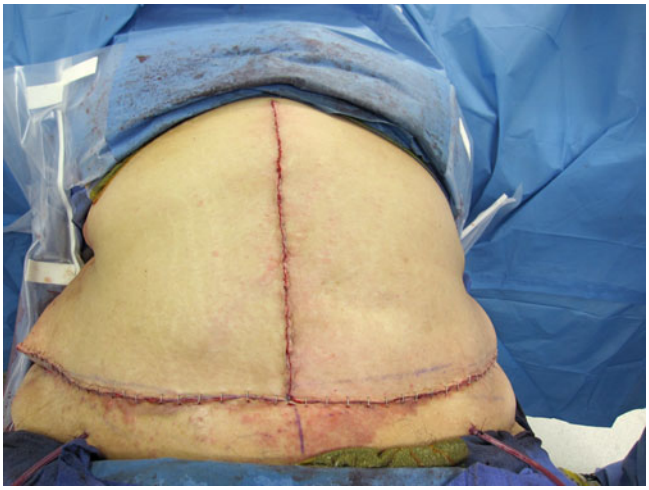


Fig. 15.12 The appearance of the abdomen following closure

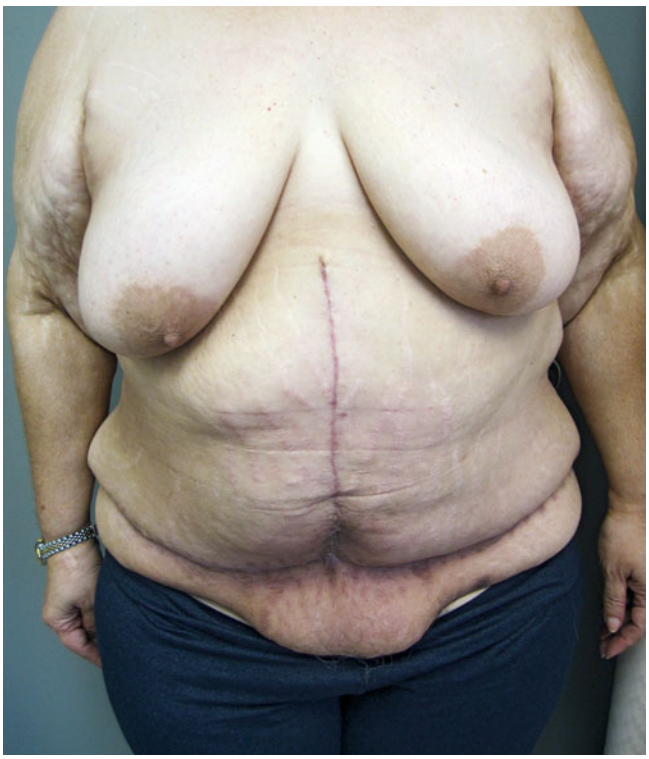


Fig. 15.13 The appearance of the abdomen 6 months following repair

encouraged to improve pulmonary status and circulation. These patients all require venous thrombo-embolism (VTE) prophylaxis in the form of pneumatic compression devices and chemoprevention using pharmaceutical agents such as Lovenox® (Sanofi-Aventis, Paris, France) or subcutaneous heparin. Nutritional status is assessed and diets are advanced as tolerated once bowel function has returned.

The length of hospital stay is variable and will depend on a variety of factors. These include but are not limited to return of bowel function, development of complications, and patient compliance. Reid and Dumanian determined that the average length of stay in patients who have component separation repair of an abdominal hernia with panniculectomy was 7.7 days [7].

Management of Complications

When performing panniculectomies in patients with abdominal hernias, complications should not be surprising. In the majority of cases, the complication will be infection, soft tissue necrosis, delayed healing, or incisional dehiscence. Reid and Dumanian demonstrated that the incidence of a major postoperative wound complication is increased six-fold when the body mass index is greater than 35, and that those patients are five times more likely to undergo reoperation [7]. Antibiotic therapy is almost always necessary for infection control, especially in patients with comorbidities. Local wound care is often sufficient; however, operative debridement may sometimes by necessary. Negative-pressure wound therapy is regularly used to minimize edema, promote wound contraction, and promote angiogenesis. Delayed closure is performed when the wound is optimized in terms of healthy granulation, tissue viability, and elimination of cellulitis.

Conclusions

Abdominal panniculectomy in conjunction with hernia repair has demonstrated success in properly selected patients. Patient satisfaction is improved, and the incidence of local wound morbidities is reduced. A variety of techniques exist by which panniculectomy can be performed; all can be successful. Perforator sparing to preserve the vascularity to the surrounding adipocutaneous tissues has facilitated the delivery of good-to-excellent outcomes. Panniculectomy should be considered in patients with a concomitant abdominal hernia and pannus.

References

1. Cooper CM, Paige KT, Beshlian KM, Downey DL, Thrilby RC. Abdominal panniculectomies: high patient satisfaction despite significant complication rates. Ann Plast Surg. 2008;61:188–96.
2. Saulis AS, Dumanian GA. Periumbillical rectus abdominis perforator preservation significantly reduces superficial wound complications in “separation of parts” hernia repairs. Plast Reconstr Surg. 2002;109:2275.
3. Butler CE, Reis SM. Mercedes panniculectomy with simultaneous component separation ventral hernia repair. Plast Reconstr Surg. 2010;125:94.
4. Hughes KC, Weider L, Fischer J, et al. Ventral hernia repair with simultaneous panniculectomy. Am Surg. 1996;62:678–81.

5. Nahas FX, Ishida J, Gemperli R, et al. Abdominal wall closure after selective aponeurotic incision and undermining. Ann Plast Surg. 1998;41:606–17.
6. Netscher DT, Wigoda P, Spira M, et al. Musculoaponeurotic plication in abdominoplasty: how durable are its affects? Aesthetic Plast Surg. 1995;19:531–4.
7. Reid RR, Dumanian GA. Panniculectomy and the separation-of-parts hernia repair: a solution for the large infraumbilical hernia in the obese patient. Plast Reconstr Surg. 2005;116:1006.
8. Lockwood TE. Superficial fascial system (SFS) of the trunk and extremities: a new concept. Plast Reconstr Surg. 1991;86: 1009–18.
9. Axer H, Keyserlingk DG, Prescher A. Collagen fibers in linea alba and rectus sheaths: I. General scheme and morphological aspects. J Surg Res. 2001;96:127–34.
10. Axer H, Keyserlingk DG, Prescher A. Collagen fibers in linea alba and rectus sheaths: II. General scheme and morphological aspects. J Surg Res. 2001;96:239–45.
11. Korenkov M, Beckers A, Koebke J, et al. Biomechanical and morphological types of the linea alba and its possible role in the pathogenesis of midline incisional hernia. Eur J Surg. 2001;167: 909–14.
12. Rath AM, Attali P, Dumas JL, et al. The abdominal linea alba: an anatomic-radiologic and biomechanical study. Surg Radiol Anat. 1996;18:281–8.
13. Blondeel PN, Van Landuyt KH, Monstrey SJ, et al. The "Gent" consensus on perforator flap terminology: preliminary definitions. Plast Reconstr Surg. 2003;112(5):1378–83.
14. Houck JP, Rypins EB, Sarfeh IJ, Juler GL, Shimoda KJ. Repair of incisional hernia. Surg Gynecol Obstet. 1989;169(5):397–9.

16 Abdominal Wall Reconstruction in Patients with an Open Abdomen and Enterocutaneous Fistulas: A Nine-Step Treatment Strategy

Rifat Latifi, Ruben Peralta, and Hassan Al Thani

Introduction

Enterocutaneous fistulas (ECFs) remain among the most serious complications of open abdomen management techniques and damage control surgery, particularly in acute care and trauma surgery. ECFs are associated with significant morbidity and mortality, despite significant advances in surgical techniques and technologies for patients with complex abdominal wall hernias. Especially challenging is the combination of ECFs and any or all of these conditions: large abdominal defects, an open abdomen, enteroatmospheric fistulas (EAFs), or stomas. The frequency of the combination of ECFs and abdominal wall hernias is unknown [1]. In addition, the percentage of patients treated for ECFs in concurrence with reconstruction surgery for large abdominal wall defects is unclear; in our practice, this percentage is about 20 % [2]. Of the ECFs, 75–85 % are postoperative, and most patients with ECFs also have abdominal wall defects (through which the ECFs become evident); therefore, the surgeon should treat both conditions in tandem. The incidence of ECFs in combination with an open abdomen, on the other hand, has been reported to be as high as 75 % [3].

R. Latifi, MD, FACS (✉)
Department of Surgery,
The University of Arizona,
1501 N. Campbell Ave, Tucson,
AZ 85724, USA

Trauma Section, Department of Surgery,
Weill Cornell Medical College,
Hamad General Hospital, Doha, Qatar
e-mail: latifi@surgery.arizona.edu

R. Peralta, MD, FACS
Trauma Section, Department of Surgery,
Hamad General Hospital,
Doha, Qatar
e-mail: rperaltamd@gmail.com

H. Al Thani, MD
Trauma Section, Department of Surgery,
Hamad General Hospital,
Doha, Qatar
e-mail: halthani@hmc.org.qa; althanih@hotmail.com

A Nine-Step Treatment Strategy

Establishing disciplined protocols and implementing a well-planned strategy will improve postoperative outcomes in patients with ECFs (whether alone or combined with an abdominal wall defect or an open abdomen). In 2008, van Gemert WG et al. [4] described a six-step strategy known as SOWATS (S = Sepsis control, O = Nutrition optimization, W = Wound care, T = Timing, A = Anatomy, and S = Surgery). We have modified this six-step strategy into a nine-step strategy, and we call it ISOWATS PL:

I = Identification and diagnosis of postoperative fistula
S = Sepsis control
O = Optimization of nutrition
W = Wound care
A = Redefining the anatomy of fistulas or abdominal wall defect
T = Timing of takedown of fistulas
S = surgery and surgical creativity
P = Postoperative care
L = Long-term follow-up.

We adhere to ISOWATS PL strategy as much as possible, although we sometimes cannot strictly follow all nine steps in certain patients, such as in patients in emergency operations.

Step 1: I = Identification of Postoperative Fistulas

Postoperatively, the surgeon must make the proper diagnosis, that is, identify the fistulas, in a timely fashion. Because the majority of fistulas are postoperative, it is often difficult to distinguish wound infections and abdominal dehiscence as a result of fistulas from serious wound infections and

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_16, © Springer Science+Business Media New York 2013

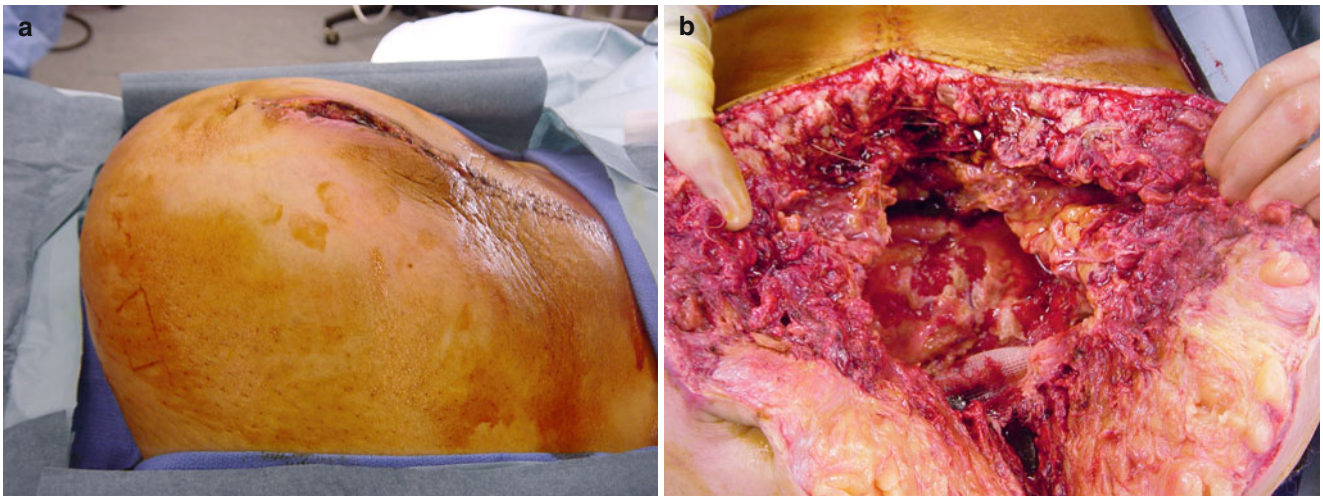


Fig. 16.1 (**a**) Large infected seroma requiring open drainage in a patient who underwent abdominal wall defect repair using synthetic mesh. (**b**) Intraoperative view of patient in (**a**). Infected synthetic mesh being removed

abdominal wall dehiscence that are not a result of fistulas (Fig. 16.1a, b). In all patients with abdominal wound dehiscence, especially after the creation of an anastomosis or anastomoses, the surgeon should be alert for the occurrence of fistulas or some other sort of catastrophe. Preoperatively, complex ventral hernias, ECFs, or EAFs must be identified, if at all possible, by whatever methods the surgeon thinks best: a fistulogram, a computed tomographic (CT) scan, or an upper gastrointestinal (UGI) study with small bowel follow-through. Most recently, the CT scan has become the standard radiographic study. However, aggressive wound exploration, often in the operating room, is required to completely assess the wound (as well as the subfascial collection and intestines lying under the sutures, which could easily erode into the lumen and cause new fistulas). Necrotic tissue needs to be debrided entirely (Fig. 16.2). The CT scan will identify any deep peritoneal or pelvic collection that could be drained and guided by CT or ultrasound.

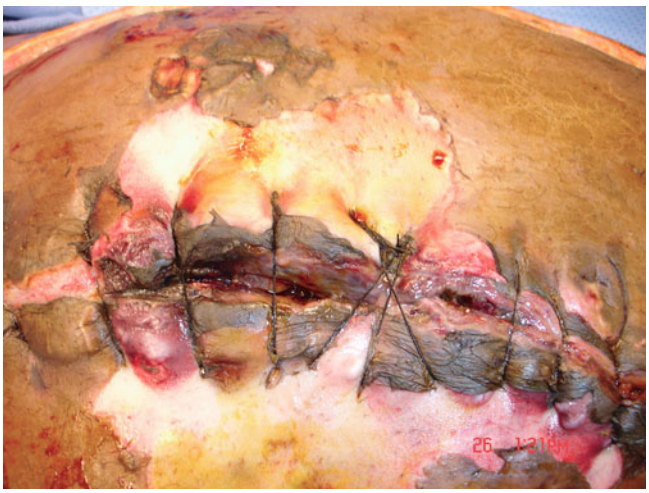


Fig. 16.2 Abdominal wall necrosis in a patient undergoing abdominal wall reconstruction with an interposition biological mesh; the patient developed intra-abdominal hypertension. Same patient is seen Fig. 16.11c

Step 2: S = Sepsis Control

The second step of the nine-step treatment strategy for treating patients with ECFs (or EAFs) is sepsis control, along with electrolyte and fluid normalization and achievement of hemodynamic stability. In the last few decades, sepsis control has undergone significant changes. In addition to early use of powerful antibiotics and goal-directed resuscitation, in critically ill patients less-invasive methods for treating intra-abdominal sepsis have become routine [5]. The mainstay of therapy for intra-abdominal abscesses remains drainage, be it surgical or percutaneous, but broad-spectrum antibiotics may be initiated and subsequently tailored based on culture results. Although intra-abdominal sepsis might be the main culprit, these patients might have other sources of sepsis, such as urinary infection, line sepsis, pneumonia, and other hospital-acquired infections, and thus require careful examination.

Step 3: O = Optimization of Nutrition

The third step is optimization of nutrition through initiation and maintenance of parenteral nutrition support or enteral feeding. In a busy practice, it is easily forgotten that patients who underwent major surgical operation need aggressive nutrition support, particularly in the perioperative period. In patients with a recent weight loss of 10–15 % or with a serum albumin level less than 3 grams/deciliter (g/dL), elective procedures should be postponed if at all possible. Albumin levels of less than 2.5 g/dL have been associated with a significant

increase in mortality and morbidity. A strong relation was reported between preoperative albumin level and surgical closure ($p < 0.001$) and mortality ($p < 0.001$) [4]. Before major surgery, the nutritional status of all patients (unless emergent surgery is required) should be optimized to the extent possible [6–13]. In a few patients, however, despite all attempts, reversing hypoalbuminemia and malnutrition will be impossible; such failure likely indicates continuous infection or sepsis or continuous losses of nutrients through fistula effluent (Figs. 16.3 and 16.4a, b). In addition, blood sugar levels should be optimized. Cessation of smoking is required.

Step 4: W = Wound Care

The fourth step is continuous wound care of these complex patients, thereby reducing the bioburden. To help avert sepsis and to improve the spirits of the patient, it is crucial to ensure proper hygiene and to avoid skin excoriations from the bile salts, intestinal fluids, or stool. Effective use of wound vacuum-assisted closure (VAC) and proper stoma equipment is important, although the evidence is still lacking [14]. Doing so, especially in patients with large open abdominal wall defects (which we have termed *fistula city*), might prove extremely difficult (Figs. 16.5a, b and 16.6). The wound VAC is meant to control the output of fistulas, but the surgeon must be cognizant of the amount of fluid that the patient loses and must ensure appropriate fluid replacement.

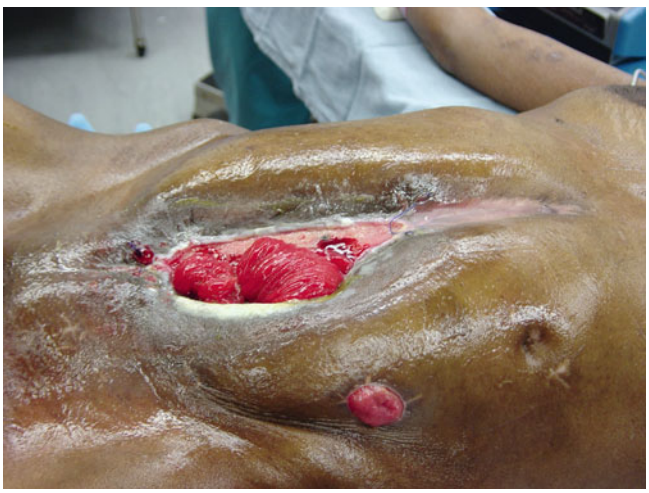


Fig. 16.3 Severely malnourished patient with multiple fistulas

Step 5: A = Redefining the Anatomy

The fifth step is redefining the anatomy. Again, if there is any question, here the surgeon should use any of the available techniques to confirm the anatomy. Previous operative reports should be studied carefully.

Step 6: T = Time of Operation or Takedown of ECF

The sixth step is probably the most important in this group of patients. How long to wait until takedown of ECFs is unclear. The surgeon should try not to intervene early if at all possible; however, these patients often continue to be septic and malnourished, so surgery itself will serve as source control. In our practice, we use the individual patient's condition as a guide rather than any strict predetermined timeline, although we to try to avoid operating in the first 2–3 months after diagnosis.

Step 7: S = Surgical Creativity

The seventh step encompasses the surgical creativity that is essential to complete the operation. The next section details the main elements that the surgeon must consider, including

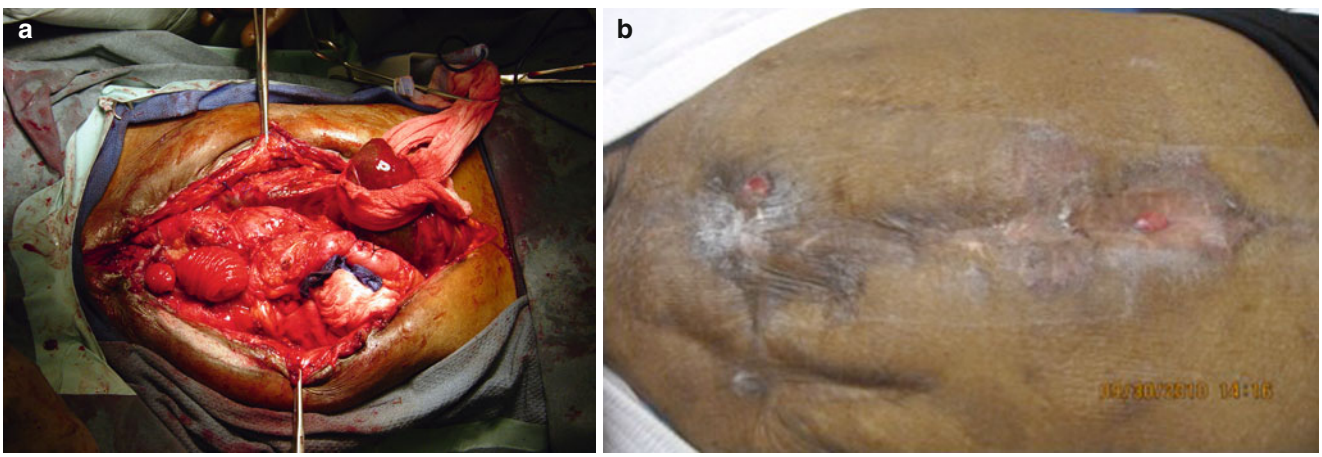


Fig. 16.4 (**a**) Intraoperative view of patient in Fig. 16.3. Left lobe of the liver is being held up with a lap pad. (**b**) Patient in Fig. 16.3 after healing from the previous operation

Fig. 16.4 (continued) (**c**) Patient in Fig. 16.3 with the senior author (the operating surgeon) a few months after post surgery

Fig. 16.5 (**a**) Large "stoma" bag. (**b**). Patient with a "stoma city," which is difficult to manage

the kind of incision, repair techniques, and mesh placement. The main surgical goals are to establish GI tract continuity and to minimize recurrence of ECFs, EAFs, hernias, and wound infections. A combination of different approaches is often required.

Surgical Approach

The abdominal wall of most patients with ECFs or EAFs is hostile; the surgeon might find that even entering the cavity itself presents a significant challenge. When possible, the

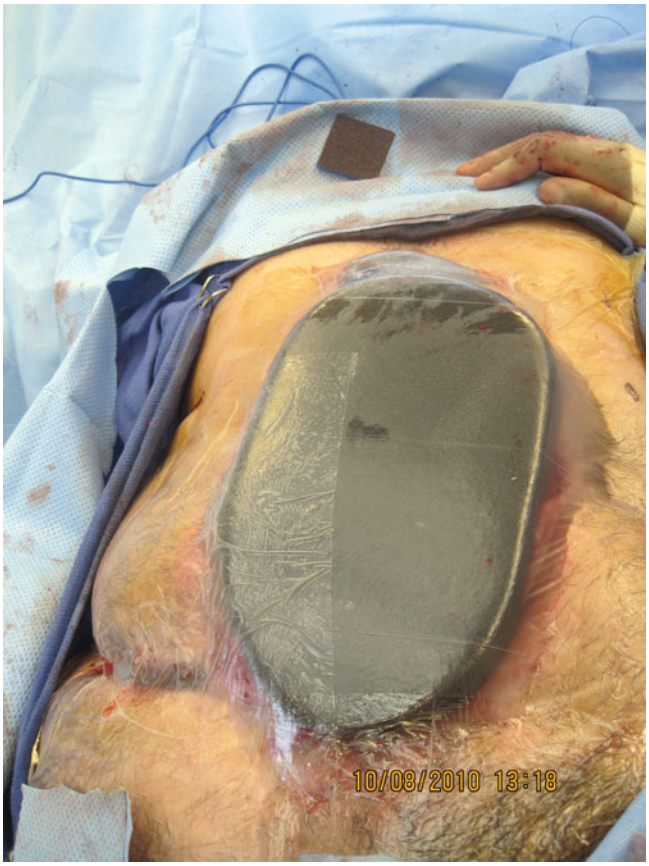


Fig. 16.6 Wound vacuum-assisted closure (VAC) in progress

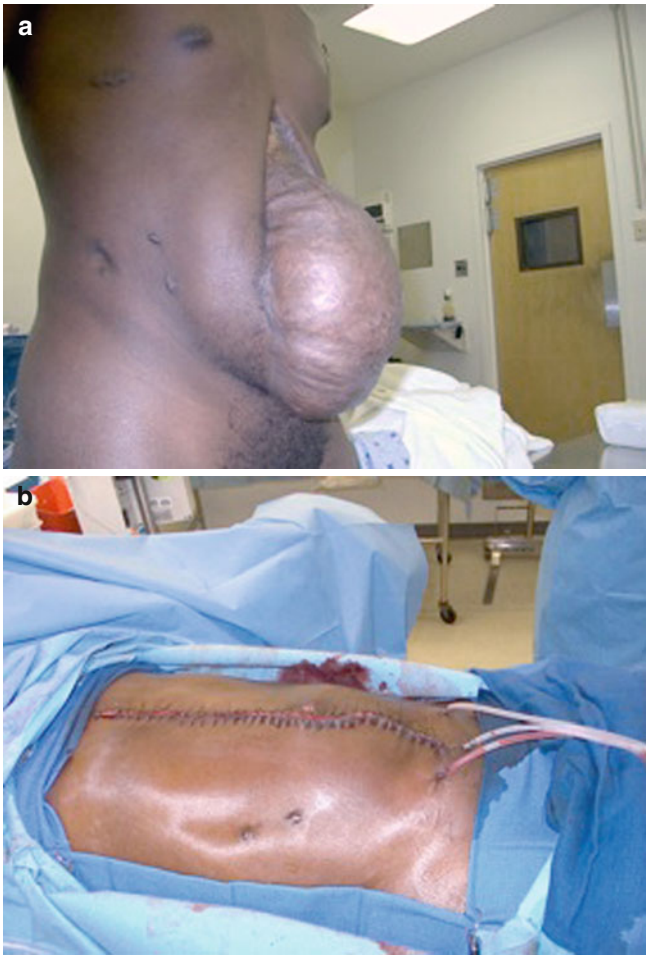


Fig. 16.7 (**a**) Skin graft in a patient managed with open abdomen after gunshot wound to the abdomen. (**b**) Same patient as in (**a**) at the end of the operation. We performed abdominal reconstruction using component separation and onlay mesh reinforcement (Fig. 16.10)

surgeon should avoid going through the same incision used in prior operations, instead attempting to enter from nonviolated areas of the abdominal wall (such as the superior epigastric region or just over the pubic region). However, doing so is usually not possible, especially in patients with prior major operations (such as laparotomy for trauma). An alternative method of entering the abdomen through a transverse incision has been advocated [11, 12], although we have not used that method in our practice.

In most patients cared for with an open abdomen, the abdominal contents are covered with a split-thickness skin graft (STSG) (Fig. 16.7a, b). Such patients require special attention to ensure the success of their completion surgery. Before the skin graft is excised, the neoskin, when pinched between the surgeon's thumb and forefinger, should be easily elevated from the underlying tissue. Some surgeons do not attempt to excise the skin graft at all, but close the abdomen over it. When excision is attempted while the skin graft is adherent, dissection is likely to result in enterotomies and to risk recurrent fistula formation [15].

One Alternative Approach

In patients with a hostile (or frozen) abdomen who developed a necrotic liver that required debridement, we have used a transthoracic approach. On multiple occasions, the senior author (R.L.) has either made an incision between the lower ribs or resected one or two lower ribs to debride the necrotic liver or pack the liver (unpublished data).

Issues with Adhesiolysis

Once the abdominal cavity is entered, the surgeon often faces a large ball of intestines wrapped by adhesions. Should these adhesions be separated? That question is as old as the surgery itself [16]. In our opinion, the surgeon should mobilize the entire segment of bowel, from the ligament of Treitz to the rectosigmoid. Doing so is tedious and time consuming, given previous abdominal surgeries and intra-abdominal inflammatory processes, and it is often complicated by new iatrogenic enterotomies. Other surgeons do not agree entirely with our approach; they suggest something in-between complete lysis, perhaps partial lysis of adhesions [17].

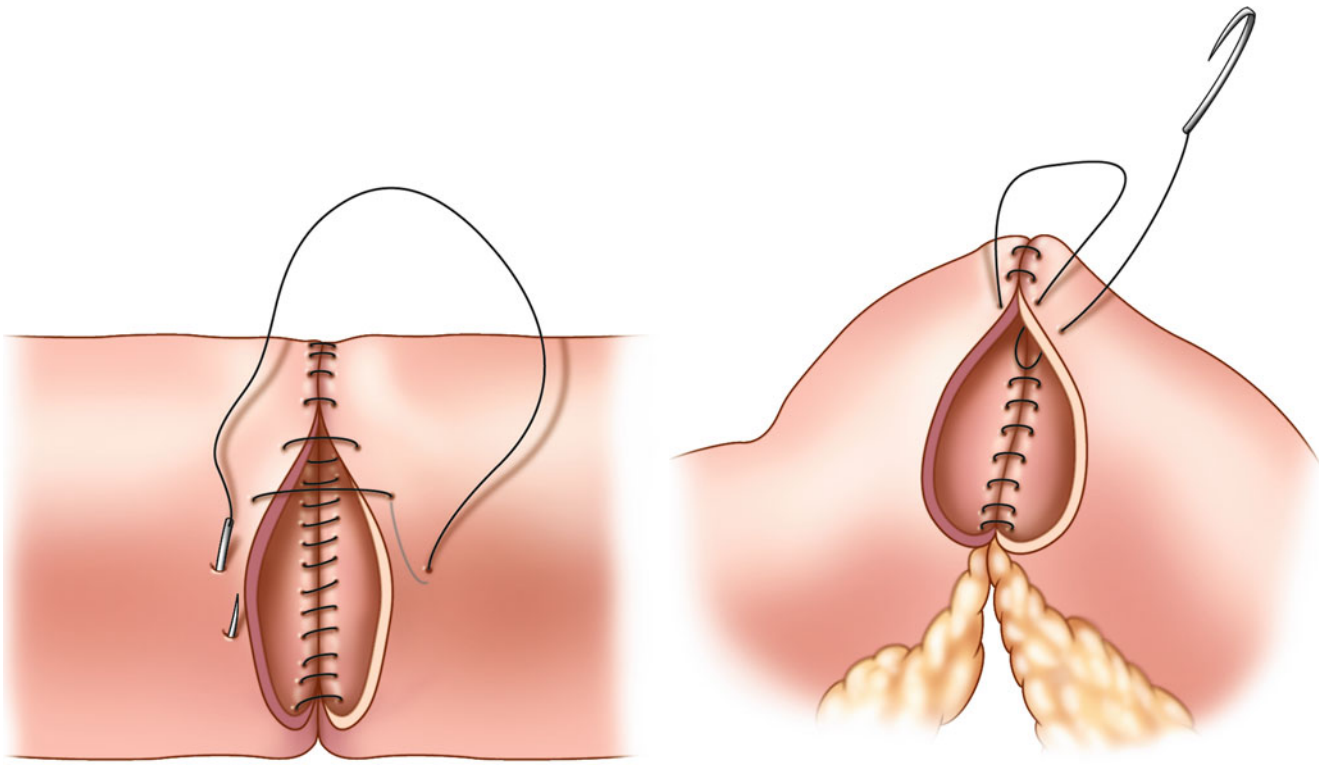


Fig. 16.8 Connell suture technique. Note that needle always points forward or outward

Fistula Resection

In patients with multiple ECFs or EAFs, resecting all of the fistulas might be challenging, but all of them must be resected [18–20]. The best scenario is when multiple fistulas are in close proximity to each other, so that the surgeon can excise the segment of fistulous tract "en masse." Yet, if the fistulas are far away from each other, more than one resection—and subsequently more than one anastomosis—might be required; all are technically challenging. Because such patients are at high risk for developing short-gut syndrome, adjunct procedures, such as strictureplasty, can help avoid removing large segments of bowel. Intraoperatively, it is important for the surgeon to identify all fistulas. Care should be taken to avoid enterotomies, but if they do occur, any inadvertent injury to the bowel must be either repaired immediately or tagged with a suture (so that it can be easily identified later during the course of the operation).

Anastomoses

For reestablishing intestinal continuity, the hand-sewn, double-layer technique (and not staplers) should be used [19]. In our practice, we prefer using continuous Vicryl™ (Ethicon, Somerville, NJ) sutures (Connell technique) (Fig. 16.8): The sutures go through the wall from the serosa to the mucosa, then from the mucosa to the serosa on the same side. The sutures then cross the incision to the serosa on the other side, and the pattern is repeated until suturing is completed. If the integrity of the anastomosis is questionable, it is reasonable to revise it or to create a proximal diverting ostomy. Excessive trimming of the mesentery, tension on the anastomosis, and inclusion of diseased bowel in the anastomosis must all be avoided [11, 12]. Operative treatment with takedown of ECFs is successful in 80–90 % of patients, although the presence of an open abdomen lowers the success rate to 77.3 % [4].

Definitive Abdominal Wall Reconstruction

Once the continuity of the GI tract has been established, as described previously, creating a new abdominal wall may represent a serious surgical challenge. Multidisciplinary approaches and advanced surgical techniques might be necessary [15]. Whatever technique is used, however, the goal is to create coverage of the abdominal cavity and to improve the patient's quality of life. Native abdominal wall can be used; if that is not possible, biologic or prosthetic mesh can be used instead. In most patients, some sort of combination of reconstruction techniques will be needed. If native tissue can be used without undue tension, then it should be used. But, if midline tissue cannot be easily approximated or if mesh reinforcement is needed (as it is in

almost all abdominal wall defects larger than 6 cm), then other techniques must be considered. For example, if midline tissue cannot be easily approximated, then lateral components need to be released, and a neoabdominal wall needs to be created. In our practice, we most commonly use tissue transposition of myocutaneous flaps through lateral component separation, as described previously [21, 22] (Fig. 16.9a–f). Component separation results in medial

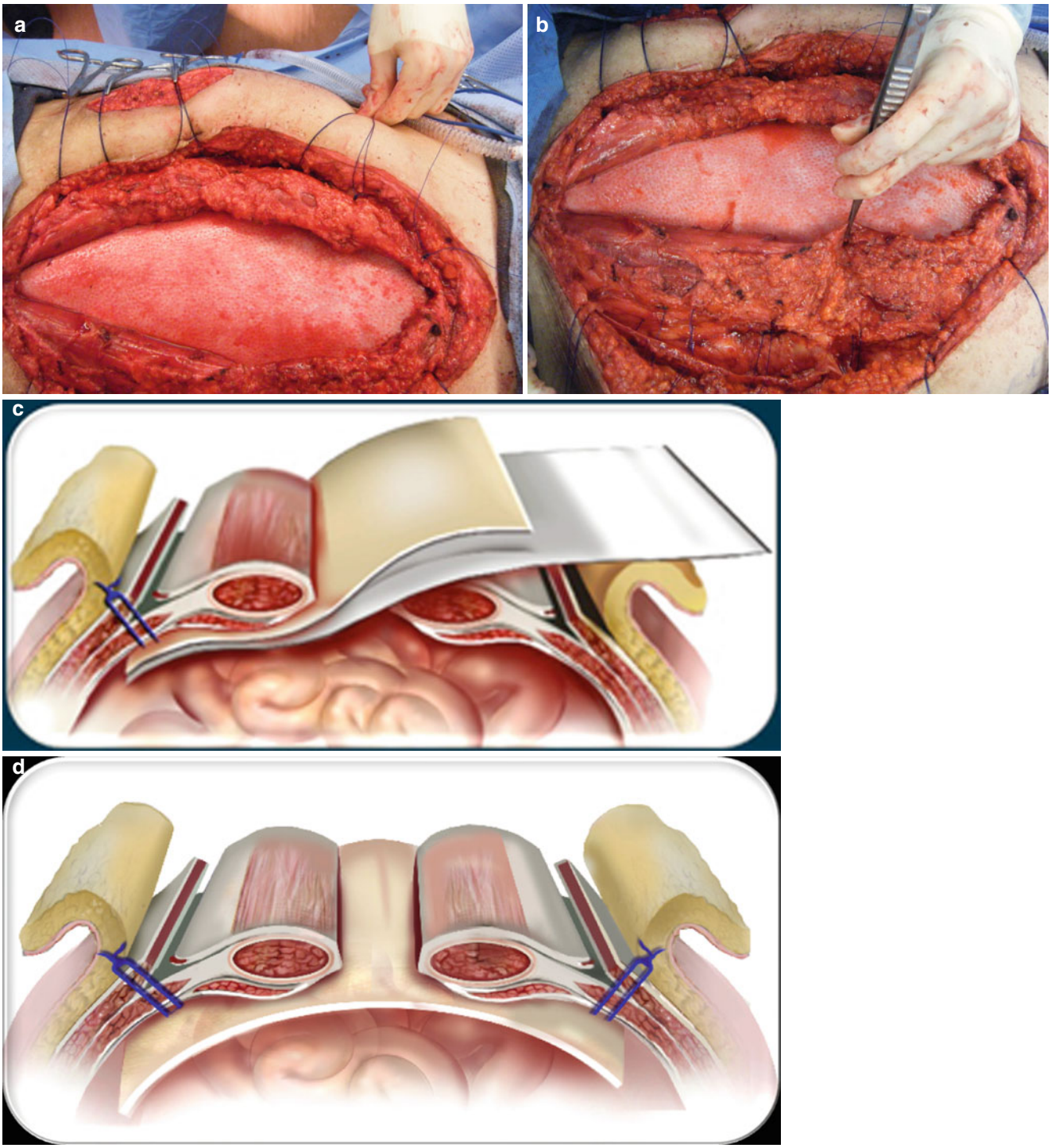


Fig. 16.9 (**a**, **b**) Abdominal wall reconstruction in a patient with right-side stoma. Component separation technique and underlay mesh were used. (**c**, **d**) Illustrator's demonstration of performing separation technique and placing the mesh as underlay. The mesh is fixed at least laterally to the separation of internal and external oblique muscles (Courtesy of LifeCell Corporation, Branchburg, NJ) (**e**) The fascia and the rectus muscle complex are approximated over the Strattice™ (LifeCell Corporation) mesh. (**f**) Final view of the abdominal reconstruction

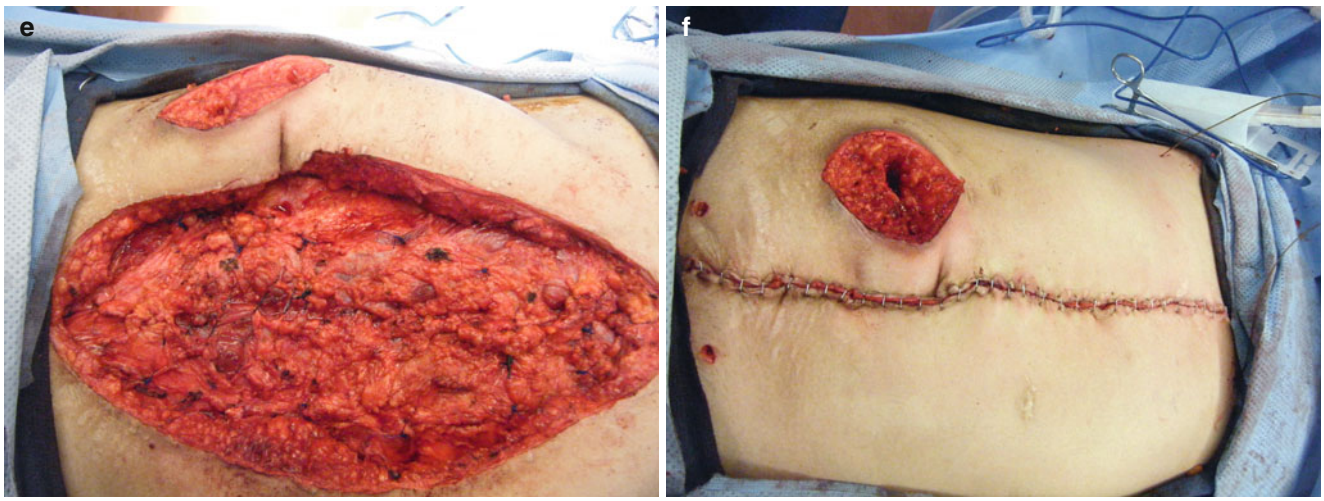


Fig. 16.9 (continued)

advancement of intact rectus myofascial units bilaterally, enabling us to close defects of up to 10 cm in the upper abdomen, 20 cm in the midabdomen, and 6–8 cm in the lower abdomen. The component separation technique is based on an enlargement of the abdominal wall surface by separating and advancing the muscular layers.

Choice of Mesh

By definition, patients with ECFs, EAFs, or stomas have contaminated wounds. Synthetic mesh has been used in the past, but it was associated with high rates of wound infection (often necessitating removal of infected mesh for source control of infection) and with other complications (such as newly created fistulas). Most recently, biologic mesh has become standard in high-risk patients with contaminated and dirty-infected wounds [23, 24]. Level I evidence, however, is needed.

In a recent study at our center, 60 patients underwent acellular dermal matrix (ADM) implantation for abdominal wall reconstruction from January 2006 through December 2009 [1]. Of the 60 patients, 4 were lost to follow-up. In the remaining 56 patients, we used two brands of ADM: AlloDerm® (LifeCell Corporation, Branchburg, NJ) in 38 patients (68 %) and Strattice™ (LifeCell Corporation) in 18 (32 %). A total of 9 patients had concomitant ECFs or EAFs. For the 9 patients with ECFs or EAFs, we used underlay placement in 4 (44 %) and interposition or bridge placement in 5 (56 %). We found that the abdominal wall reconstruction results in patients with versus without concomitant ECFs or EAFs did not statistically differ in terms of the rates of overall complications, of recurrence, and of infectious complications.

Others have also reported that ADM implantation can be safely used to repair large and complex ventral hernia defects in patients with clean-contaminated or dirty-infected wounds [23–29]. In our 2006–2009 study mentioned previously, of the 56 patients who underwent ADM implantation with either AlloDerm or Strattice, 35 had contaminated fields as defined by the presence of intra-abdominal or soft tissue infections, stomas, or fistulas [1]. Of those 35 patients, most of them—26 (74 %)—had grade 4 infections, per a hernia-grading system [27]. That recently created grading system (Table 16.1) is used to classify the risk for infectious complications to help surgeons decide on the technique and potentially on the mesh to be used. Grade 1 refers to a low risk for infections or complications in patients who have no history of wound infections; grade 2 indicates comorbidities such as smoking, diabetes, obesity, a suppressed immune system, and COPD; grade 3 refers to those with previously contaminated wound infections, stomas, or intraoperative violations of the GI tract; and grade 4 indicates infected mesh and septic foci. Obviously, grades 3 and 4 present serious medical and surgical challenges for the patient and for the health care team, whether led by a general surgeon, trauma surgeon, or plastic surgeon. But, even grade 2 means that patients may harbor a significant risk and need to be thoroughly evaluated preoperatively; otherwise, a significant problem could arise.

Our results suggest that biologic mesh implantation is a valid option for complex abdominal wall reconstruction in the high-risk trauma and acute care surgery population. One group has suggested staged care in patients with giant abdominal wall defects without the use of permanent mesh [28]. In that group's report on 274 patients, absorbable mesh implantation with component separation for definitive abdominal wall reconstruction provided effective temporary abdominal wall defect coverage with a low fistula rate. We have used this technique in the reconstruction of large abdominal defects.

But, most surgeons attempt to complete abdominal wall reconstruction at the time of hernia repair or at the time of takedown of ECFs or EAFs, even in contaminated fields.

Table 16.1 Hernia grading system

Grade 1	Grade 2	Grade 3	Grade 4
Low risk	Comorbidities	Previously contaminated wounds	Infected mesh
Low risk for complications	Smoking	Previous wound infections	Infected mesh
No history of wound infections	Obesity	Stomas present	Septic dehiscence
No significant comorbidities	Diabetes	Violations of the gastrointestinal tract	
	Immunosuppression		
	COPD		

Reprinted with permission of Elsevier from DiCocco et al. [30]
COPD chronic obstructive pulmonary disease

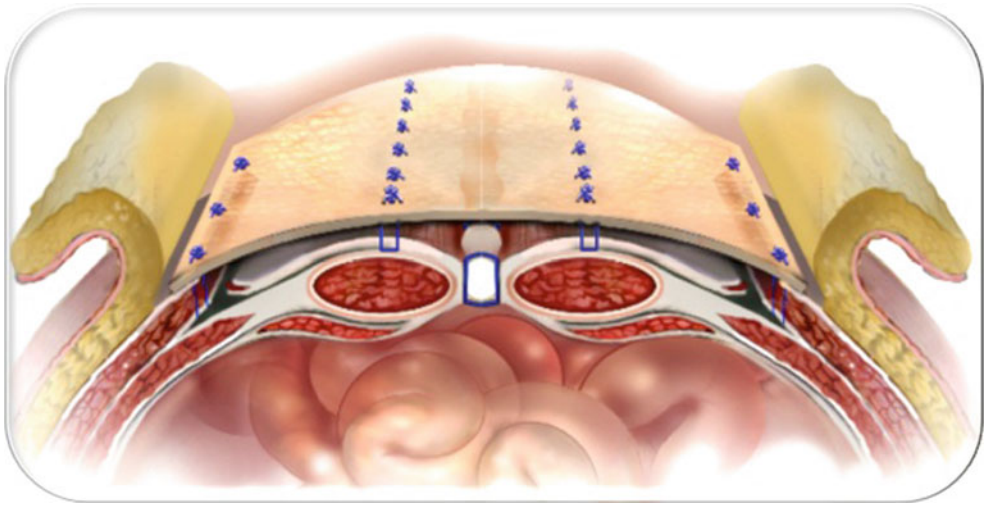


Fig. 16.10 Onlay mesh. Illustrators view (Courtesy of LifeCell Corporation, Branchburg, NJ)

In our practice, we aim to complete the definitive procedure in a single operation. On occasion, we have used the principle of damage control, returning the next day or so to complete, if at all possible, the operation. Since 2005, in all of our patients with clean-contaminated or dirty-infected wounds, we have used biologic mesh, primarily AlloDerm and Strattice. Our experience with other biologic mesh products, such as Surgisis® (Cook Biotech, West Lafayette, IN) and Veritas® (Synovis, St. Paul, MN), has not been good; we have not used Surgisis or Veritas for the last 5–6 years.

When there is native tissue to cover the mesh, we use four to five drains that stay in for 10–15 days. With underlay placement, we use one large drain between the mesh and fascia, and then we use three to four drains over the fascia and under the skin and subcutaneous tissue; to avoid drain displacement, we fix all of the drains to tissue with fine chromic sutures.

Mesh Placement

In our practice, the three most common techniques used to place mesh during abdominal wall reconstruction are onlay placement (Fig. 16.10), underlay placement (Fig. 16.9c, d), and interposition or bridge placement (Fig. 16.11a, b, c). Either open or laparoscopic surgical techniques can be used to repair abdominal wall defects, but in patients with ECFs or EAFs, the open approach is more common.

Onlay Placement

Technically, onlay placement (Fig. 16.10) is the easiest way to place mesh. We used this technique at the beginning of our practice; we still use it when we are able to approximate the abdominal wall edges without any major dissection. The key element of this approach is fixing the mesh both laterally and over the edge of the midline. We prefer fixing mesh to fascia using absorbable sutures, either interrupted or continuous (Fig. 16.11a, b, c). The main objective is to reestablish closure. We use three or four large, closed-suction drains ([19] French [8]) under the subcutaneous tissue, and we keep them in until the individual drain output is less than 25 ml over 24 h.

Underlay Placement

In our practice, underlay placement (Fig. 16.9a–f) has now become the main technique of mesh placement. It is more involved, but once it is learned and perfected, it does not add significant operative time. We prefer underlay placement to minimize the incidence of seromas associated with the repair of abdominal wall defects. The key element of this technique is freeing the abdominal wall from any adhesions, as far laterally as possible, both on the posterior and the anterior aspect.

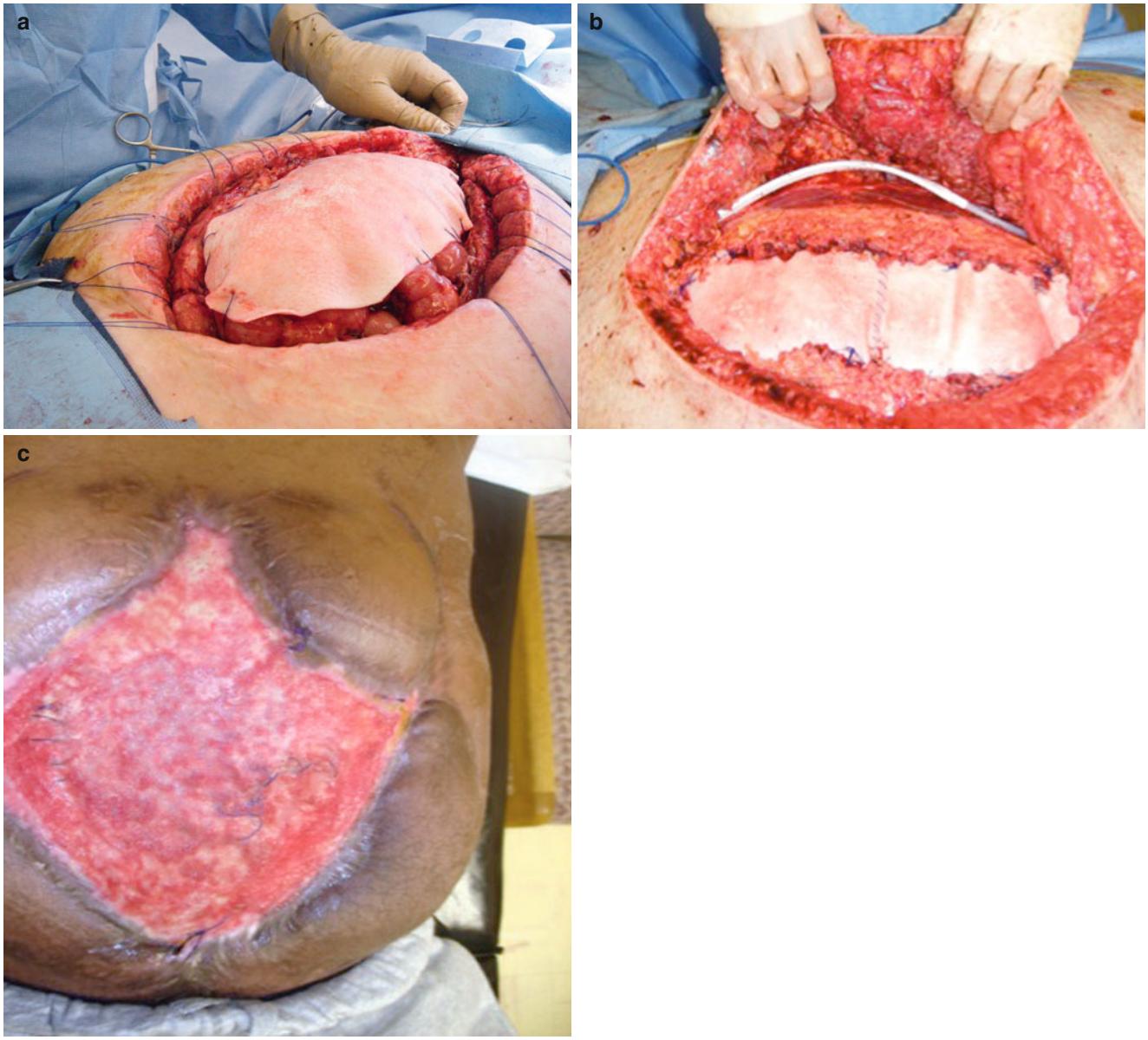


Fig. 16.11 (**a**). Interposition Strattice graft in a patient who sustained a gunshot wound and was managed with open abdomen. (**b**) Interposition graft in a patient who, despite component separation, required bridging with a graft. Skin and subcutaneous tissue were adequate to cover the graft over the drains. (**c**) Same patient as in Fig. 16.2. Skin and subcutaneous necrotic tissue were removed, and AlloDerm was eventually covered with skin graft

Placement of the interrupted sutures should ensure complete stretching of mesh once sutures are tight (Fig. 16.11a–c). Suture placement techniques vary, but we prefer the parachuting technique and the use of direct vision at all times. Our direct-vision parachuting technique minimizes the potential for bowel injury during fixing of mesh on the abdominal wall. If lateral component release is used, we prefer placing sutures in the anterior abdominal wall as far laterally as possible; clearly, the surgeon must include the medial edge of the external oblique fascia. Doing so is an important technical step: It prevents bulging laterally at the release component site, and the patient might think bulging is a new hernia.

Interposition or Bridge Placement

When the abdominal wall has lost its domain and the surgeon cannot bring together its medial edges (because the wall has been either removed or retracted laterally completely), then the only remaining option is to use mesh as a bridge (Fig. 16.11a–c). The surgeon must ensure that the suture bites are placed at least 2 cm into the muscles and fascia. If at all possible, the surgeon must avoid tacking the mesh on

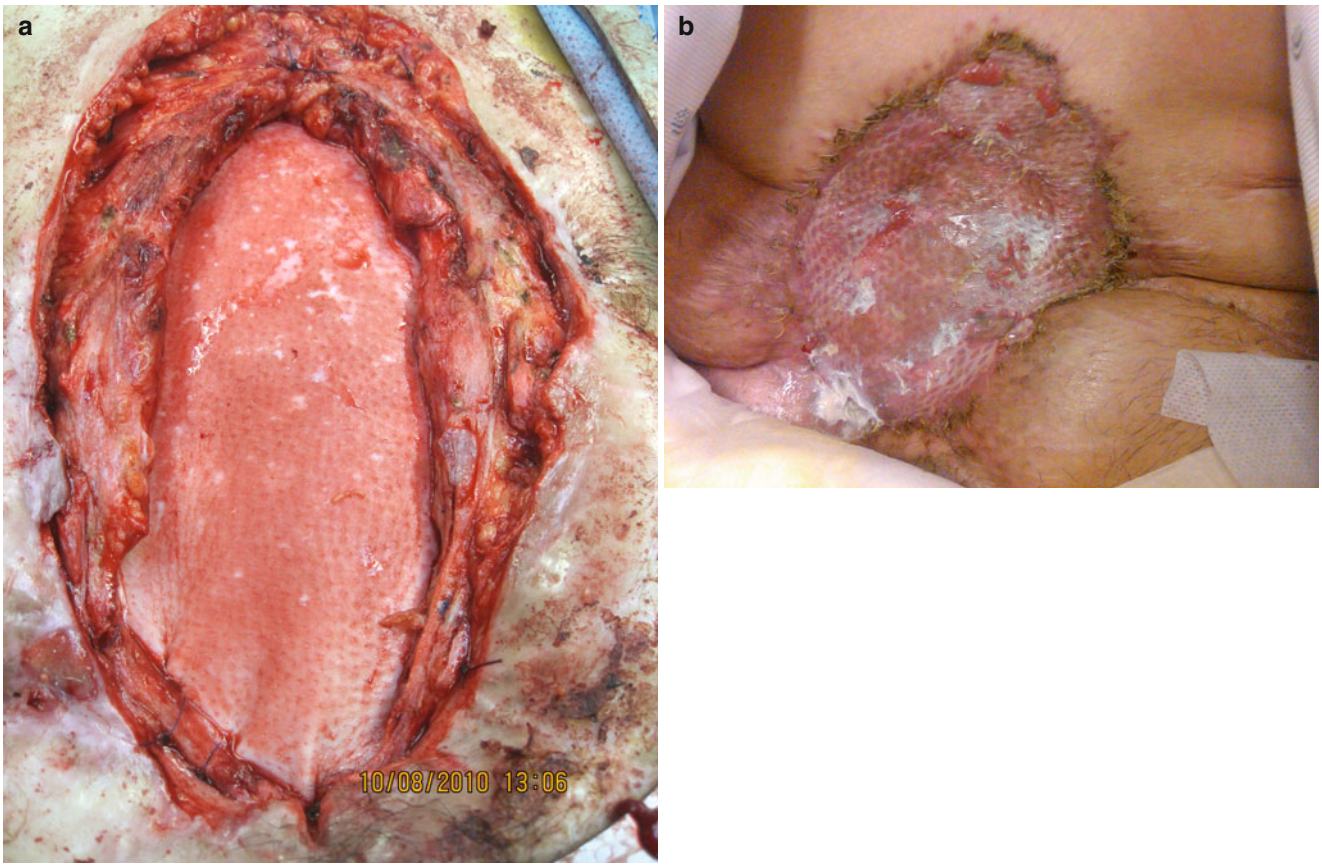


Fig. 16.12 (**a**) A 41-year-old male managed with interposition graft who has lost abdominal wall domain, including skin and subcutaneous tissue. (**b**) After a few weeks of being managed with vacuum-assisted closure (VAC), skin graft was applied successfully

the edge of the fascia, given the risk of herniation or suture failure. Closed-suction drainage may prevent seromas, which have the potential to become infected and thereby to jeopardize the integrity of the closure. When mesh is used as a bridge and when there is no skin or subcutaneous tissue to cover the mesh, then we use wound VAC with continuous irrigation, which keeps the mesh moist and speeds the process of granulation for later skin grafting (Fig. 16.12a, b). Detailed operative notes are mandatory and should be written by the most senior surgeon (Fig. 16.13).

Step 8: P = Postoperative Care

Postoperative care of patients after major exploratory laparotomy with takedown of fistulas and abdominal wall reconstruction is as complex as the operation itself. Such patients require continuation of parenteral nutrition until full return of GI tract function, at which time they may be able to resume full oral intake. Postoperatively in our practice, we prefer to give patients massive doses of vitamin C: 2 g intravenously every 4 h for at least 1 week. We also administer vitamin E, zinc, selenium, and, when appropriate, vitamin A beyond the standard doses in total parenteral nutrition. The most common complications include wound infections and other surgical site complications (20–45 %), hernia recurrence (up to 20 %), fistula recurrence (up to 47 %, depending on the type of mesh used), small bowel obstructions, and pain. The real complication rate, however, can be extremely high, up to 82 % [29, 31].

Step 9: L = Long-Term Follow-Up

All patients undergoing complex reconstruction require long-term follow-up. We suggest following up these patient at least yearly. Data for long-term effects of these complex abdominal wall reconstructions are lacking, however [29]. Based on the surgical technique used in the repair of the hernia defect, the hernia recurrence rate could be as high as 64 % at 10 years when mesh is not used [31]. Others [32] have demonstrated that Mersilene™ (Ethicon) mesh has a greater incidence of ECF formation and a recurrence rate that is three times greater. In a study of long-term follow-up of patients with abdominal wall reconstruction of planned hernia after major trauma, the hernia recurrence rate was

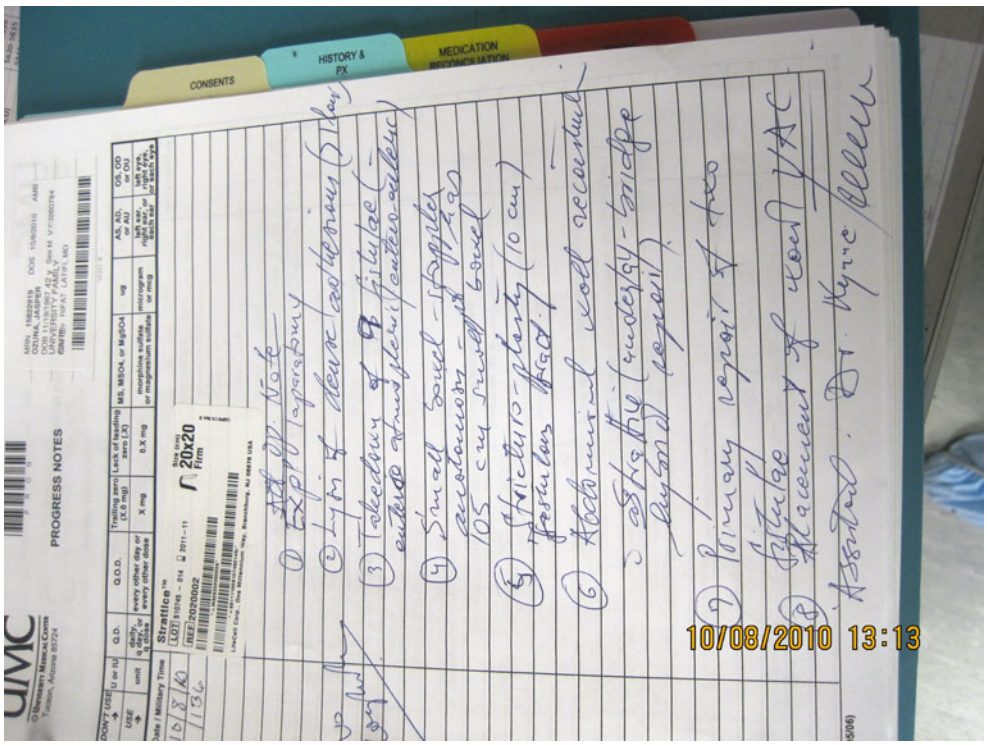


Fig. 16.13 Detail of handwritten operative notes

14 % [30]. Lower recurrence rates of 5 % were observed when the modified component separation technique with or without mesh was employed. Increased BMI and female gender were associated with recurrence. When a modified Rives-Stoppa repair was used, the results were much better, despite the fact that the majority of the patients (60 %) had significant comorbidity, and 30 % of these patients had one or more incisional hernia recurrence. The hernia recurrence rate in this group of this patient population was 5 % [33]. A modified onlay technique for the repair of complicated incisional hernias with a mean follow-up time of 64 months had a 16 % hernia recurrence rate [34].

Summary

Surgical management of abdominal wall defects, including ECFs or EAFs, is often associated with major hernias and other complexities. Careful planning and advanced surgical techniques are required, often involving the use of biologic mesh or composite tissue transfer. Treatment of ECFs in patients with large abdominal wall defects is challenging, but with proper techniques, results can be excellent. See Fig. 16.14 for an algorithm that will help with decisions regarding what type of repair should be used in abdominal wall reconstruction. Biologic mesh is the mesh of choice in such patients [35].

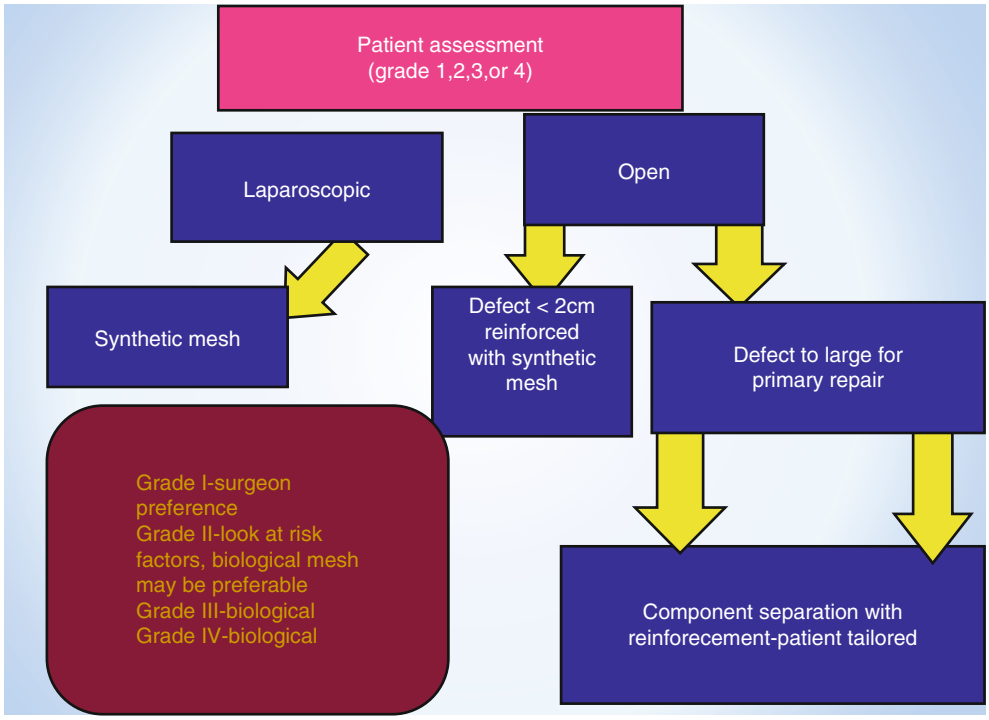


Fig. 16.14 Algorithm for use when deciding what type of repair should be implemented in abdominal wall reconstruction

References

1. Latifi R, Gustafson M. Abdominal wall reconstruction in patients with enterocutaneous fistulas. Eur J Trauma Emerg Surg. 2011; 37:241–50.
2. Latifi R, Joseph B, Kulvatunyou N, Wynne JL, O'Keeffe T, Tang A, et al. Enterocutaneous fistulas and a hostile abdomen: reoperative surgical approaches. World J Surg. 2012;36(3):516–23.
3. Becker HP, Willms A, Schwab R. Small bowel fistulas and the open abdomen. Scand J Surg. 2007;96(4):263–71.
4. Visschers RG, Olde Damink SW, Winkens B, Soeters PB, van Gemert WG. Treatment strategies in 135 consecutive patients with enterocutaneous fistulas. World J Surg. 2008;32(3):445–53.
5. Marshall C, Maier RV, Jimenez M, Dellinger EP. Source control in the management of severe sepsis and septic shock: an evidence-based review. Crit Care Med. 2004;32(11):S513–26.
6. Lloyd DAJ, Gabe SM, Windsor ACJ. Nutrition and management of enterocutaneous fistula. Br J Surg. 2006;93:1045–55.
7. Campos AC, Andrade DF, Campos GM. A multivariate model to determine prognostic factors in gastrointestinal fistulas. J Am Coll Surg. 1999;188:483–90.
8. Li J, Ren J, Zhu W, Yin L, Han J. Management of enterocutaneous fistulas: 30-year clinical experience. Chin Med J (Engl). 2003;116: 171–5.
9. Hollington P, Mawdsley J, Lim W, Gabe SM, Forbes A, Windsor AJ. An 11-year experience of enterocutaneous fistula. Br J Surg. 2004;91:1646–51.
10. Edmunds LH, Williams GM, Welch CE. External fistulas arising from the gastrointestinal tract. Ann Surg. 1960;152:445–71.
11. Berry SM, Fischer JE. Classification and pathophysiology of enterocutaneous fistulas. Surg Clin North Am. 1996;76:1009–18.
12. Evenson AR, Fischer JE. Current management of enterocutaneous fistula. J Gastrointest Surg. 2006;10:455–64.
13. Soeters PB, Ebeid AM, Fischer JE. Review of 404 patients with gastrointestinal fistulas: impact of parenteral nutrition. Ann Surg. 1979;190:189–202.
14. Gregor S, Maegele MMD, Sauerland S, Krahn JF, Peinemann F, Lange S. Negative pressure wound therapy: a vacuum of evidence? Arch Surg. 2008;143(2):189–96.
15. Rodriguez ED, Bluebond-Langner R, Silverman RP, Bochicchio G, Yao A, Manson PN, et al. Abdominal wall reconstruction following severe loss of domain: the R. Adams Cowley Shock Trauma Center algorithm. Plast Reconstr Surg. 2007;120(3):669–80.
16. Lord Jr JW, Howes EL, Jolliffe N. The surgical management of chronic recurrent intestinal obstructions due to adhesions. Ann Surg. 1949;129(3):315–22.
17. Schmidt BJ, Hinder RA. Abdominal adhesions: to lyse or not to lyse? J Clin Gastroenterol. 2005;39:87–8.
18. Joyce MR, Dietz DW. Management of complex gastrointestinal fistula. Curr Probl Surg. 2009;46:384–430.
19. Schecter WP, Hirshberg A, Chang DS, Harris HW, Napolitano LM, Wexner SD, et al. Enteric fistulas: principles of management. J Am Coll Surg. 2009;209(4):484–91.
20. Martine JL, Luque-de-Leon E, Juan Mier AE, Blanco-Benavides R, Robledo F. Systematic management of postoperative enterocutaneous fistulas: factors related to outcomes. World J Surg. 2008;32:436–44.
21. Albanese A. Eventracion mediana xifoumbilical gigante. Metodo Para Su tratamiento. Rev Asoc Med Argent. 1951;15–30:376–8.
22. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86(3):519–26.
23. Patton Jr J, Berry S, Kralovich KA. Use of human acellular dermal matrix in complex and contaminated abdominal wall reconstructions. Am J Surg. 2007;193:360–3.
24. Awad S, Baumann D, Bellows C et al. Prospective multicenter clinical study of single-stage repair of infected or contaminated abdominal incisional hernias using Strattice™ reconstructive tissue matrix (RICH Study). Poster presentation American College of Surgeons Clinical Congress, Washington, DC, 3–7 Oct 2010
25. Butler CE, Langstein HN, Kronowitz SJ. Pelvic, abdominal, and chest wall reconstruction with AlloDerm in patients at increased risk for mesh-related complications. Plast Reconstr Surg. 2005;116(5):1263–75. discussion 1276–7.

26. Diaz Jr JJ, Guy J, Berkes MB, Guillamondegui O, Miller RS. Acellular dermal allograft for ventral hernia repair in the compromised surgical field. Am Surg. 2006;72(12):1181–7; discussion 1187–8.
27. The Ventral Hernia Working Group, Breuing K, Butler C, Ferzoco S, Franz M, Hultman C, et al. Incisional ventral hernias: review of the literature and recommendations regarding the grading and technique of repair. Surgery. 2010;148(3):544–58.
28. Jernigan TW, Fabian TC, Croce MA, Moore N, Pritchard FE, Minard G, et al. Staged management of giant abdominal wall defects: acute and long-term results. Ann Surg. 2003;238(3):349–55; discussion 355–7.
29. Peralta R, Latifi R. Long term outcomes of abdominal wall reconstruction. What are the real numbers? World J Surg. 2012;36(3): 534–8.
30. DiCocco JM, Magnotti LJ, Emmett KP, et al. Long-term follow-up of abdominal wall reconstruction after planned ventral hernia: a 15-year experience. J Am Coll Surg. 2010;210:686–95.
31. Luijendijk RW, Hop WJC, van den Tol MP, et al. A comparison of suture repair with mesh repair for incisional hernia. N Eng J Med. 2000;343:392–8.
32. Leber GE, Garb JL, Alexander AI, et al. Long-term complications associated with prosthetic repair of incisional hernias. Arch Surg. 1998;133:378–82.
33. Igbal CW, Pham TH, Joseph A, et al. Long term outcome of 254 complex incisional hernia repairs using the modified Rives-Stoppa technique. World J Surg. 2007;31:2398–404.
34. Poelman MM, Langenhorst JF, Schellenkens JF, et al. Modified onlay technique for the repair of the more complicated incisional hernias: single-centre evaluation of a large cohort. Hernia. 2010; 14:369–74.
35. Szczerba SR, Dumanian GA. Definitive surgical treatment of infected or exposed ventral hernia mesh. Ann Surg. 2003;237(3): 437–41.

Abdominal Wall Closure in Recipients of Intestinal and Multivisceral Transplants

17

Gennaro Selvaggi, David Mark Levi, and Andreas G. Tzakis

Introduction

Closure of the abdominal wall at the end of a complex operation such as intestinal transplantation is often a challenging feat. Multiple factors lead to loss of abdominal domain and adequate volume in such recipients: age and size mismatch between graft and recipient; previous abdominal surgeries with massive bowel resections; presence of enterocutaneous fistulas that need to be excised; presence of desmoid tumors or similar within the abdominal wall that need to be resected as well; or intestinal graft swelling at the end of the surgical procedure.

In addition, stomas and enteral access devices such as gastrostomy tubes are created at the end of the transplant, further increasing the amount of intact abdominal wall domain necessary to complete the surgery.

Despite the use of graft size reduction techniques or a mismatch of donor to recipient body weight in favor of the recipient, such strategies cannot always be utilized in the clinical practice.

Multiple techniques can be used to close the abdominal wall in such patients. Primary closure can be accomplished with the use of component separation techniques or rotational/advancement flaps [1]. Secondary closure is possible with the development of a granulation tissue bed followed by skin grafting [2]. A variety of mesh types can be used as well to bridge the gap between the fascial edges. Both artificial (absorbable and nonabsorbable) and biological meshes have been utilized [3–5]; all have their own advantages (easy availability) and disadvantages (danger of infections, fistula formation, and cost).

Two recent techniques have been described that can facilitate closure and restore the integrity of the abdominal compartment in these patients: abdominal wall transplantation and allograft fascia of the rectus muscle [6, 7].

This chapter focuses on a description of these last techniques, the indications for use, description of the procurement, and for the abdominal wall transplant, discussion of some ethical considerations.

Abdominal Wall

Graft Retrieval

The abdominal wall graft is a composite tissue transplant that is vascularized and denervated, with full-thickness layer of the anterior abdominal wall retrieved from cadaveric donors [6]. It is composed of the rectus abdominis muscles, their investing fascia, underlying peritoneum, overlying subcutaneous tissue, and skin. The anatomic boundaries of the graft are the subcostal margin superiorly, the symphysis pubis inferiorly, and the edges of the rectus abdominis muscles laterally. The size of the graft varies depending on the size of the donor. The blood supply is based on the inferior epigastric vessels, left in continuity with the femoral and iliac vessels. This provides a long vascular pedicle and facilitates implantation and positioning of the graft in the recipient. An alternative option is the use of the inferior epigastric vessels bilaterally alone, with implantation performed by microsurgical technique [8].

The abdominal wall graft is obtained from cadaveric donors after brain death, during standard multiorgan procurement procedure. Specific consent is obtained from the donor's family for its procurement and transplantation. Median sternotomy splits in bilateral subcostal incisions which are then

G. Selvaggi, MD
Department of Clinical Surgery,
University of Miami, Miami, FL, USA

Department of Liver and GI Transplant,
University of Miami, 1801 NW 9th Ave.,
Miami, FL 33136, USA
e-mail: gselvaggi@med.miami.edu

D.M. Levi, MD • A.G. Tzakis, MD, PhD, Dhe Mult (✉)
Department of Surgery,
Carolinas Medical Center,32861,
Charlotte, NC 28232, USA
e-mail: david.levi@carolinashealthcare.org; tzakisa@ccf.org

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_17, © Springer Science+Business Media New York 2013

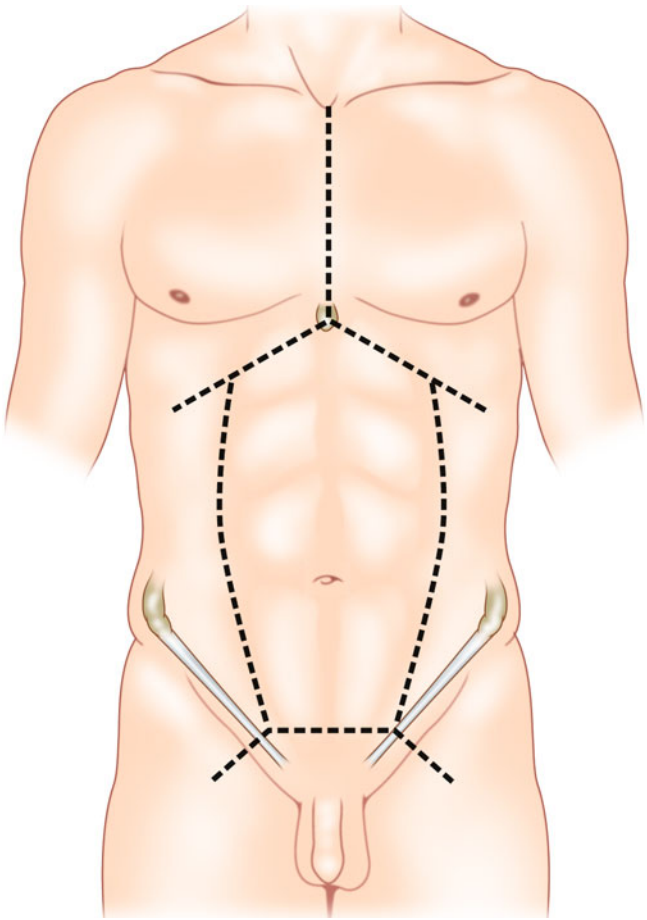


Fig. 17.1 Lines of incision for abdominal wall graft retrieval

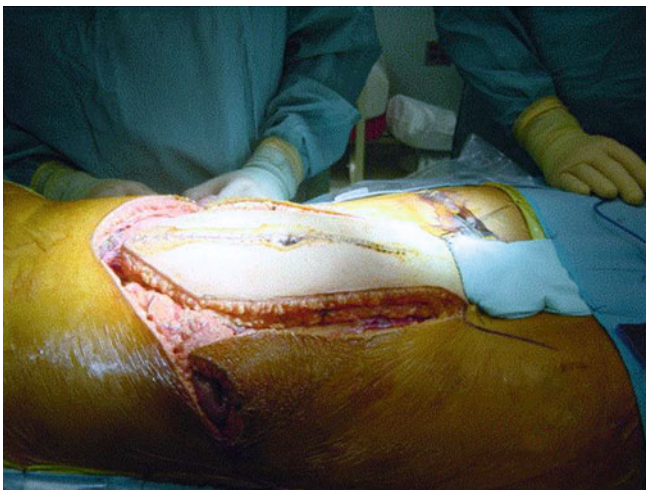


Fig. 17.2 Detail of intraoperative retrieval of abdominal wall graft

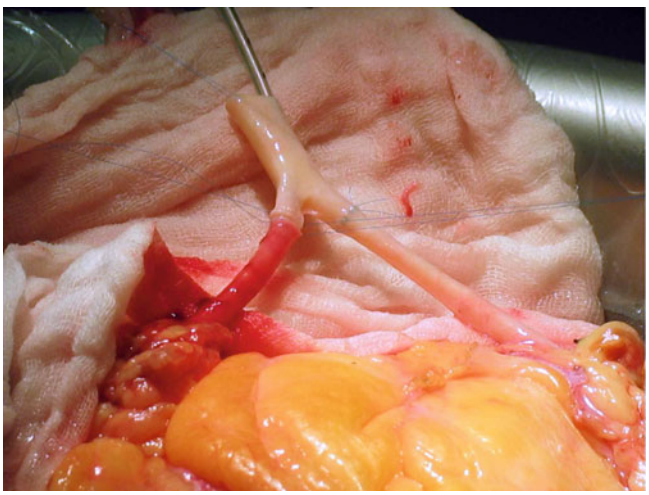


Fig. 17.3 Arterial reconstruction of abdominal wall graft. The two external iliac arteries are connected to a Y graft for single implantation

carried down as parallel longitudinal incisions along the lateral edges of both rectus muscle sheaths from the costal margin to the inguinal ligaments (Fig. 17.1). Inferiorly, a transverse suprapubic incision joins the two longitudinal incisions. The femoral vessels are exposed bilaterally. The graft remains attached to the vascular pedicles and is wrapped with cold towels (Fig. 17.2). All other organs are flushed with cold preservation solution and removed. The aortic cannula is then reinserted distally, and the external femoral arteries are clamped. The abdominal wall graft is then flushed with cold preservation solution. The abdominal wall graft is then removed in continuity with the common femoral and iliac vessels. Instead of cleaning the major vessels, the graft is procured with a large amount of adjoining pelvic soft tissue. The graft is then transported in cold preservation solution.

Implantation

At the back table, the abdominal wall graft is prepared by cleaning the base of the iliac vessels but leaving the soft tissue around the inferior epigastric vessels takeoff to avoid injury. The internal iliac and distal common femoral vessels are ligated. If the vascular pedicle is too redundant, the external and common iliac vessels can be shortened and reconstructed (Figs. 17.3 and 17.4). The abdominal wall graft is implanted to the recipient's common iliac artery and vein at the end of the intestinal transplant, in a procedure similar to a renal graft (Fig. 17.5). Alternatively, the distal aorta and inferior vena cava can be used as a site of anastomosis. On reperfusion, bleeding from the skin edges or the rectus muscle confirms good perfusion of the graft.

The abdominal wall graft is finally sutured to the recipient's abdominal wall during closure at the level of the fascia and the skin. The graft can be rotated up to 90° to best cover the abdominal wall defect of the recipient; however, in such cases, care must taken to avoid kinking or twisting of the vessels.

Timing

In most cases, the abdominal wall graft is procured from the same donor as the intestinal graft, and the transplant occurs

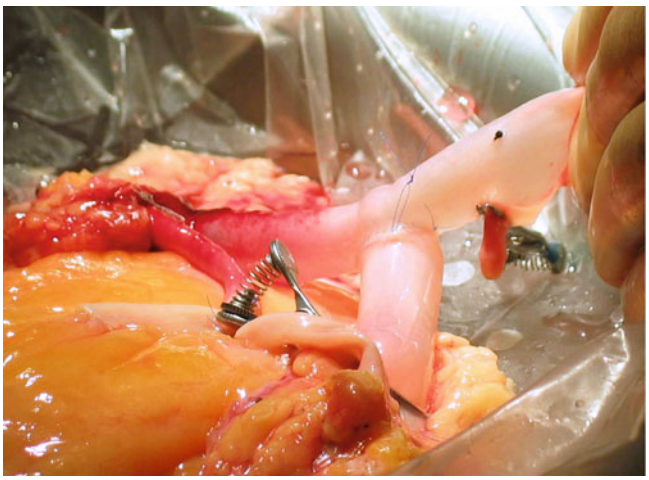


Fig. 17.4 Venous reconstruction of abdominal wall graft. The two external iliac veins are connected to a Y graft for single implantation

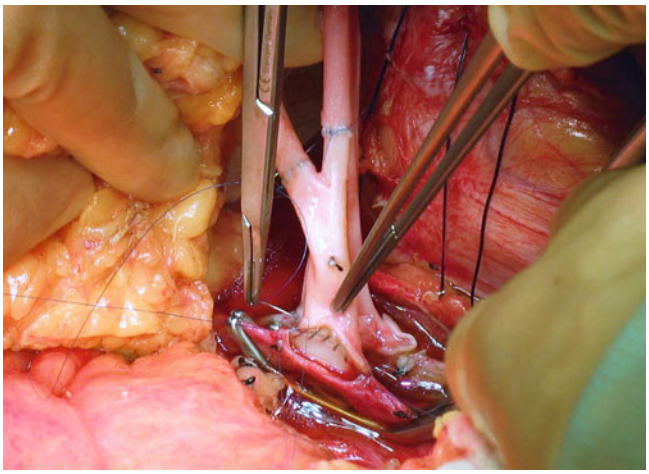


Fig. 17.5 Implantation of the abdominal wall graft vessels onto the iliac vessels of the recipient

at the end of the intestinal transplant procedure. In some cases, however, especially in the pediatric population, the initial donor for the abdominal wall graft is small in size. In such cases, the abdominal wall can be procured at a later time from a separate cadaveric donor. This time delay can allow the clinicians to ascertain that the abdomen cannot indeed be closed primarily or by the other previously mentioned techniques. If an abdominal wall graft is still required, a larger donor can be used from whom a larger abdominal wall graft can be retrieved.

Monitoring of the Graft

In addition to clinical exam, the graft can be monitored, in the same manner as all free flap grafts, by Doppler analysis; during this analysis, the probe is placed at the inferior borders of the graft, and arterial and venous flow is detected. Any signs of erythema or maculopapular

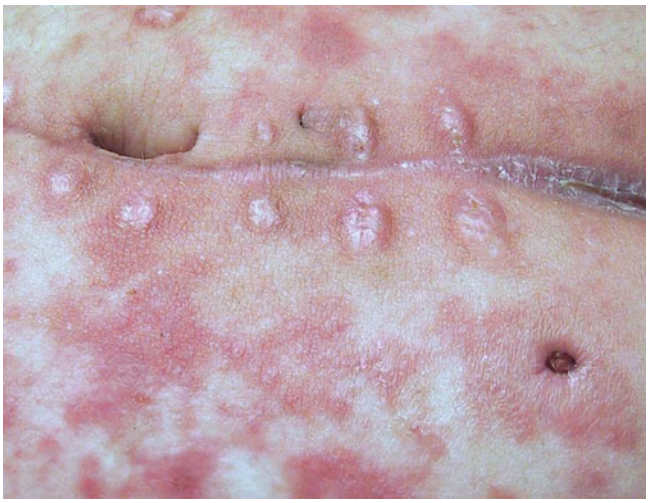


Fig. 17.6 Details of skin of abdominal wall graft during rejection episode. A maculopapular rash is visible. Punch biopsy site is also shown

eruption should prompt a skin biopsy to confirm the diagnosis of rejection (Fig. 17.6). Pathological reading of the punch biopsy is performed according to previously published guidelines [9, 10].

Immunosuppression/Rejection

All recipients of an abdominal wall graft are intestinal graft recipients and thus receive significant doses of immunosuppressive medications because of the high immunogenic nature of the intestinal graft. The presence of the abdominal wall graft does not require an increase or change in the immunosuppressive protocols. Induction immunosuppression is given with alemtuzumab (Campath 1-H®, Genzyme, Cambridge, MA), tacrolimus (Prograf®, Astellas Pharma US, Deerfield, IL) and no maintenance corticosteroids [6]. Campath is administered intravenously on postoperative days 1 and 4. Prograf is usually started in the early postoperative period, first intravenously, then enterally as gastrointestinal function recovers.

Rejection of the abdominal wall graft manifests clinically as an erythematous skin rash that spares the surrounding skin of the native abdomen. Punch biopsy of the skin can confirm the diagnosis, which is characterized at a microscopic level by perivascular, inflammatory cell infiltrates in the superficial dermis, with the presence of reactive lymphocytes and red cell extravasation at higher power [9]. Rejection of the wall graft responds well to corticosteroid treatment. Interestingly, abdominal wall graft rejection does not always correlate to simultaneous episodes of intestinal rejection; rather, the two grafts seem to act independently. Last, few episodes of rejection have been observed with the full abdominal wall graft, perhaps signifying a lesser degree of immunogenicity of the composite graft as compared to a skin graft alone.

Results

Overall, two centers have published clinical series of abdominal wall transplants: At the University of Miami, we described 12 cases, and the University of Bologna described 3 cases [6–8]. At the University of Miami, the abdominal wall graft was implanted using the iliac vessels; in the second series from Italy, the abdominal wall was implanted with a microsurgical technique using the inferior epigastric vessels. Although the overall survival of the patients was only 5/15, none of them passed away from complications related to the abdominal wall graft. Two grafts in the University of Miami series had to be removed because of vascular thrombosis. Four patients experienced rejection of the skin on the abdominal graft, which was successfully treated with steroid boluses.

Ethical Considerations

In contrast to other composite tissue transplantation, such as hand and face transplantation, abdominal wall transplantation has not generated the same magnitude of controversy or concern. First, it has been described in few subjects. Second, it is reserved for patients who are already undergoing another "lifesaving" transplant, namely, intestinal transplant; because of that, abdominal wall recipients are already going to be subjected to the risks of immunosuppression, and their immunosuppression regimen is not going to be altered because of the inclusion of the abdominal wall. Last, the purpose of the abdominal wall graft, although important, is relatively simple because its main purpose is to remain viable and cover the abdominal viscera, without need for functionality or innervations such as in a hand or face transplant.

Fascia of Rectus Muscle

The experience from the use of the abdominal wall transplant showed that the layer that conferred strength to the closure was based on the rectus muscle sheaths, anterior and posterior. As an evolution of the use of the vascularized abdominal wall, the avascular fascia of the rectus muscle could be used as an allogeneic biological mesh to close large abdominal wall defects when it is still possible to mobilize the skin and subcutaneous tissue of the abdomen for primary closure [7].

The first report of such a technique was in 2007, combining the results of three transplant centers for a total of 16 cases. Since then, other reports have been published, with one case using the posterior leaflet of the rectus muscle in continuity with the round ligament of the liver in a liver transplant recipient [11].

The rationale for the use of such tissue is that it is going to be used in recipients of solid organ transplants who are already receiving immunosuppression; in addition, the tissue is avascular and poorly cellular, making it poorly immunogenic. The fascia can be easily retrieved at no additional cost during the retrieval of the other solid organs; it does not require flushing with preservation solution because it is not a vascularized graft, and it can be easily stored until time of implant.

Graft Retrieval

At the beginning of the surgery in cadaveric donors after brain death, a cruciate incision is carried onto the skin, and flaps of skin and subcutaneous tissue are mobilized all the way to the subcostal line, the pubic symphysis, and the lateral wall of the abdomen. The incision is then carried to divide the abdominal wall's muscular-aponeurotic layer similar to an abdominal wall graft, bilateral subcostal laterally to the edge of the rectus muscle, then down longitudinally to the pelvic brim. The flap is reflected inferiorly after dividing the round ligament to the liver and is wrapped in a wet towel. At the end of retrieval of all organs, the lower part of the abdominal wall is divided, and the tissue is removed and placed in a bag containing preservation solution (Fig. 17.7).

At the back table, the rectus muscle is sharply divided and removed from the anterior and posterior leaflets of the fascia, thus creating a two-layer fascial surface that is lined by peritoneum on the posterior layer (Fig. 17.7).

Storage and Implantation

The fresh fascia can be used immediately at the end of the transplant procedure, or it can be fresh frozen according to standard protocol by the local tissue bank. If used fresh, the fascia can be stored at 4 °C for up to 24 h. If fresh frozen fascia is used, it can be thawed in warm saline solution just before use.

During the implantation procedure, the fascia is trimmed to size according to the abdominal wall defect in the recipient. The anterior and posterior leaflets can be sutured together at the edges for easier implantation. The fascia is sutured under some tension to the edges of the native abdominal wall fascia using interrupted sutures (Fig. 17.8). The recipient's skin and subcutaneous layers are mobilized laterally as far as possible and then brought together for primary closure. It is advisable to leave a drain in the subcutaneous space for a few days to drain eventual fluid collections.

Timing

The rectus muscle fascia can be used at the end of the transplant procedure or a few days later when the edema of the intestinal graft has subsided; alternatively, the fascia

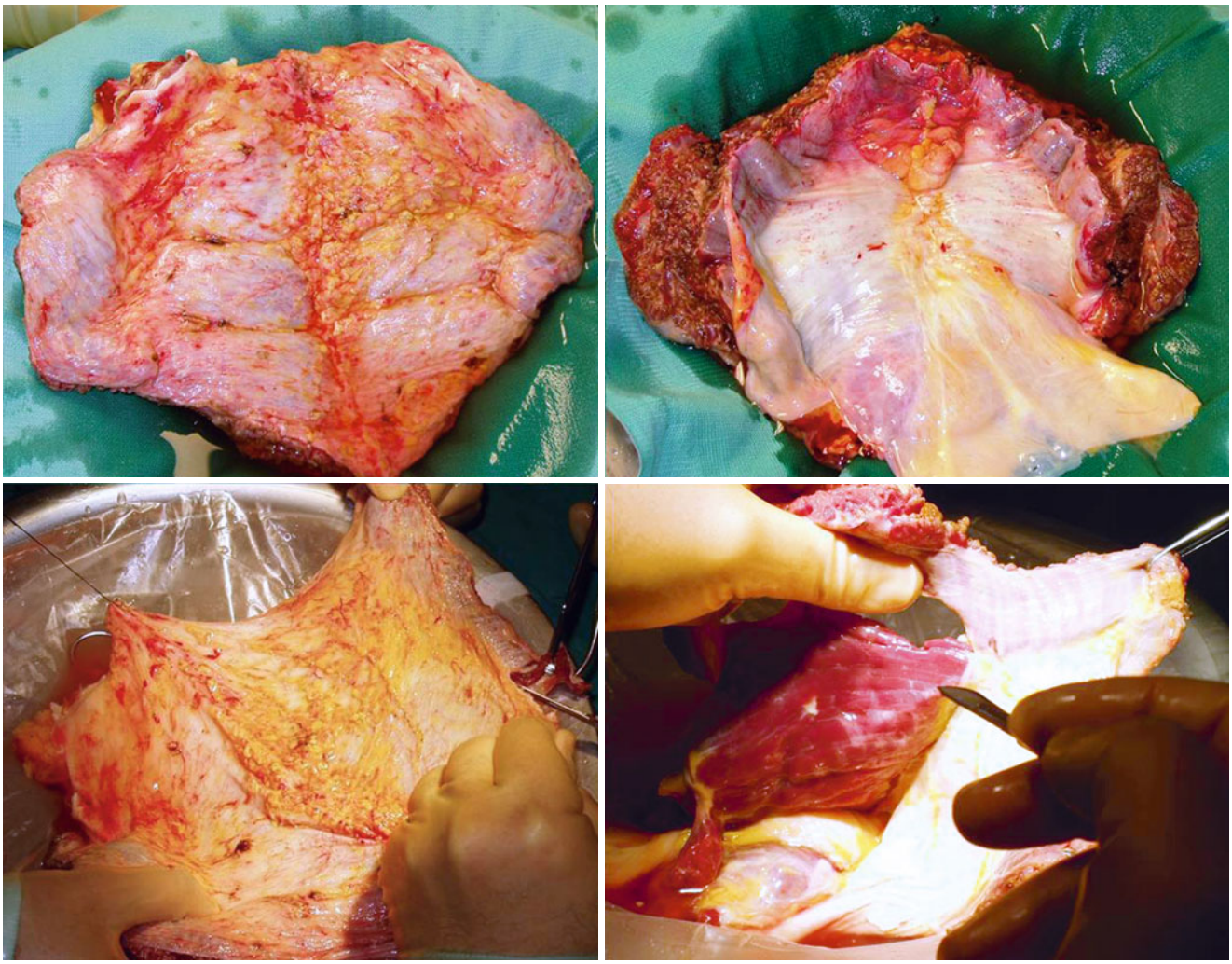


Fig. 17.7 Preparation of fascia of the rectus muscle. *Top left*: The fascia is retrieved from a cadaver donor. *Top right*: Posterior aspect with peritoneal layer. *Bottom left*: The external oblique muscle is sharply divided. *Bottom right*: The rectus muscle is sharply divided from the anterior and posterior aponeurotic fascia

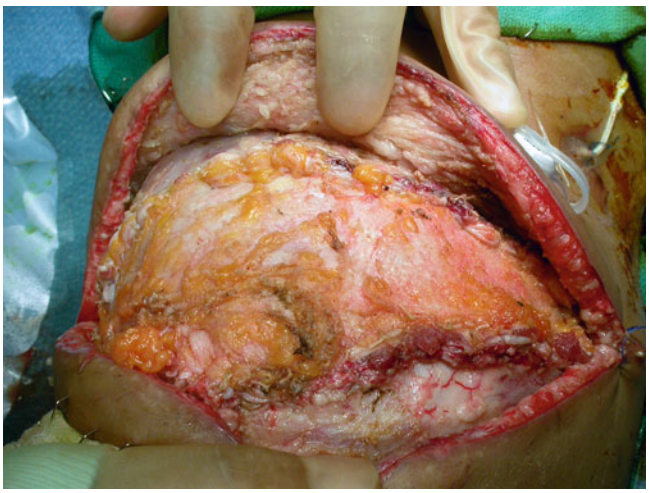


Fig. 17.8 Rectus muscle fascia sutured to the edges of a large abdominal wall defect in a pediatric recipient of a multivisceral transplant. Skin and subcutaneous flaps have been raised to allow primary closure

can be placed on the day of transplant and subsequently reduced in size a few days later when bowel edema has resolved to achieve a tighter closure. We have also utilized the rectus muscle fascia to electively repair incisional hernia in solid organ transplant recipients at a later time from the initial transplant.

Monitoring of the Graft and Immunosuppression

The fascia of rectus muscle does not require additional use of immunosuppression in transplant recipients who are already receiving immunosuppression. This type of graft, being avascular and with poor cellularity, is not highly immunogenic; therefore, no particular monitoring is needed.

Results

The first series of 16 cases was published as a joint effort of three transplant centers [7]. Currently at the University of Miami, 31 cases have been performed for various indications, ranging from abdominal closure at the time of transplant to repair of incisional hernia long after transplant. Infection of the graft has been observed in only a few cases, and no incisional or recurrent incisional hernia has been observed in the recipients.

Conclusions

Closure of the abdominal wall defects in recipients of intestinal and multivisceral transplantation can be difficult at times, especially in the pediatric population and in those patients with multiple previous abdominal surgeries. Standard techniques used in general surgery, such as component separation techniques or the use of mesh, do help in a majority of cases. The use of the rectus muscle fascia as a biological mesh is a relatively easy, inexpensive, and widely available option in patients already immunosuppressed. For those complex patients in whom those techniques are not sufficient, transplantation of the vascularized abdominal wall can be used in selected cases with satisfactory results.

References

1. Alexandrides IJ, Liu P, Marshall DM, et al. Abdominal wall closure after intestinal transplantation. Plast Reconstr Surg. 2000;106:805.
2. Carlsen BT, Farmer DG, Busuttil RW, et al. Incidence and management of abdominal wall defects after intestinal and multivisceral transplantation. Plast Reconstr Surg. 2007;119:1247–55.
3. DiBenedetto F, Lauro A, Masetti M, et al. Use of prosthetic mesh in difficult abdominal wall closure after small bowel transplantation. Transplant Proc. 2005;37:2272–4.
4. Asham E, Uknis ME, Rastellini C, et al. Acellular dermal matrix provides a good option for abdominal wall closure following small bowel transplantation: a case report. Transplant Proc. 2006;38:1770–1.
5. Sheth J, Sharif K, Lloyd C, et al. Staged abdominal closure after small bowel or multivisceral transplantation. Pediatr Transplant. 2012;16:36–40.
6. Levi DM, Tzakis AG, Kato T, et al. Transplantation of the abdominal wall. Lancet. 2003;361:2173–6.
7. Gondolesi G, Selvaggi G, Tzakis A, et al. Use of the abdominal rectus fascia as a nonvascularized allograft for abdominal wall closure after liver, intestinal and multivisceral transplantation. Transplantation. 2009;87:1884–8.
8. Cipriani R, Contedini F, Santoli M, et al. Abdominal wall transplantation with microsurgical technique. Am J Transplant. 2007;7:1304–7.
9. Bejarano PA, Levi D, Nassiri M, et al. The pathology of full-thickness cadaver skin transplant for large abdominal defects: a proposed grading system for skin allograft acute rejection. Am J Surg Pathol. 2004;28:670–5.
10. Cendales LC, Kanitakis J, Schneeberger S, et al. The Banff 2007 working classification of skin-containing composite tissue allograft pathology. Am J Transplant. 2008;8:1–5.
11. Agarwal S, Dorafshar AH, Harland RC, et al. Liver and vascularized posterior rectus sheath fascia composite tissue allotransplantation. Am J Transplant. 2010;10:2712–6.

Minimally Invasive Component Separation in the Repair of Large Abdominal Wall Defects

18

Eva Barbosa and Fernando Ferreira

Introduction

The component separation technique for the treatment of large abdominal defects was popularized by Ramirez et al. in 1990 [1]. However, subsequent literature reviewed the results of this technique, pointing out some problems, such as a relatively high recurrence rate and postoperative issues such as ischemia and frank necrosis [2, 3]. Nevertheless, the component separation technique became appealing for the treatment of complex patients, especially in the contaminated setting where synthetic mesh is not recommended [4, 5] and in massive hernias with loss of domain [6], thus avoiding complex mutilating muscle flaps as an alternative reconstructive technique. To avoid the early problems described with open component separation, novel minimally invasive techniques appeared in the literature and are discussed in this chapter.

Definition of Large Abdominal Defects

It is difficult to find in the literature a consensus terminology to classify abdominal wall defects. Many terms, such as massive hernia, large abdominal defect, loss of domain, and complex abdominal hernia, coexist and are not clearly defined. This is a drawback when it comes to achieving a clear and common scientific language to compare results between procedures and centers. Some groups have proposed classification systems for incisional ventral hernias to fill this gap and allow comparison of publications and standardization of terminology [7, 8]. Prospective studies are still needed to assess the clinical relevance of these classification studies, and probably an individual classification for complex abdominal defects is required. In these abdominal defects, it is also extremely important to evaluate each patient's risk for surgery-associated complications and thereby select the best surgical technique for a specific individual. With this purpose in mind, the Ventral Hernia Working Group created an important grading system of risk assessment that was included in a working algorithm for repair of incisional ventral hernias [9].

The size of the defect is a constant variable included in every system proposed, and a minimum 10 cm for the definition of large hernias is generally accepted. However, how accurately the abdominal wall defect can be measured in a consistent and reproducible manner has yet to achieve consensus. Preoperative or intraoperative measures have some degree of surgeon bias, and some authors defend a computed tomographic (CT) scan for accurate and reproducible measures of the abdominal defect [10].

Use of the term *loss of domain* can be referenced to the 1940s [11], but this also has no standard definition in the literature. It usually refers to a massive hernia with visceral contents outside its fascial boundaries in a manner that their return to the abdominal cavity cannot be made simply [12] without a high chance of developing respiratory complications and abdominal compartment syndrome. The relationship between viscera outside and inside fascial boundaries is yet to be determined as a definition of loss of domain, especially because it is important to keep in mind other aspects besides size, given

E. Barbosa, MD, MSc (✉)
Department of Surgery: Colo-Rectal and Abdominal Wall Surgery,
Hospital Pedro Hispano - Rua Dr. Eduardo Torres,
4464-513 Senhora da Hora, Sra da Hora, Portugal

Department of Anatomy, ICBAS - University of Porto,
Praça de Gomes Teixeira, Porto, Portugal
e-mail: evatamar@gmail.com

F. Ferreira, MD
Department of Surgery,
Upper Gastrointestinal and Abdominal Wall Surgery,
Hospital Pedro Hispano, Sra da Hora, Portugal

Department of Medicine - University of Porto,
Praça de Gomes Teixeira Porto, Portugal
e-mail: med1873@gmail.com

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_18, © Springer Science+Business Media New York 2013

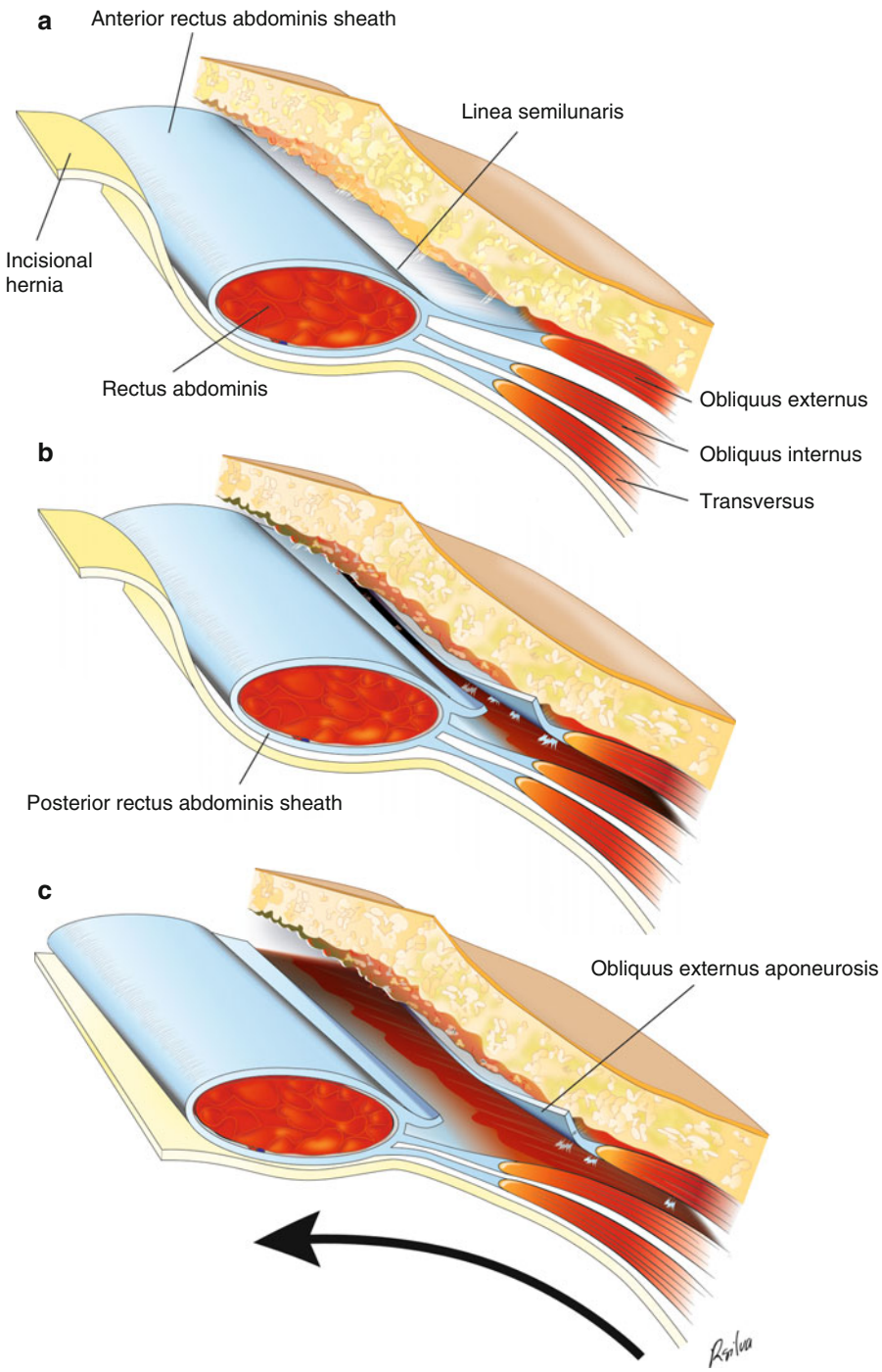


Fig. 18.1 (**a**) Normal anatomy of the abdominal wall. (**b**) Section of the external oblique 1–2 cm lateral to the semilunaris line. (**c**) Dissection of the external oblique muscle from the internal oblique to allow the muscle complex formed by the rectus–internal oblique–transversus abdominis to slide toward the abdominal midline (Drawing by Dr. António Rodrigues da Silva. Courtesy of Dr. Rodrigues da Silva)

that smaller defects may have important repercussions for ventilation considering the previous comorbidities of the patient.

In summary, size is not the only issue when considering the complexity of an abdominal wall defect and consequently when choosing the best closing method. Other issues, such as patient comorbidities or the presence of an enterocutaneous fistula [13] or an infected mesh, pose important technical decisions.

Component Separation Technique

The concept of component separation involves the release of the external oblique fascia from the anterior rectus sheath, starting 5–6 cm above the rib cage to the inguinal ligament, causing the midline slide of the muscle complex formed by the rectus–internal oblique–transversus abdominis (Fig. 18.1a, b, c). Extra mobilization can be achieved by release of the posterior rectus sheath (Fig. 18.2).

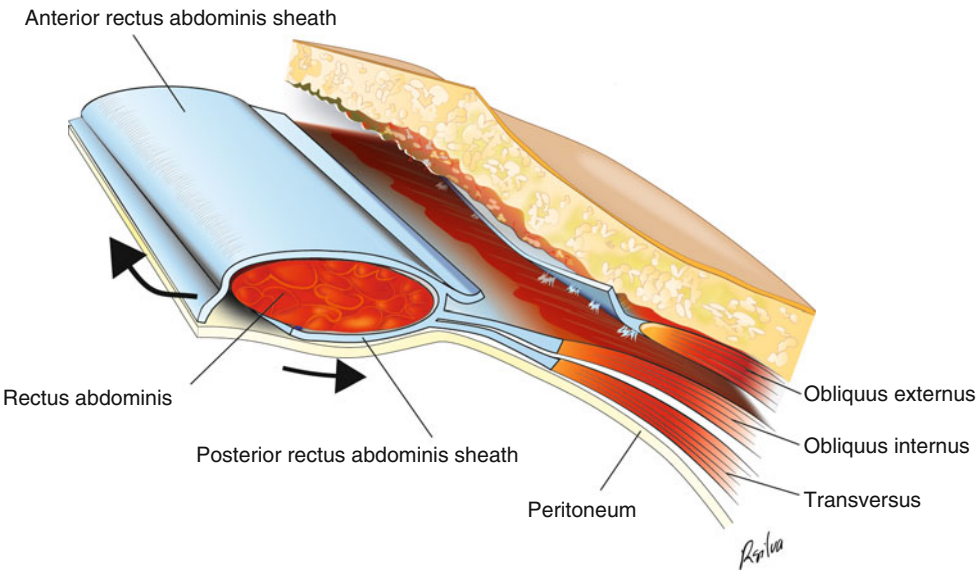


Fig. 18.2 Section of the posterior rectus sheath to allow extra mobilization of the rectus complex (Drawing by Dr. António Rodrigues da Silva. Courtesy of Dr. Rodrigues da Silva)

The component separation technique, besides having the capability to close large abdominal defects without using prosthetic material, reconstructs a functional abdominal wall. This is impossible to achieve in the classical methods of mesh bridging without midline approximation [14]. Laparoscopy in ventral incisional hernias is increasing in popularity as it has several advantages over the open techniques, including reduction in infection rates, postoperative pain, and postoperative ileus [15]. Despite its many advantages, the laparoscopic reconstruction technique involves intraperitoneal mesh bridging of the defect, which does not achieve a dynamic physiologic reconstruction. However, with the association of video-assisted component separation, it is possible, in selected cases, to achieve midline closure. The combination of component separation and hernia laparoscopic repair gives the patient the benefits of both techniques with high functional results and low recurrence rates [16, 17], but data in the literature are still scarce. Unfortunately, in large and complex abdominal wall defects, laparoscopy might be technically challenging and therefore not feasible.

Since the original technique of component separation was described, many variations have been made, mostly to avoid the morbidity associated with extensive cutaneous flaps. Even in the open technique, perforating vessels must be preserved to avoid skin ischemia, significantly lowering the morbidity of the procedure [18, 19].

Minimally Invasive Component Separation Technique

Introduction

When it comes to defining minimally invasive component separation, a wide range of different techniques appears in the literature instead of a single well-defined approach. This concept can be divided into two large subgroups with a fundamental distinguishing characteristic: the use or nonuse of video-assisted equipment to perform fascial dissection. To understand the different techniques under the same general name, we summarize the surgical approaches and descriptions based on these two subgroups.

Minimally Invasive Component Separation Technique Without the Use of Video-Assisted Equipment

Smaller incisions can achieve the same final goal on the release of the external oblique fascia and achieve the goal of avoiding the large skin flaps and injury to perforating vessels. Dumanian and colleagues have published a number of data concerning this technique [10]. They used a transverse subcostal incision to gain access to the external oblique fascia and performed the component separation under direct vision; their release took about 15–20 min. Buttler and Campbell published their data on approaching the external oblique fascia through a tunnel created from the midline incision, avoiding two additional lateral incisions [20]. In this study, no description was made of operative times for the component separation alone, making comparison to other methods difficult.

It is necessary to keep in mind that all these approaches are in fact less invasive, with lower complication rates than modified classical open techniques.

Video-Assisted Component Separation Technique

Many different names are used under the same basic technical principles: endoscopic component separation, video-assisted

component separation, and laparoscopic component separation. *Laparoscopy* derives from the Greek words *lapara*, which means "the soft parts of the body between the rib margins and hips" or "loin," and *skopeo*, which means "to see or view or examine" [21]. By analogy with laparotomy, it generally implies the entrance in the abdominal cavity to examine or perform a procedure inside the abdomen, which actually does not happen in the component separation technique, although the same surgical material is used. *Endoscopy* is derived from the Greek words *endon* ("within") and *skopeo* ("examine") [21]. Usually, procedures take place through the endoscope itself, with imaging guidance through image projection on a screen. Video-assisted surgery is a procedure that is aided by the use of a video camera that captures and projects the image on a television screen. It is our opinion that, despite the points of truth in every designation, the one that most accurately corresponds to component separation is video assisted, and it is described further in this chapter.

Comparing Results from Different Component Separation Techniques

When comparing component separation techniques, there appears to be a general consensus regarding the beneficial effects of minimally invasive techniques compared to open component separation, especially regarding postoperative pain and skin complications [22–25]. However, one of the main questions posed is whether the minimally invasive component separation technique can offer the same rectus advancement as the open technique. Knowing that the release of the external oblique fascia alone does not promote complete advancement, it is mandatory to add the dissection of the external from the internal oblique muscle, moving the external oblique as laterally as possible, usually to the posterior axillary line. Rosen and colleagues have used a porcine model and demonstrated an average of 86 % of the myofascial advancement with video-assisted component separation compared with a formal open release [26]. No similar comparative study exists between different minimally invasive techniques.

A comparison of operative times, rectus complex advancement, complications, and costs regarding the different minimally invasive procedures studies is definitely needed. Problems pointed out in the video-assisted approaches are the costs and extra material involved when compared to the minimally invasive procedures without video assistance. Rosen and colleagues reported that the total direct costs associated with video-assisted and open component separation techniques were actually similar, but the comparison was not made between different minimally invasive procedures [27]. It would be interesting to compare minimally invasive methods to account for the total cost for a video-assisted procedure, but despite the importance of randomized studies, it is not easy to enroll patients as each patient frequently represents a unique reality that cannot be solely based on the evaluation of some parameters such as hernia size, body mass index (BMI), or comorbidities. These patients usually represent extremes instead of daily realities, and many other factors account for global cost and success, such as the use of synthetic or biological meshes.

Preoperative Care

Treating massive and complex abdominal defects does not start the day before surgery. It is usually a long path until final reconstruction is achieved. A detailed plan with alternative options for successful closure should be discussed with the patient.

When managing an open abdomen, it is important to make sure all the intra-abdominal problems are resolved. The use of CT or other appropriate imaging is helpful and adequate. In these critically ill patients, it is important to ensure that they are in the recovery phase of their illness, with fluid control for an optimized negative fluid balance, good nutritional status, and exclusion of any major infection. Although surgical aggression promotes another catabolic phase before the final recovery phase, the closure of the open abdomen ends a vicious cycle of proinflammation. With this in mind, the patient should be in the best physiological status before reconstructive surgery.

Nutritional status is essential for postoperative recovery and should never be underestimated before any kind of major abdominal reconstruction. Special consideration should be given to the high-output intestinal fistula. The intestinal rehabilitation previous to surgery is often a challenging, difficult step for the patient, the family, and the physician. Dealing with high-output enterocutaneous fistulae is an extra burden for a physically and mentally exhausted patient. Even when no nutritional parameters are altered except for weight lost over 10 %, their physiological reserve is at the limit. These individuals may not be able to recover well after surgery, increasing the probability of infection and poor wound healing [28].

Determining the size of the defect is a critical step for meticulous detailed surgery preparation and future success. Our measurement is estimated in two ways:

1. Transverse and longitudinal measurements when the patient is lying in the supine position. These parameters allow the calculation of the area of the hernia equivalent to that of an ellipse;
2. Measurement of the defect with a CT scan in every patient prior to surgery. It is our experience that CT measurement is usually smaller compared to directly measuring the patient either pre- or intraoperatively. However, CT scan

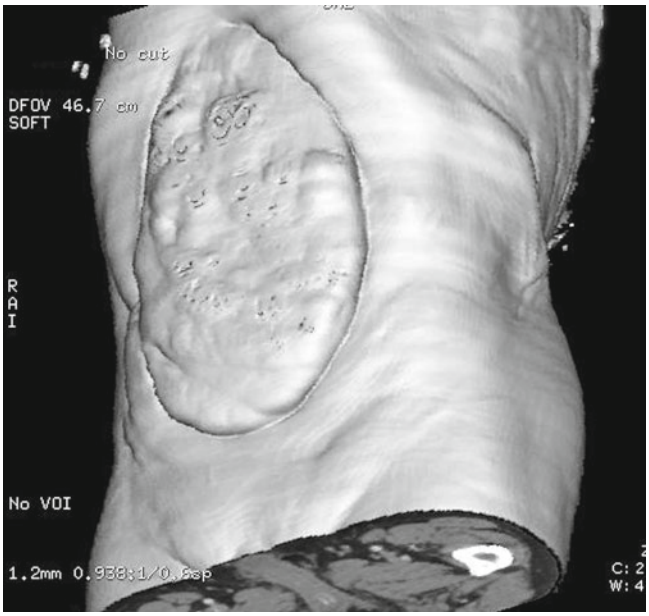


Fig. 18.3 Computed tomographic (CT) three-dimensional (3D) reconstruction as a tool for preoperative surgical technique programming

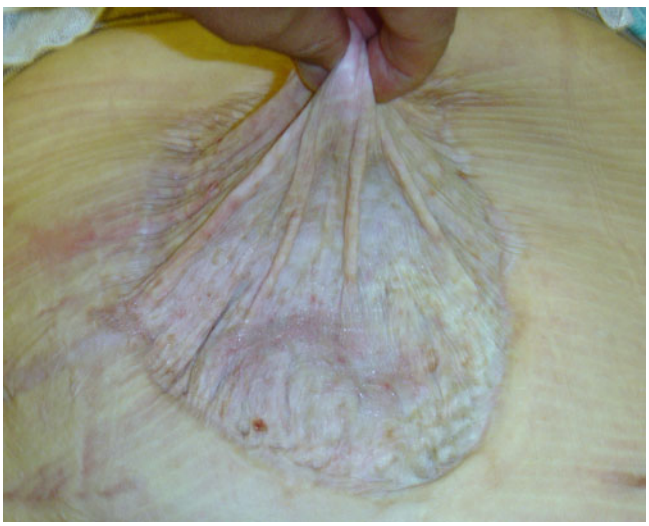


Fig. 18.4 Skin pinch of the mature graft

measures are more objective, limiting any surgeon's bias [10]. Another important aspect of ordering a CT scan before every reconstruction is the evaluation of rectus muscle status given that the component separation technique relies on the integrity of these muscles. In conjunction with radiology, we are currently developing three-dimensional (3D) CT reconstructions to fully access the complexity of the abdomen and properly plan surgery (Fig. 18.3).

When dealing with planned ventral hernias with a previous skin graft, it is best to allow enough time before reconstruction, usually 9–12 months [29], to lower the risk of bowel injury during adhesiolysis (Fig. 18.4). Closure of the patient's skin without any grafts can be approached earlier.

Assessing healthy skin status is essential for a good outcome and independent from the reconstruction of the abdominal wall. It is crucial to anticipate lack of skin coverage and provide adequate surgical technique either through skin expanders or flaps.

Whenever possible, one must consider including the management of bowel and abdominal reconstruction in a single step or a two-step approach with bowel reconstruction before the definitive repair of the abdominal wall to avoid a contaminated procedure that may increase postoperative morbidity. This, however, has its risks, as a patient will undergo two major operations.

Risk factors should be assessed, especially those known in the literature to predict postoperative complications, such as obesity, smoking, chronic pulmonary lung disease, immunosuppression, and diabetes [30]. These risk factors pose important issues to take into consideration when choosing the appropriate closing technique.

Contamination also plays a role in preoperative planning. Potential contamination is expected with previous wound infection (either superficial or deep), presence of a stoma, or violation of the gastrointestinal tract as defined by the Hernia Working Group [9]. If the surgical field is expected to be contaminated (grade III in the hernia grading system), consider applying a minimally invasive approach and a biological instead of a synthetic mesh as suggested by the Hernia Working Group [9].

Intraoperatively, it is extremely important to reduce fluids to strictly the necessary amount. Restrictive and goal-directed fluid policy has proven useful in reducing bowel edema and postoperative complications in a number of surgical areas [31]. We think this concept can also be safely applied when dealing with abdominal closure of massive defects. Good muscle relaxation is mandatory during the procedure to avoid excessive tension and technical difficulties. Thoracic epidural analgesia should be the standard of care as recent studies showed a positive effect in lowering the intra-abdominal pressure [32, 33]. This type of specific analgesia leads to abdominal muscle relaxation, lowering the risk of pulmonary-associated complications.

Antibiotics are given 30 min prior to the beginning of surgery, and the choice depends on the type and degree of contamination of the wound. Finally, the surgery should be reviewed with the patient to discuss realistic expected problems and recommendations with few but significant details, such as stopping smoking at least a month in advance.

The success of this surgery requires careful planning, attention to detail, and early involvement of other specialists, such as an anesthesiologist and an intensive care specialist, in the entire process.

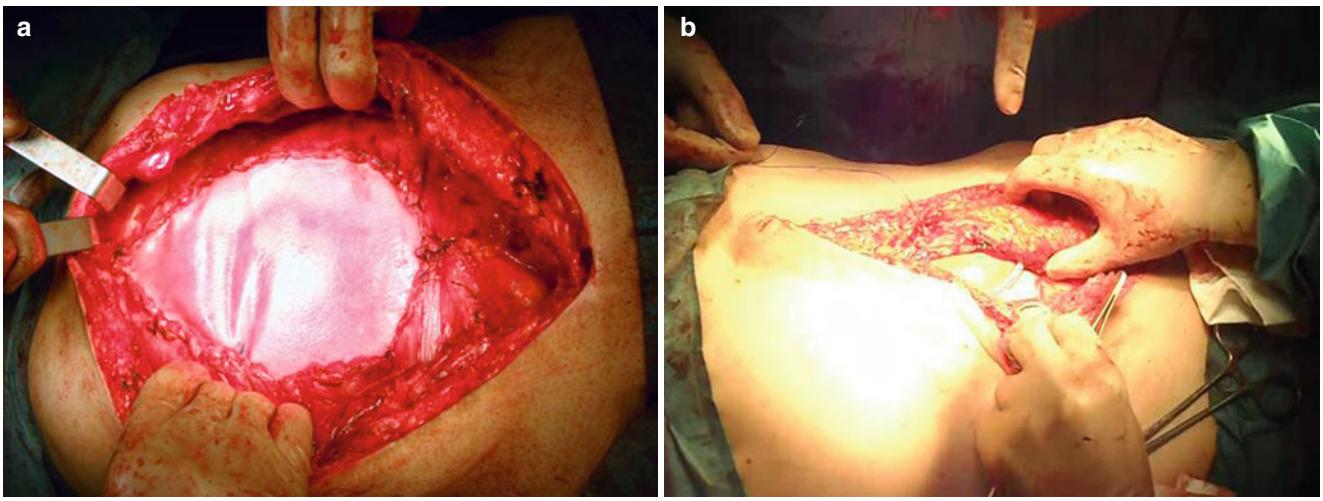


Fig. 18.5 (**a**) Dissection of the posterior rectus sheath. As it was impossible to close the sheath in the midline, a biological mesh was placed intraperitoneally and fixed with transabdominal sutures. Inferior partial closure of the posterior sheath was performed, with a running suture over the mesh. (**b**) Anterior rectus sheath closure with a running suture over a closed-suction drain

Surgical Technique: General Considerations

Following video-assisted component separation and laparotomy, the type and diameter of the mesh to be used and an adequate fixation technique must be selected. When treating complex and large abdominal wall defects, the choice of mesh is critical for success. We prefer biological over synthetic essentially when we have a previously rejected, contaminated, synthetic mesh or in a contaminated operative field with simultaneous bowel reconstruction and complex abdominal reconstruction. We preferably use two sites for mesh placement: retrorectus and intraperitoneal. Either way, for proper abdominal wall reconstruction, it is extremely important to have wide mesh overlap of the abdominal defect under correct physiological tension. Floppy mesh will increase complications, such as seromas and poor mesh integration, and in bridged defects, especially with biological meshes, a bulging will be seen. The rectrorectus technique implies wide mobilization of the posterior rectus sheath, but although described in the classical technique, it is not desirable to extend it to the transversalis fascia as the lateral abdominal wall is weakened by the component separation. The posterior rectus sheath is then closed in the midline with a running monofilament suture. The mesh is placed anterior to the posterior sheath and properly secured with sutures placed transabdominally using a suture passer. Afterward, the anterior rectus sheath is closed over the mesh with a running monofilament suture.

Sometimes, it is not possible to totally close the posterior sheath, but its mobilization allows us an extra few centimeters to achieve the necessary mobilization of the muscle complex formed by the rectus–internal oblique–transversus to slide over the midline and achieve closure. In this case, the mesh is placed intraperitoneally, and after proper fixation as described next, we fix the posterior rectus sheath to the mesh with a running suture (Fig. 18.5a, b).

If complete midline closure is impossible or the rectus muscles are not wide enough to sustain proper mesh overlap, some bridging will be necessary; in this scenario, we prefer to place the mesh intraperitoneally. This can be challenging to achieve after a video-assisted component separation that lacks the large skin flaps of open procedures. We use a "clock" transabdominal technique to secure the mesh with 12 corresponding "hour" sutures. The sutures are secured to the mesh and then passed through the abdominal wall with a suture passer. Some authors find it useful to introduce the laparoscope intra-abdominally at the end of the surgery and secure the rest of the mesh with tackers [34]. This may diminish the risk of bowel entrapment and cause difficulty in mesh incorporation, which leads to increasing associated complications. We use this technique depending on the type of mesh applied as tackers are easy to place through a synthetic mesh but not through a biologic mesh. When some bridging is needed, we also add a running suture of the rectus fascia to the mesh, reducing its subcutaneous exposition. Although challenging when biologic meshes are applied, we feel this can be accomplished safely and may have a positive outcome in terms of complications, including seromas; however, further studies are needed.

Step-by-Step Surgical Technique

Clear, preoperative landmarks are drawn on the abdominal wall. This allows everyone on the team to perceive the anatomic landmarks and major defects, facilitating understanding and communication (Fig. 18.6).

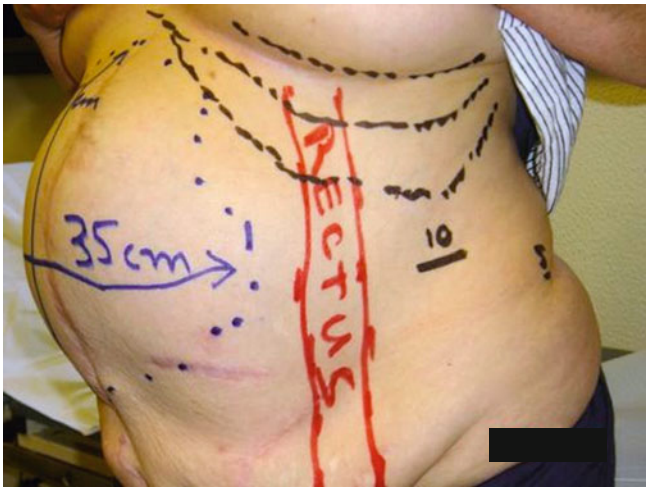


Fig. 18.6 Abdominal wall anatomical landmarks and defect

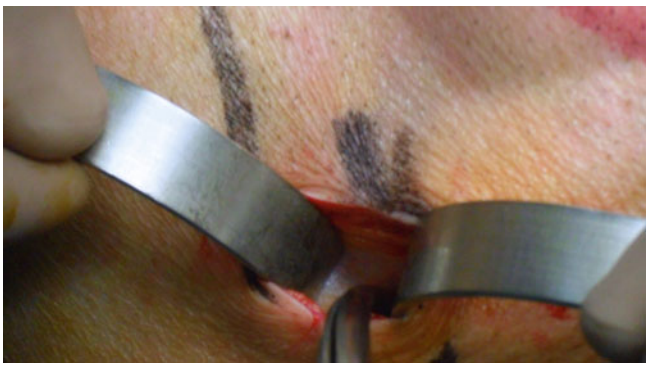


Fig. 18.7 Opening of the external oblique muscle fascia through a 1- to 2-cm incision on the tip of the 11th rib

Step 1

Start with a 1- to 2-cm incision under the tip of the 11th rib, usually on the anterior axillary line. Continue dissection of the anatomical planes until the external oblique fascia is identified (Fig. 18.7). Open the muscle fascia and make a blunt dissection of the underlying plane, between the external and internal oblique, to make step 2 easier (Fig. 18.8).

Step 2

Insert the trocar balloon (Spacemaker™ Plus Dissector System, Covidien, Dublin, Ireland) (Fig. 18.9). After creating an avascular plane with blunt dissection between the muscles with the trocar balloon, connect it to the CO_2 insufflator, aiming for a pressure of 8–12 mmHg (Fig. 18.10). Introduce a 10-mm 30° camera after removing the balloon (Fig. 18.10).

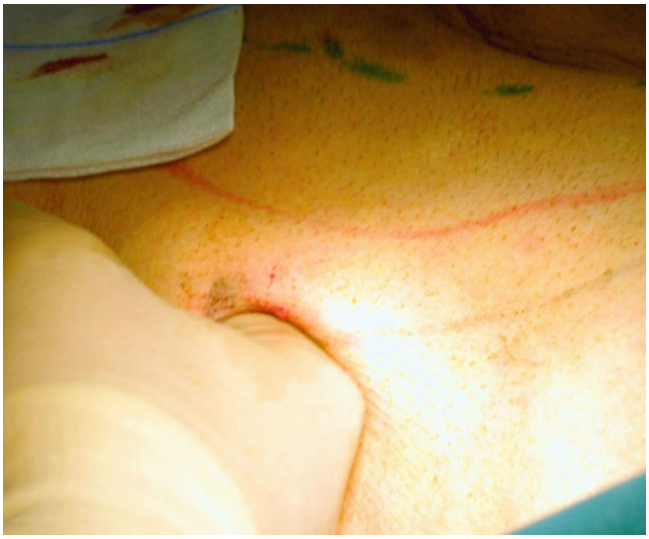


Fig. 18.8 Blunt dissection of the underlying plane of the external oblique, making insertion of the trocar balloon easier

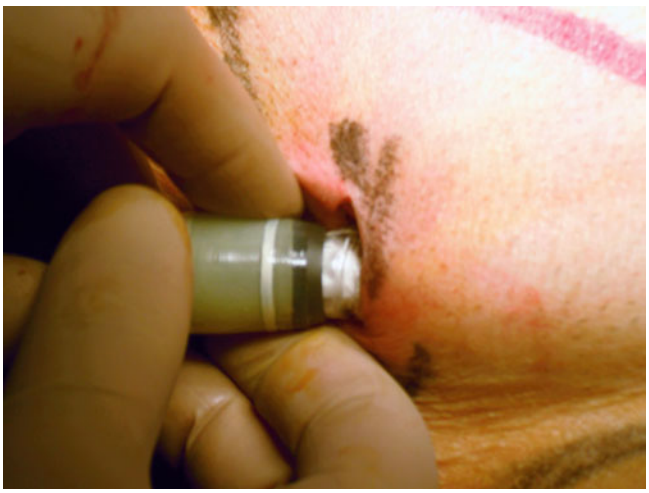


Fig. 18.9 Insertion of the trocar balloon for blunt dissection of the avascular plane between the external and internal oblique muscles

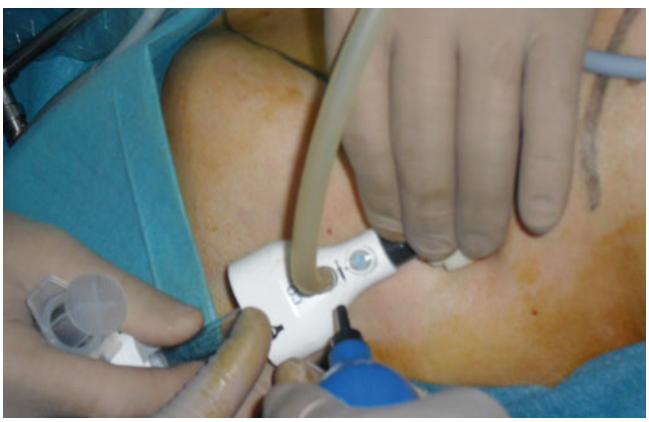


Fig. 18.10 Connection of the CO_2 insufflator

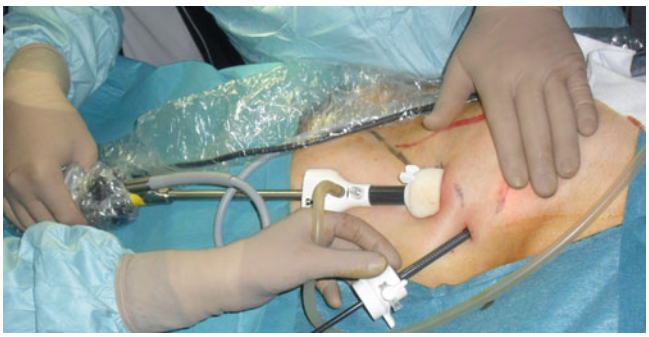


Fig. 18.11 Insertion of a 10-mm 30° camera and introduction of a working 5-mm trocar in the posterior axillary line as it is a difficult working angle

Step 3

Introduce a 5-mm trocar as posteriorly as possible, at the level of the posterior axillary line, to provide a good dissection angle (Fig. 18.11).

Make sure to identify the area above the line of the fascia of the external oblique, lateral to the semilunaris line, and cut it all the way to the inguinal ligament (Fig. 18.12). It is extremely important not to cut the semilunaris line or else a complex and difficult-to-solve defect will result.

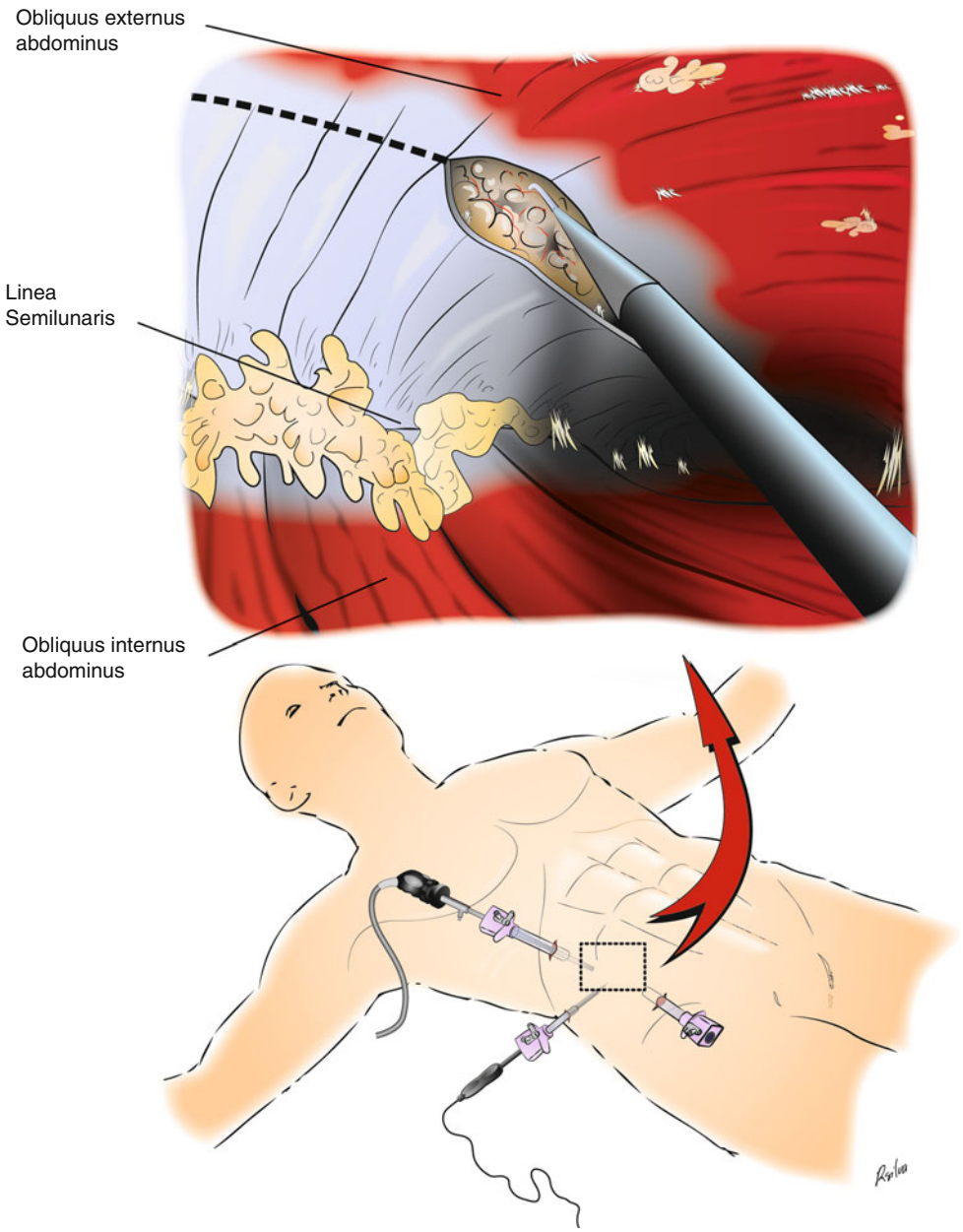


Fig. 18.12 Trocar placement view and image projected on the screen. Section of the external oblique fascia lateral to the semilunaris line (Drawing by Dr. António Rodrigues da Silva. Courtesy of Dr. Rodrigues da Silva)

Step 4

Introduce another 10-mm trocar in the right iliac fossa to extend the component separation 5–6 cm above the costal margin. Here, it is important to use cautious hemostatic dissection as the muscular fibers tend to bleed.

Step 5

It is important during this process to make sure the external oblique is well dissected from the internal oblique to achieve the best rectus advancement.

Step 6

Sealed suction drains are placed through the most caudal trocar incision at the end of the surgery.

Postoperative Care

As previously explained, effective analgesia, ideally with a thoracic epidural catheter, is extremely important for a good outcome. This aids in avoiding intra-abdominal hypertension (IAH) and helps to prevent respiratory complications, especially in patients with chronic obstructive pulmonary disease (COPD).

After correction of massive hernias with loss of domain, there is always a concern that the return of abdominal contents to the abdominal cavity may induce diaphragmatic compression and raise the intra-abdominal pressure, eventually leading to abdominal compartment syndrome. Agnew and colleagues published data from abdominal volumetric studies that proved the existence of significant increased volume after component separation, providing less pulmonary restriction and consequent complications [35]. Care is taken to administer respiratory kinesiotherapy to high-risk pulmonary patients in the early postoperative period and, in some cases, preoperatively.

Unless patients are admitted to the intensive care unit (ICU), they sit for 6–12 h after surgery. Early walking, as early as postoperative day 1, is stimulated.

Drains are left in place until less than 30 mL a day output is achieved.

Most of the patients who experienced minimally invasive component separation, although going through a major abdominal wall reconstruction, recovered faster and with less morbidity than those who experienced an open technique. Most of the differences between the two groups were because of greater skin complications and postoperative pain in the open group. Usually, patients are discharged around the sixth or seventh postoperative day. Longer hospital stays are often related to previous comorbidities instead of the procedure itself. Heavy physical activity is usually postponed until 8 weeks after surgery, but the cutoff depends on individual characteristics and type of surgery.

Special Situations

The Open Abdomen

A vast majority of open abdomens are primarily closed without planned ventral hernias [36]. Yet, in some cases, this is simply impossible, especially in severe abdominal trauma or in a nontrauma setting with abdominal catastrophes. When closure cannot be achieved easily by suturing fascia, several techniques may be used to gradually assist in the closure of the abdomen with associated negative-pressure wound closure. Some examples are ABRA® (Canica Design, Almonte, Ontario, Canada) [37–39], Wittmann Patch® (Starsurgical, Burlington, WI) [40], and mesh-assisted gradual closure [41]. Even with all of these available procedures, in some cases ventral hernia repair must be avoided, and these techniques cannot be applied. In this setting, the component separation technique can be used to achieve primary closure, usually with biological mesh reinforcement.

To achieve maximum results from this technique, it is extremely important that the open abdomen be a grade I or II [42]. This represents an abdominal wall without adhesions to the underlying bowel. Only in this manner can complete abdominal rectus complex advancement be achieved (Figs. 18.13, 18.14, and 18.15). If the patient has a temporary

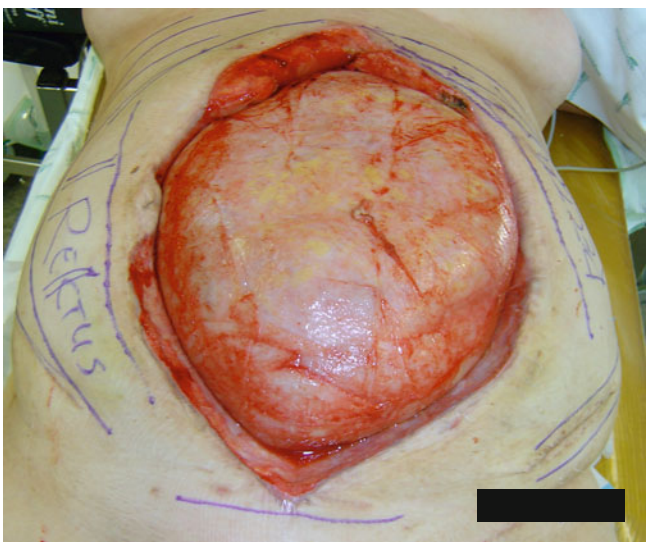


Fig. 18.13 Open abdomen grade IIa with a massive defect after postoperative shock caused by a large spontaneous retroperitoneal hematoma. Previously treated with ABThera™ (KCI, San Antonio, TX)

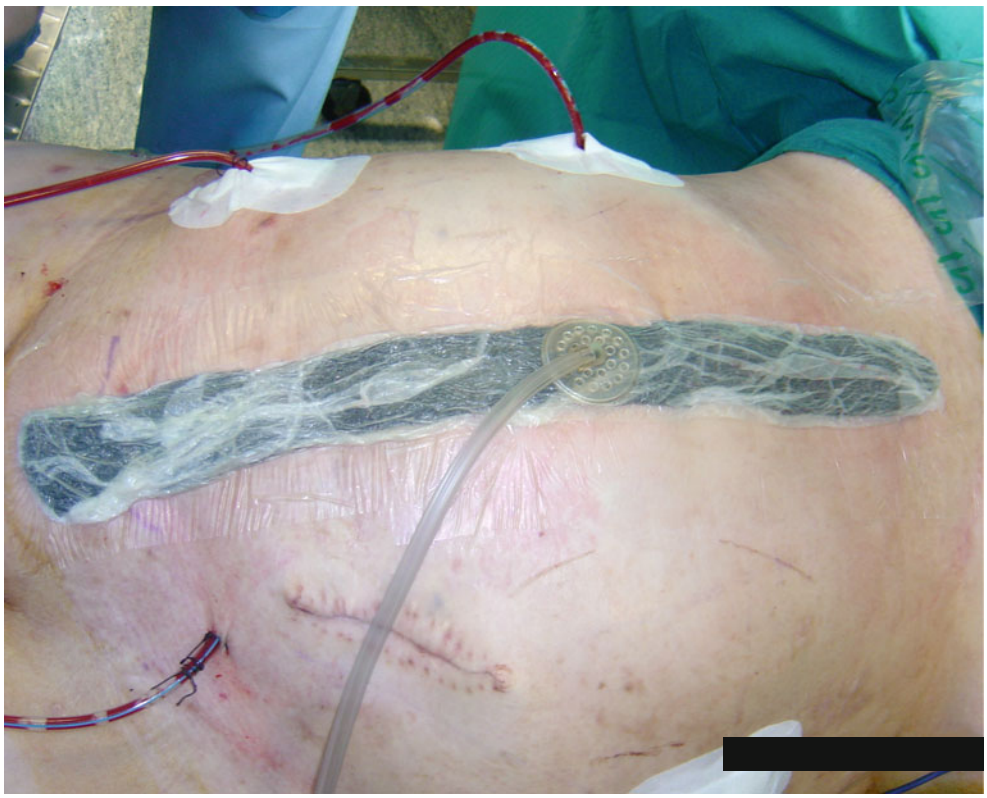


Fig. 18.14 Abdominal reconstruction with minimally invasive component separation on the right and open component separation technique with perforating vessel preservation on the left because of a previous stomal hernia repair with synthetic mesh that was removed during the laparostomy. Underlay biological mesh with some degree of bridging was necessary to achieve reconstruction. Skin closure with staples protectively sealed with negative-pressure wound therapy (V.A.C.® GranuFoam™ [KCI, San Antonio, TX] over a non adherent primary contact wound dressing, impregnated with silver [Atrauman® Ag from Hartmann, Heidenheim-Germany]) applied to the wound because of the high risk of infection

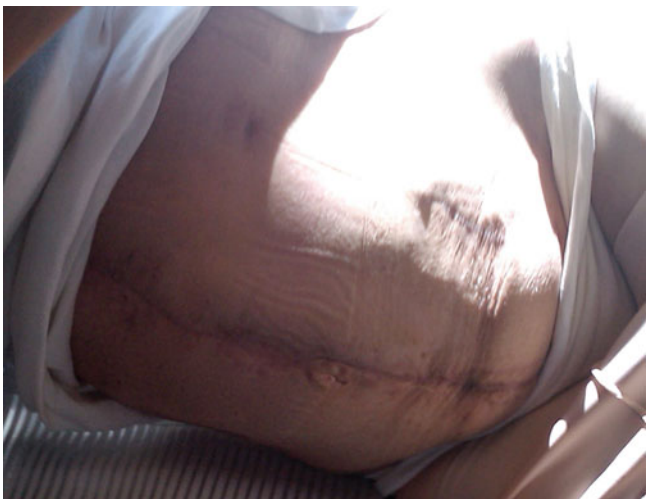


Fig. 18.15 Two months after surgery, fully recovered with a functional abdominal wall even during abdominal contraction while standing up from the supine position

stoma and an open abdomen, it is best to save component separation for the definitive surgery.

Even in difficult cases, such as cirrhosis with ascites, the minimally invasive component separation technique can achieve abdominal physiological closure with low morbidity (Figs. 18.16 and 18.17a, b), but this mostly depends in institutional expertise.

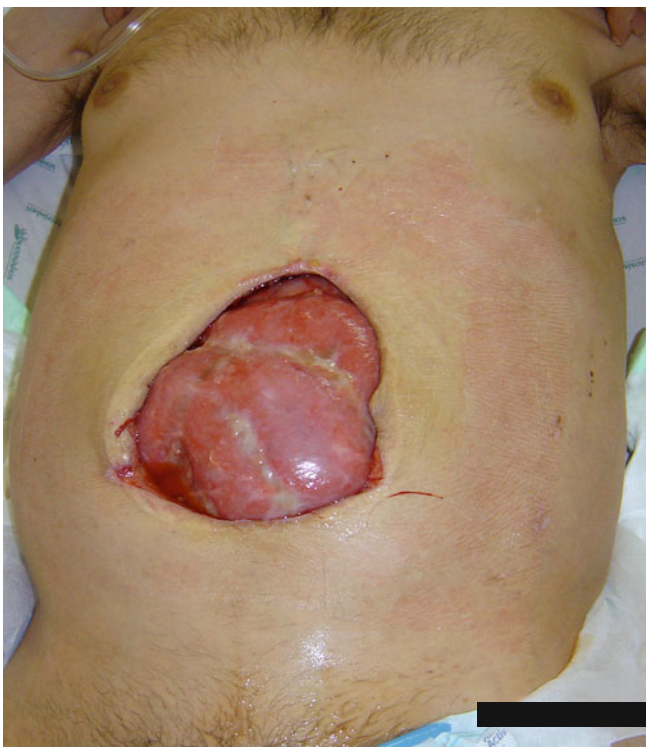


Fig. 18.16 A patient with cirrhosis with multiple eviscerations and infected ascites after a strangulated umbilical hernia and small bowel resection. Child-Pugh B score

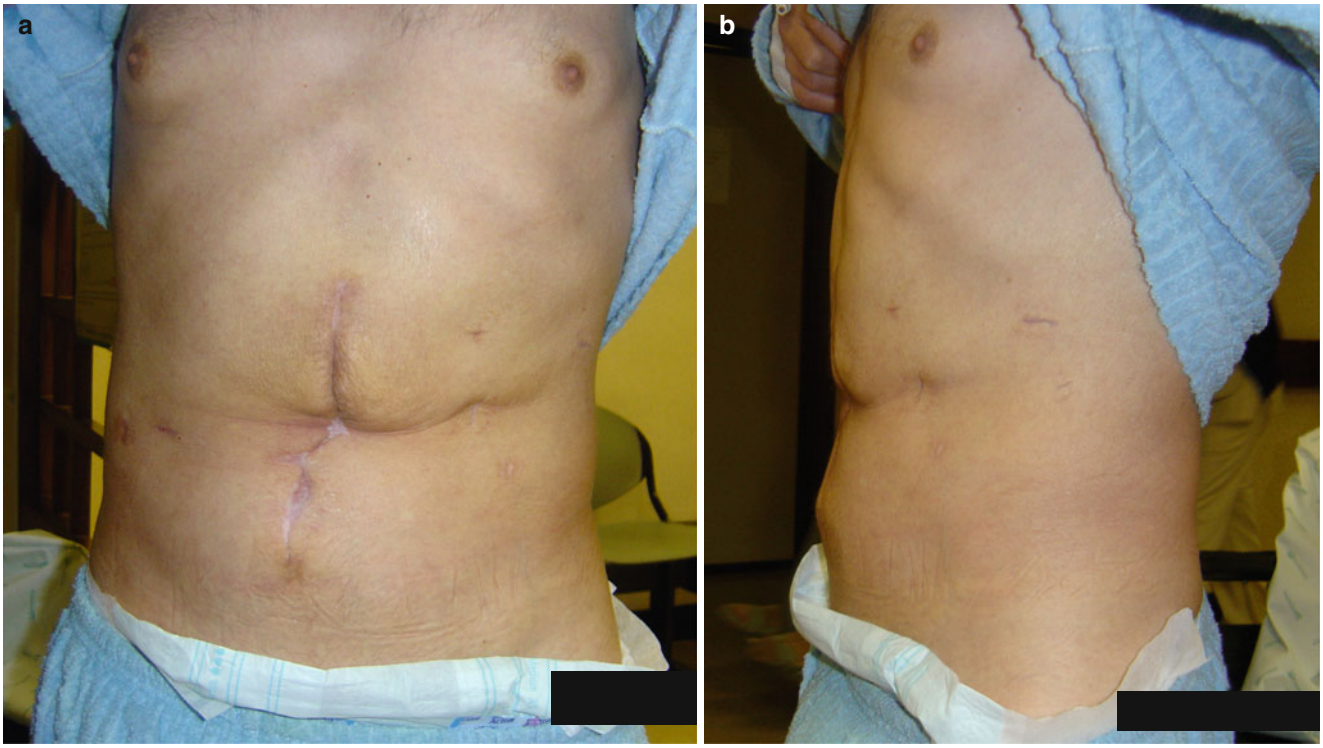


Fig. 18.17 (**a**, **b**). Seven weeks postoperatively after video-assisted component separation technique achieving midline closure and reinforcement with biological mesh

The Use of Tissue Expanders

Some patients with massive abdominal wall defects are expected to have significant abdominal wall retraction and fibrosis, minimizing the advancement of the rectus muscle during component separation. In these cases, tissue expanders prior to surgery may aid in obtaining a successful reconstruction [43, 44]. To achieve major rectus advancement, tissue expanders are placed between the internal and external oblique muscles and are gradually filled for up to 4 months (Figs. 18.18 and 18.19). This will create a foreign body response and a thick fibrotic capsule. When video-assisted component separation is performed, the anatomical landmarks are distorted, and a minimally invasive procedure can be difficult. In these cases, a minimally invasive approach without video assistance or an open procedure should be considered.

When tissue expanders are subcutaneously inserted because of lack of skin, the video-assisted component separation is not compromised and may be performed in a standard way.

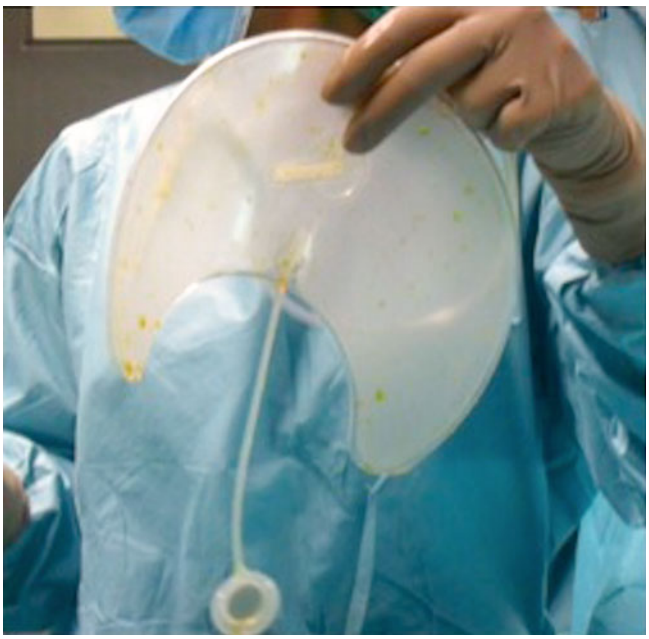


Fig. 18.18 Tissue expander is inserted between the external and internal oblique muscles and left up to 4 months with gradual saline filing

Stomas

There are few reports in the literature on the use of a minimally invasive component separation technique and stomas. Rosen and coworkers described the use of a myofascial advancement flap combined with other techniques for the simultaneous repair of large midline incisional and

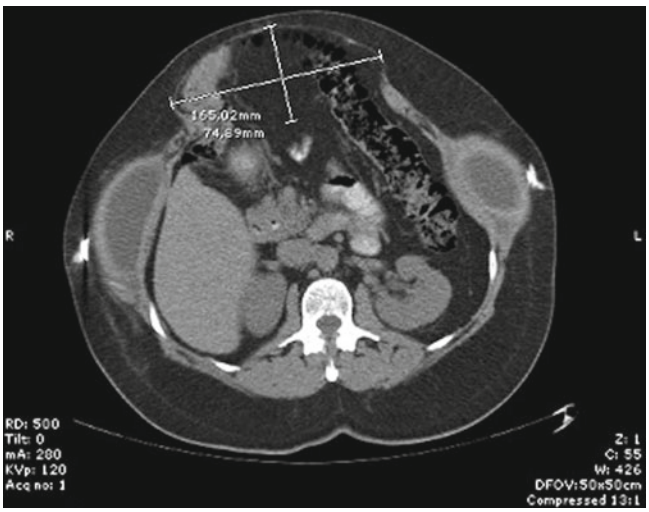


Fig. 18.19 Computed tomographic (CT) scan of the tissue expanders placed between the external and internal oblique muscles

parastomal hernias, with good results [45]. In our experience, a preoperative CT assessment to determine the position of the stoma is critical for decision making. A transrectus and not a pararectus stoma must exist to proceed with a video-assisted component separation technique; otherwise, bowel injury and complex defects may result. When relocation of the stoma is warranted, the procedure must start with a minimally invasive procedure on the future side of the stoma. After relocation of the stoma, safer component separation can also be performed on the ipsilateral side with adequate mesh reinforcement.

Previous Component Separation

Repeating a component separation is feasible but poorly described in the literature. The main issues are the real value of successful recurrent hernia repair, adding a new component separation, and the possibility of achieving it by another minimally invasive procedure because fibrosis is expected. More studies are needed to answer these questions.

Summary

The minimally invasive component separation technique is a feasible and reproducible technique. This procedure allows, for many large defects, restoration of the abdominal midline, helping to promote a physiological abdominal reconstruction. If complete midline restoration is not possible, component separation helps in reducing the abdominal wall defect, decreasing the amount of prosthetic material necessary for a bridge repair, respecting as much as possible the physiology and movement of the abdominal wall.

The minimally invasive component separation technique has many advantages over open techniques, avoiding large skin flaps and consequent problems related to wound healing. Additional studies are needed to compare different minimally invasive techniques regarding advancement myofascial flaps. Further studies also are needed to analyze costs.

Finally, it is important to keep in mind that a minimally invasive component separation technique is only one of the helpful parts of the puzzle in the treatment of large and complex abdominal defects. Proper planning and attention to detail are important for successful achievement of abdominal closure.

References

1. Ramirez OM, Ruas E, Dellon AL. "Components separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86(3):519–26.
2. Lowe 3rd JB, Lowe JB, Baty JD, Garza JR. Risks associated with "components separation" for closure of complex abdominal wall defects. Plast Reconstr Surg. 2003;111(3):1276–83; quiz 1284–5; discussion 1286–8.
3. de Vries Reilingh TS, van Goor H, Rosman C, et al. "Components separation technique" for the repair of large abdominal wall hernias. J Am Coll Surg. 2003;196(1):32–7.
4. de Vries Reilingh TS, van Geldere D, Langenhorst B, et al. Repair of large midline incisional hernias with polypropylene mesh: comparison of three operative techniques. Hernia. 2004;8(1):56–9.
5. Rosen MJ, Jin J, McGee MF, et al. Laparoscopic component separation in the single-stage treatment of infected abdominal wall prosthetic removal. Hernia. 2007;11(5):435–40.
6. Hadad I, Small W, Dumanian GA. Repair of massive ventral hernias with the separation of parts technique: reversal of the "lost domain". Am Surg. 2009;75(4):301–6.
7. Korenkov M, Paul A, Sauerland S, et al. Classification and surgical treatment of incisional hernia. Results of an experts' meeting. Langenbecks Arch Surg. 2001;386(1):65–73.
8. Muysoms FE, Miserez M, Berrevoet F, et al. Classification of primary and incisional abdominal wall hernias. Hernia. 2009;13(4):407–14.
9. Breuing K, Butler CE, Ferzoco S, et al. Incisional ventral hernias: review of the literature and recommendations regarding the grading and technique of repair. Surgery. 2010;148(3):544–58.
10. Ko JH, Wang EC, Salvay DM, et al. Abdominal wall reconstruction: lessons learned from 200 "components separation" procedures. Arch Surg. 2009;144(11):1047–55.
11. Moreno IG. Chronic eventrations and large hernias; preoperative treatment by progressive pneumoperitoneum; original procedure. Surgery. 1947;22(6):945–53.
12. Harth K, Rosen MJ. Repair of ventral abdominal wall hernias. ACS surgery: principles and practice, Intellectual properties. Ontario: Decker; 2010.
13. Wind J, van Koperen PJ, Slors JF, Bemelman WA. Single-stage closure of enterocutaneous fistula and stomas in the presence of large abdominal wall defects using the components separation technique. Am J Surg. 2009;197(1):24–9.
14. Dumanian GA, Denham W. Comparison of repair techniques for major incisional hernias. Am J Surg. 2003;185(1):61–5.
15. Turner PL, Park AE. Laparoscopic repair of ventral hernias: pros and cons. Surg Clin North Am. 2008;88:85–100.

16. Malik K, Bowers SP, Smith CD, et al. A case series of laparoscopic components separation and rectus medialization with laparoscopic ventral hernia repair. J Laparoendosc Adv Surg Tech A. 2009;19(5):607–10.
17. Moazzez A, Mason RJ, Katkhouda N. A new technique for minimally invasive abdominal wall reconstruction of complex incisional hernias: totally laparoscopic component separation and incisional hernia repair. Surg Technol Int. 2010;20:185–91.
18. Saulis AS, Dumanian GA. Periumbilical rectus abdominis perforator preservation significantly reduces superficial wound complications in "separation of parts" hernia repairs. Plast Reconstr Surg. 2002;109(7):2275–80; discussion 2281–2.
19. Clarke JM. Incisional hernia repair by fascial component separation: results in 128 cases and evolution of technique. Am J Surg. 2010;200(1):2–8.
20. Butler CE, Campbell KT. Minimally invasive component separation with inlay bioprosthetic mesh (MICSIB) for complex abdominal wall reconstruction. Plast Reconstr Surg. 2011;128(3): 698–709.
21. Stedman TL. Stedman's medical dictionary. 28th ed. Philadelphia: Lippincott Williams & Wilkins; 2006.
22. Maas SM, de Vries RS, van Goor H, et al. Endoscopically assisted "components separation technique" for the repair of complicated ventral hernias. J Am Coll Surg. 2002;194(3):388–90.
23. Albright E, Diaz D, Davenport D, Roth JS. The component separation technique for hernia repair: a comparison of open and endoscopic techniques. Am Surg. 2011;77(7):839–43.
24. Giurgius M, Bendure L, Davenport DL, Roth JS. The endoscopic component separation technique for hernia repair results in reduced morbidity compared to the open component separation technique. Hernia. 2012;16(1):47–51.
25. Harth KC, Rosen MJ. Endoscopic versus open component separation in complex abdominal wall reconstruction. Am J Surg. 2010;199(3):342–6; discussion 346–7.
26. Rosen MJ, Williams C, Jin J, et al. Laparoscopic versus open-component separation: a comparative analysis in a porcine model. Am J Surg. 2007;194(3):385–9.
27. Harth KC, Rose J, Delaney CP, et al. Open versus endoscopic component separation: a cost comparison. Surg Endosc. 2011;25(9): 2865–70.
28. Latifi R. Nutritional therapy in critically ill and injured patients. Surg Clin North Am. 2011;91:579–93.
29. DiCocco JM, Magnotti LJ, Emmett KP, et al. Long-term follow-up of abdominal wall reconstruction after planned ventral hernia: a 15-year experience. J Am Coll Surg. 2010;210(5):686–95, 695–8.
30. Blatnik JA, Harth KC, Aeder MI, Rosen MJ. Thirty-day readmission after ventral hernia repair: predictable or preventable? Surg Endosc. 2011;25(5):1446–51.
31. Rahbari NN, Zimmermann JB, Schmidt T, et al. Meta-analysis of standard, restrictive and supplemental fluid administration in colorectal surgery. Br J Surg. 2009;96(4):331–41.
32. Hakobyan RV, Mkhoyan GG. Epidural analgesia decreases intraabdominal pressure in postoperative patients with primary intra-abdominal hypertension. Acta Clin Belg. 2008;63(2):86–92.
33. Santos C, Grade P, Ferreira F, et al. The success of epidural analgesia in reducing intra-abdominal pressure in severe acute pancreatitis – a case report. Acta Clin Belg. 2009;64(3):277.
34. Rosen MJ. Atlas of abdominal wall reconstruction. Philadelphia: Elsevier; 2012.
35. Agnew SP, Small Jr W, Wang E, et al. Prospective measurements of intra-abdominal volume and pulmonary function after repair of massive ventral hernias with the components separation technique. Ann Surg. 2010;251(5):981–8.
36. Regner JL, Kobayashi L, Coimbra R. Surgical strategies for management of the open abdomen. World J Surg. 2012;36(3):497–510.
37. Verdam FJ, Dolmans DE, Loos MJ, et al. Delayed primary closure of the septic open abdomen with a dynamic closure system. World J Surg. 2011;35(10):2348–55.
38. Reimer MW, Yelle JD, Reitsma B, et al. Management of open abdominal wounds with a dynamic fascial closure system. Can J Surg. 2008;51(3):209–14.
39. Ferreira F, Barbosa E, Guerreiro E, et al. Fascial closure following severe abdominal compartment syndrome: a case report regarding an efficient combination of dynamic abdominal wall closure and negative pressure wound therapy. Acta Clin Belg. 2009;64(3):275.
40. Weinberg JA, George RL, Griffin RL, et al. Closing the open abdomen: improved success with Wittmann Patch staged abdominal closure. J Trauma. 2008;65(2):345–8.
41. Acosta S, Bjarnason T, Petersson U, et al. Multicentre prospective study of fascial closure rate after open abdomen with vacuum and mesh-mediated fascial traction. Br J Surg. 2011;98(5):735–43.
42. Bjorck M, Bruhin A, Cheatham M, et al. Classification—important step to improve management of patients with an open abdomen. World J Surg. 2009;33(6):1154–7.
43. Jacobsen WM, Petty PM, Bite U, Johnson CH. Massive abdominal-wall hernia reconstruction with expanded external/internal oblique and transversalis musculofascia. Plast Reconstr Surg. 1997;100(2):326–35.
44. Admire AA, Dolich MO, Sisley AC, Samimi KJ. Massive ventral hernias: role of tissue expansion in abdominal wall restoration following abdominal compartment syndrome. Am Surg. 2002;68(5):491–6.
45. Rosen MJ, Reynolds HL, Champagne B, Delaney CP. A novel approach for the simultaneous repair of large midline incisional and parastomal hernias with biological mesh and retrorectus reconstruction. Am J Surg. 2010;199(3):416–20; discussion 420–1.

Laparoscopic Techniques in the Repair of Large Defects

19

Selman Uranues

Introduction

The prevalence of ventral hernia is similar in men and women and increases with age [1]. Hernias occur after 4–11 % of elective operations [2]; in the United States, there are 250,000 incisional hernia operations per year [3]. After emergency surgery, patients are much more likely to develop wound complications and incisional hernias [4, 5]. The recurrence rate is considerably higher, with up to 50 % recurrence after both direct closure of large primary hernias and repair of incisional or recurrent hernia without mesh [4, 5]. Recurrent incisional hernia is a common long-term complication after open repair of large abdominal wall hernias. Although repair of incisional and recurrent hernia remains a challenge in general surgery, the use of mesh may reduce the recurrence rate to 11–18 % [6]. Mesh repair in open technique requires a large incision and extensive fascial dissection on both sides, entailing large wounds and a high rate of wound complications, such as seromas and infections [7].

Large abdominal wall defects are usually caused by an incisional hernia recurrence following multiple laparotomies; these defects are technically challenging because of the destruction of abdominal wall structures and the presence of extensive intra-abdominal adhesions. It is suspected that both the hernia and the adhesions have an impact on gastrointestinal quality of life (GIQLI) [8]. Autopsy data indicate that adhesions are to be expected in 67 % of cases with a previous laparotomy [9]. Clinically, adhesions were found on laparotomy in 93 % of patients who had previously undergone one or more laparotomies [9].

In nonspecialized centers, it is often thought that these patients are not good candidates for laparoscopy. Recent literature confirmed that laparoscopic repair of ventral hernias can have a low recurrence rate, minimal postoperative morbidity, early mobilization, and shorter hospital stay [10–13].

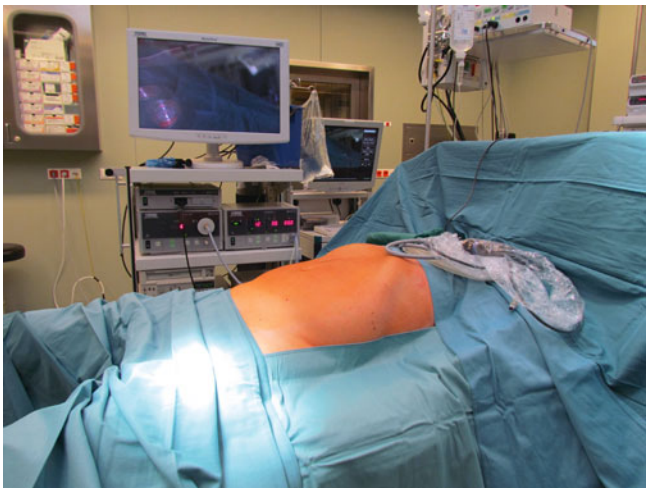


Fig. 19.1 Setting in the operating room with the surgical team on one side and the laparoscopy tower on the other

Patient Preparation, Equipment, and Positioning

Patients are instructed to shower with an antiseptic wash lotion (Betadine® liquid soap) the evening and morning before the operation. We view this as an important measure for infection prophylaxis, especially with obese patients. Preoperative bowel preparation is not needed. The patient should abstain from food for 6 h and from liquids for 2 h before the operation.

The surgical team almost always consists of the surgeon and a camera assistant, who stands with the surgeon on the same side of the patient. With few exceptions, abdominal wall hernias are operated from the side with the laparoscopy tower opposite, usually around the level of the umbilicus at the beginning of the procedure (Fig. 19.1). It is ideal when the laparoscopy equipment is ceiling mounted and can be

S. Uranues, MD, FACS
Department of Surgery, Medical University of Graz,
Auenbruggerplatz 29, 8036 Graz, Austria
e-mail: selman.uranues@medunigraz.at

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_19, © Springer Science+Business Media New York 2013

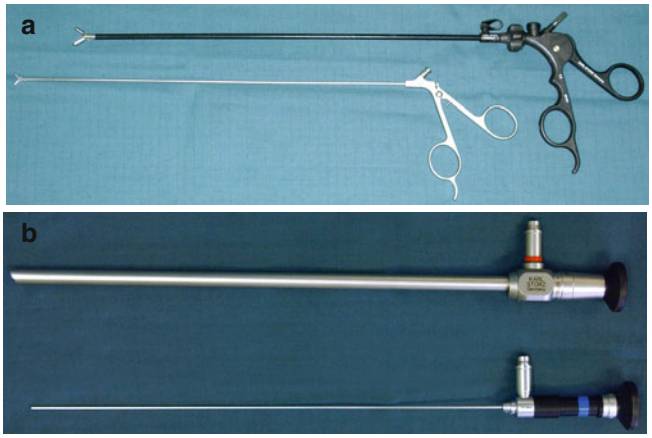


Fig. 19.2 (**a**, **b**) Instruments and 5- and 2-mm optics

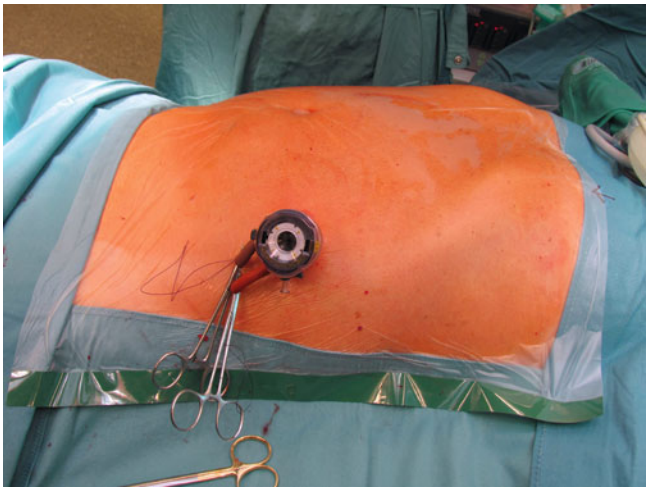


Fig. 19.3 Fixation of the first trocar with a suture pulled through a tourniquet

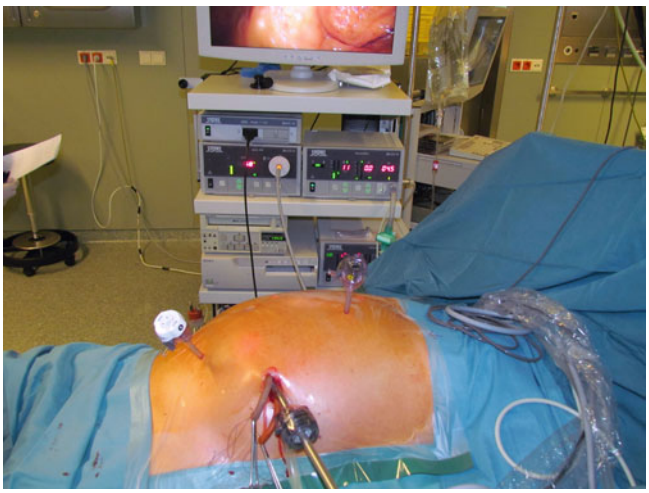


Fig. 19.4 Trocar sites

shifted up and down easily or when there are several monitors around the operating table. A modern optical-and-camera system is essential for the patient's safety. The diameter of the optic (10, 5, or a minioptic of 2–3.5 mm) depends on the surgeon's preference and the extent and location of the hernia. A 30° or 45° optic is ideal as it can be turned easily for optimal viewing of parts of the abdominal wall or intestines that may be hidden behind adhesions. The angled optic facilitates the view of the abdominal wall and manipulation of the mesh, especially while it is being fixed and when the transfascial sutures are pulled through. During the operation, almost without exception only two atraumatic graspers, a dissector and curved scissors, are used. The diameter of the trocars depends on the instruments preferred (Fig. 19.2).

Because an approximately 2-cm incision is always needed for the later introduction of the mesh, a 10-mm port should always be introduced first as the optic trocar. It is safest to use an open technique to introduce this trocar. To avoid loss of gas caused by open access, the edges of the fascia are adapted to the trocar with one or two sutures passing through tourniquets (Fig. 19.3). The camera should be as far as possible from the hernia opening and lie between the two working trocars. To this end, a site halfway between the costal arch and the iliac crest on the right or left flank is usually chosen. The two working trocars with a diameter of 5 mm or less are placed as far apart as possible to establish optimal triangulation. The trocars should be introduced at an angle of 60° in the direction of the hernia so that the abdominal wall and hernia sac can be reached more easily for safe adhesiolysis (Fig. 19.4). Here, prominent landmarks such as the ribs, pelvic bone, and pubic bone should be borne in mind as they can limit the maneuverability of the trocars and instruments.

One hour before surgery, the patient receives intravenous antibiotic prophylaxis. A Foley catheter is inserted routinely and left in place until the patient is mobilized. A nasogastric tube is inserted when anesthesia is induced and removed at the end of the operation. The patient always undergoes surgery in the supine position. On the surgeon's side, the arm is fixed to the patient's flank to allow as much space as possible for the surgical team. The patient should be so stabilized on the operating table that the table can be turned in any direction during the procedure. In this way, the intestines can be displaced by gravity, facilitating easy manipulation during adhesiolysis. The abdomen is widely prepped on either side, above the xiphoid cephalad and below the pubis onto the upper thighs. The abdomen is draped sterilely, and the abdominal skin is completely covered with a transparent adhesive drape.

Surgical Technique

Surgery for abdominal wall hernias is generally standardized and consists of two steps: adhesiolysis and repair of the hernia with intraperitoneal mesh.

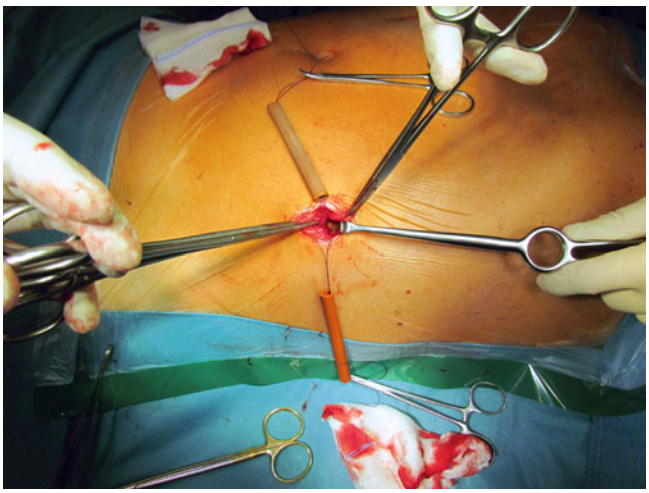


Fig. 19.5 Access to the abdominal cavity in open technique

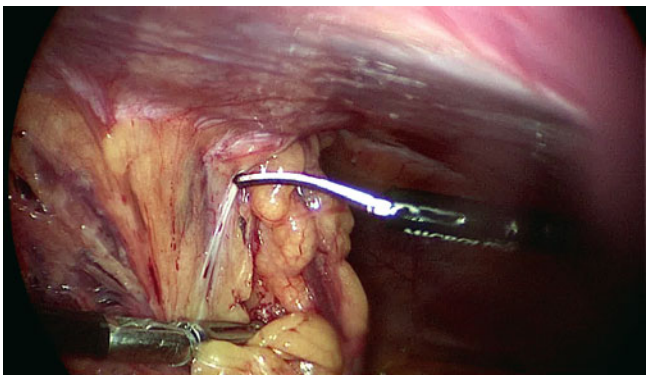


Fig. 19.6 Careful dissection of the adhesions with push-and-pull technique using a grasper and scissors

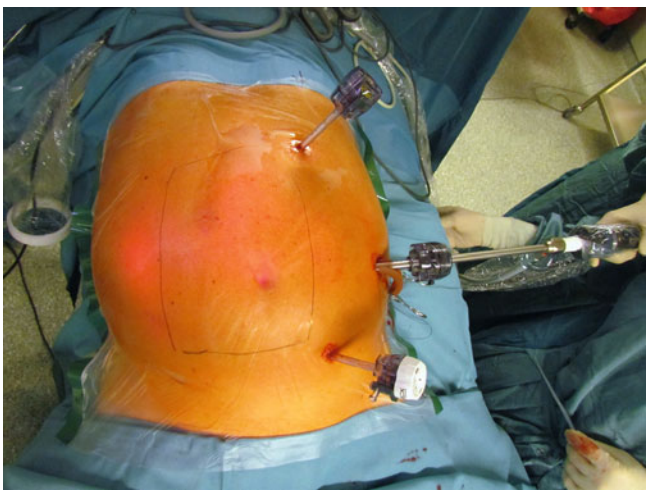


Fig. 19.7 The area covered by the mesh is marked on the abdominal wall

The first skin incision is made at the greatest possible distance from the scars of previous laparotomies. This corresponds to a location on the right or left flank far lateral to the rectus muscle along the anterior axillary line. The dissection is deepened, incising the fascia and carefully splitting the muscles until the peritoneal cavity is accessed under direct vision. Before the first trocar is inserted under direct vision, two strong, nonresorbable sutures are passed through all the fascia and muscle layers of the abdominal wall and fixed with a tourniquet (Fig. 19.5). Then, after digital palpation of the peritoneal space and separation/loosening of nearby adhesions, a 10/11 trocar is introduced anterior to the large intestine. The fascial sutures are pulled taut with the tourniquets so that no gas is lost during the operation.

The pneumoperitoneum is set at 12 mmHg, followed by the introduction of a 30° telescope. Two additional 5-mm or smaller ports are introduced under visual control. It is important that the working trocars in the upper and lower abdomen are so placed that there is sufficient distance for placement of the mesh, with all four abdominal quadrants within reach.

If there are adhesions, exposure is achieved by pushing and pulling with atraumatic graspers. Grasping with the instruments should be performed carefully as long as the structures in the adhesions are not well defined (Fig. 19.6). It is always possible that there are loops of intestine in or behind the fatty tissue of the omentum. Sometimes, it is helpful when the surgeon uses the grasper with the right hand and presses against the abdominal wall with the left hand in the area of the adhesions. This can lessen the distance to the adhesions in the uplifted dome of the abdominal cavity. The intra-abdominal gas infiltrates into the fatty tissue and adhesions, forming a soap-like foam that makes it easier to loosen the adhesions.

Matted adhesions or bands are divided by sharp dissection with cold scissors. Under normal circumstances, energy-based devices are not used to divide adhesions, although these devices can be useful when the teres hepatic ligament has to be severed or the urinary bladder must be separated from the anterior abdominal wall. The goal of the lysis of adhesions is to expose 4–5 cm of anterior abdominal wall around the fascial defect. Care should be taken to avoid any unnecessary dissection of adhesions within the bowel loops. With obese patients and those with numerous scars from previous operations, it may not be possible to probe and detect all herniations prior to surgery. For this reason, adhesions should be lysed in the areas of all the scars so any such undiagnosed hernias are not overlooked. The hernia sac is always left in place.

The fascial defect is determined by probing and pressing through the abdominal wall. The size of the mesh is determined by briefly releasing the pneumoperitoneum and using a pen to mark an area extending 4–5 cm all around the hernia and measuring it (Fig. 19.7). Then, the pneumoperitoneum is reestablished, and the entire team changes gloves. Only then is an expanded polytetrafluoroethylene (ePTFE) GORE® DUALMESH® W.L. Gore & Associates, Inc. AP2400-EN146 placed on the table and tailored to fit the measurements. The mesh is marked on the side toward the fascia, with arrows

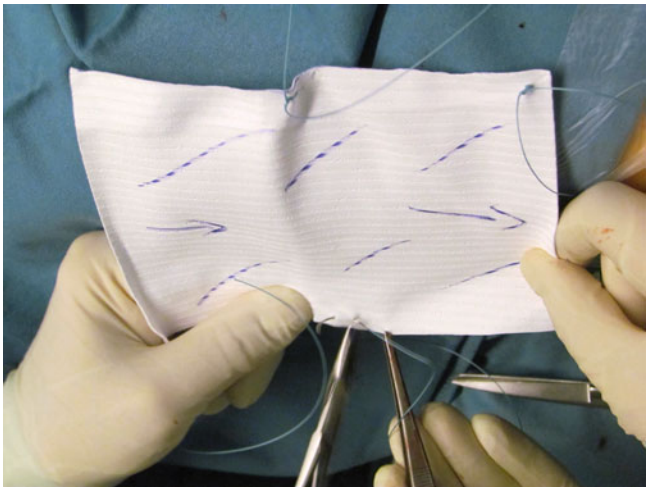


Fig. 19.8 Dualmesh with transfascial fixation sutures

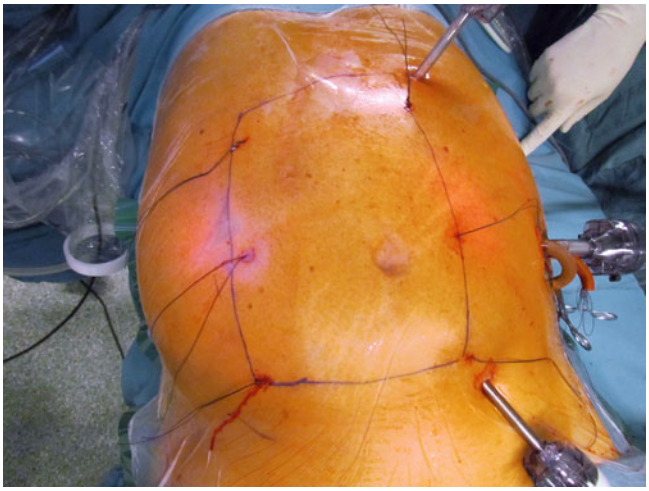


Fig. 19.10 View of the transfascial sutures after they are knotted

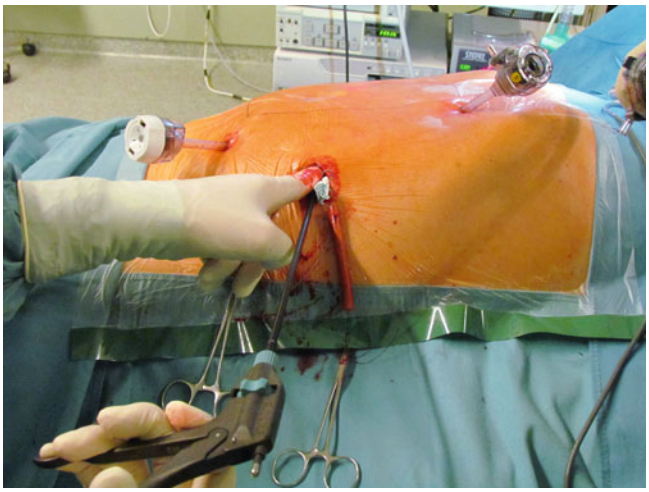


Fig. 19.9 Introduction of the mesh through the site of the optic trocar

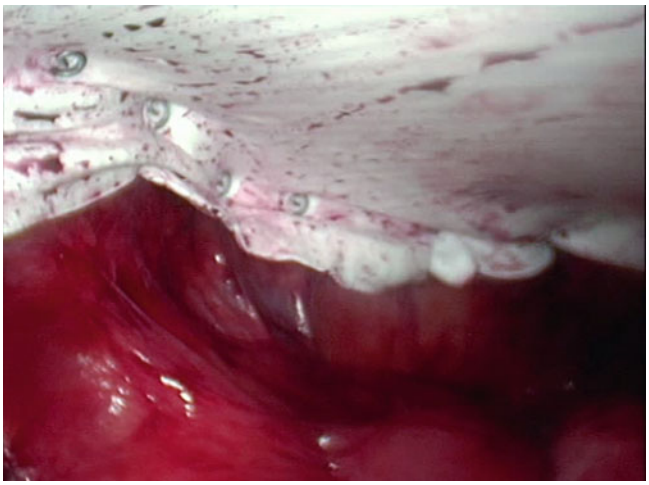


Fig. 19.11 Intra-abdominal view of the Dualmesh after completed fixation

indicating the cranial or caudal end. On the four corners and in the middle of them, a total of eight nonresorbable sutures are placed in a U shape and knotted (Fig. 19.8). The sutures should be cut to a length of 10–15 cm so that later it will be easier to grasp them with the suture passer and pull them out. The sutures are then placed in the mesh, which is rolled up along the long edge. The optic trocar is removed, and the mesh is inserted digitally or with a grasper through this incision into the abdominal cavity (Fig. 19.9).

At this point, the trocar is replaced, the tourniquets are drawn tight, and the pneumoperitoneum is reestablished. The mesh can be unrolled and put into position with the fascial side facing up. First, the cranial sutures on the opposite side are pulled through with a suture passer or Endo Close™ Covidien,15 Hampshire Street, Mansfield, MA 02048. To this end, a 2-mm incision is made with a pointed blade in the marked area. Both sutures of a pair come out through the same skin incision, but through a separate fascial puncture, so that there is a tissue bridge of 0.5–1 cm between the two strands of the same suture pair. After both strands are drawn through separate punctures one after another, the mesh is drawn to the abdominal wall and fixed by pulling the threads. In the same way, the strands at the next site are drawn through the fascia through small skin incisions, taking care that the mesh is pulled taut. Only when the last sutures have been pulled through are they knotted, with the knots pushed in to the level of the fascia (Fig. 19.10).

The distance between the transfascial sutures is fixed with spiral tacks. At this stage, the surgeon uses the nondominant hand to press the tip of the tacking device (ProTac™) Covidien,15 Hampshire Street, Mansfield, MA 02048 as close as possible onto the abdominal wall to ensure secure fixation of the mesh on the fascia (Fig. 19.11). The mesh can also be fixed with absorbable tacks. This, however, contradicts our philosophy of hernia repair using nonresorbable materials if the fixation tacks are absorbable and will

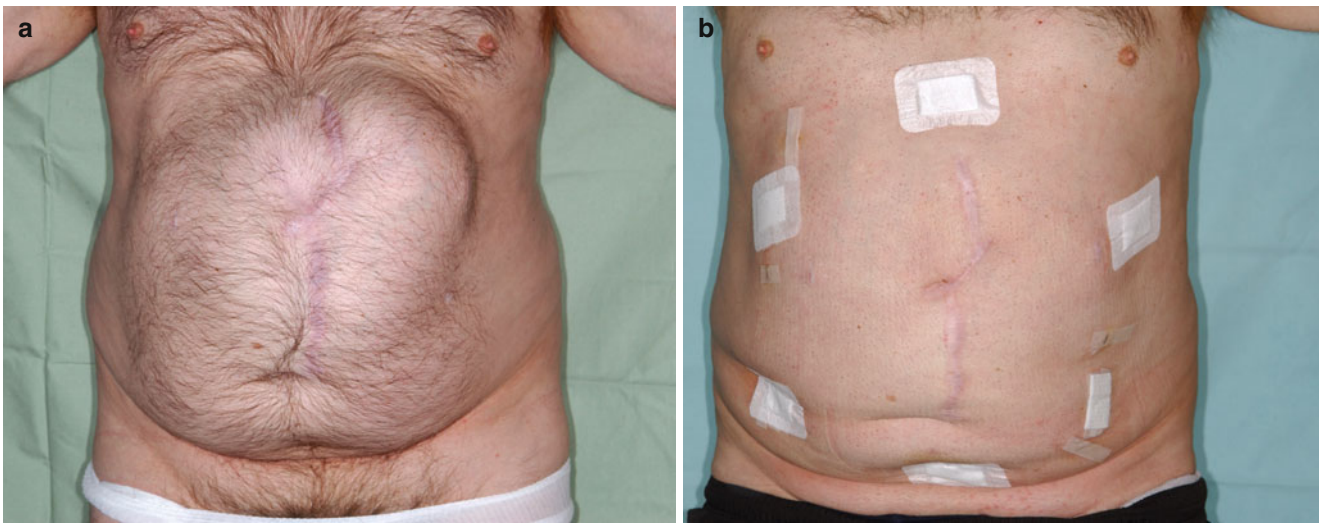


Fig. 19.12 (**a**) Large incisional hernia after several attempts at open repair. (**b**) Postoperative view after laparoscopic repair

disappear after a certain time. There is as yet no convincing evidence for the argument that absorbable tacks cause fewer adhesions or nonabsorbable tacks cause significant adhesions or small bowel obstruction.

When fixation is completed, the abdominal cavity is again inspected for occult bleeding or intestinal wall lesions. Any blood is suctioned off. All the instruments are removed, and the pneumoperitoneum is released. All incisions larger than 5 mm are closed with fascial sutures.

Postoperative Care

In the case of a longer operation, a second dose of antibiotics is given after surgery. Patients are mobilized after 4–6 h and receive fluids orally. On the first postoperative day, food is given as tolerated. It must be borne in mind that in the first postoperative days the transfascial fixation sutures cause severe pain, and analgesic therapy should be used accordingly. The addition of anti-inflammatory medication improves the analgesic effect and helps reduce swelling at the surgical site. Patients with larger hernias should wear an elastic abdominal girdle for 2–3 months. Many patients develop a seroma or hematoma in the hernia sac that has been left in place. These only rarely are clinically evident and should not be drained or punctured. The girdle prevents the development of larger seromas or helps large collections to regress.

Complications and Outcome

Significant complications can occur during the introduction of the trocars or adhesiolysis. Significant bleeding from the abdominal wall can be avoided if attention is paid to the anatomical position of the epigastric vessels. Bleeding from these vessels requires enlargement of the trocar incision and safe closure of the vessel under direct vision. Smaller bleeds can usually be stopped with electrocoagulation. Because the first trocar is introduced with an open technique, accidental visceral injuries are rare and are recognized immediately. Usually, these are serosal defects or rarely smaller full-thickness bowel lesions that can be sutured. Small serosal defects or minor lacerations of the small intestine during adhesiolysis can be sutured laparoscopically without conversion to open technique.

If there is a larger intestinal laceration with significant spillage of intestinal content, especially if the large bowel is involved, conversion should be considered. The problem in this case is not the safe repair of the lesion but the possibility of contamination of the mesh. Thermal injury of the bowel is a serious problem and should be attended to, possibly with excision of the intestinal wall and conversion to open hernia repair. A mesh infection is a severe complication and usually necessitates antibiotic therapy and removal of the mesh [14]. In general, prevention of mesh infection remains the best strategy.

Other postoperative complications influencing the outcome are postoperative ileus and thromboembolic events. The incidence for both is between 1 and 2 % [8, 10]. The recurrence rate mainly depends on the number of previous repair attempts and the size of the hernia. In general, the recurrence rate tends to be lower than 5 % [8, 13].

In summary, abdominal wall hernias, both primary and incisional, are a common problem. The advances in laparoscopic technique as well as mesh engineering have had a positive influence on results Fig. 19.12a, b. Although laparoscopic repair of large abdominal wall hernias may be challenging, it has the potential to be considered as a primary approach, regardless of patient status or hernia complexity [10]. Today, we can say that laparoscopic technique is the standard method for the treatment of large primary and incisional hernias.

References

1. National Centre for Health Statistics. Combined surgery data (NHDS and NSAS) data highlights. 2011. http://www.cdc.gov/nchs/about/major/hdasd. Accessed 1 Oct 2011.
2. Mehrabi M, Jangjoo A, Tavoosi H, Kahrom M, Kahrom H. Long-term outcome of Rives-Stoppa technique in complex ventral incisional hernia repair. World J Surg. 2010;23:4–15.
3. Breuing K, Butler CE, Ferzoco S, Franz M, Hultman CS, Kilbridge JF, et al. Incisional ventral hernias: review of the literature and recommendations regarding the grading and technique of repair. Surgery. 2010;148:544–58.
4. Rudmik LR, Dchieman C, Dixon E, Debru E. Laparoscopic incisional hernia repair: a review of the literature. Hernia. 2006;10:110–9.
5. Langer S, Christiansen J. Long-term results after incisional hernia repair. Acta Chir Scand. 1985;151:217–9.
6. Heniford BT, Park A, Ramshaw BJ, Voeller G. Laparoscopic ventral and incisional hernia repair in 407 patients. J Am Coll Surg. 2000;190:645–50.
7. Forbes SS, Eskicioglu C, McLeod RS, Okrainec A. Meta-analysis of randomized controlled trials comparing open and laparoscopic ventral and incisional hernia repair with mesh. Br J Surg. 2009;96:851–8.
8. Uranues S, Salehi B, Bergamaschi R. Adverse events, quality of life, and recurrence rates after laparoscopic adhesiolysis and recurrent incisional hernia mesh repair in patients with previous failed repairs. J Am Coll Surg. 2008;207:663–9.
9. Menzies D, Ellis H. Intestinal obstruction from adhesions—how big is the problem? Ann R Coll Surg Engl. 1990;72:60–3.
10. Birch DW. Characterizing laparoscopic incisional hernia repair. Can J Surg. 2007;50:195–201.
11. Harold K, Mekeel K, Spitler J, Frisella M, Merritt M, Tessier D, et al. Outcomes analysis of laparoscopic ventral hernia repair in transplant patients. Surg Endosc. 2009;23:1835–8.
12. Sajid MS, Bokhari SA, Mallick AS, Cheek E, Baig MK. Laparoscopic versus open repair of incisional/ventral hernia: a meta-analysis. Am J Surg. 2009;197:64–72.
13. Phillips JD, Nagle AP. Minimally invasive approaches to incisional hernia repairs. J Long Term Eff Med Implants. 2010;20:117–28.
14. Collage RD, Rosengart MR. Abdominal wall infections with in situ mesh. Surg Infect. 2010;11:311–8.

Perioperative Surgical Consideration of Patient Undergoing Abdominal Wall Reconstruction

20

Ruben Peralta and Rifat Latifi

Introduction

Component separation is a surgical technique commonly used for closure of the open abdomen in the semiacute setting, and in the later stage of the continuum of care, it is part of scheduled procedures for abdominal wall reconstruction in patients with major abdominal wall defect. In a published article from 1951, Dr. Alfonso Albanese originally described an abdominal eventration after exploratory laparotomy in a 4-year-old child. The operative plan consisted of three steps: (1) opening and resecting the hernia sac with adhesiolysis; (2) freeing of the rectus abdominis muscles by incising, in a longitudinal incision, the external oblique muscles; and (3) abdominal wall closure by approximation of the rectus abdominis muscle edges to the midline [1]. More recently, the technique was popularized by Ramirez and colleagues in 1990 [2]. With cadavers, they demonstrated that, by using an avascular plane, the external oblique muscle could be separated from the internal oblique muscle, thereby producing an advancement of approximately 10 cm around the waistline and facilitating the closure.

R. Peralta, MD, FACS
Trauma Section, Department of Surgery,
Hamad General Hospital,
Doha, Qatar
e-mail: rperaltamd@gmail.com

R. Latifi, MD, FACS (✉)
Department of Surgery,
The University of Arizona,
1501 N. Campbell Ave.,
Tucson, AZ 85724, USA

Trauma Section, Department of Surgery
Weill Cornell Medical College,
Hamad General Hospital,
Doha, Qatar
e-mail: latifi@surgery.arizona.edu

Preoperative Preparation

We addressed in detail the preoperative evaluation and optimization of the patient undergoing a major surgical procedure to achieve reconstruction of the abdominal wall defect in Chap. 5 of this book.

Indications for and Timing of Surgery

While one particular procedure is not ideal for closure of all types of abdominal wounds and defects, component separation is indicated in abdominal wall reconstruction after failure of abdominal wound closure after laparotomy and in midline abdominal wall defects where the skin coverage over the hernia is intact (type 1) [3]. Additional considerations for abdominal wall closure that require various reconstruction techniques include large defect size (>40 cm^2); absence of stable skin coverage (type 2); recurrence of defect after prior closure attempts; infected or exposed mesh; in patients with a history of radiation, chronic corticosteroids use where the abdominal wall tissues may be compromised; and patients with major concomitant gastrointestinal tract complications (i.e., enteroatmospheric fistulas) [3–10].

As with any complex medical condition, the timing of the procedure depends on the preoperative evaluation, the physiological condition of the patient, and the anatomical conditions of the tissues. The procedure is not recommended in the acute setting because, during this period, most of the patients experience systemic inflammatory response syndrome, which is associated with frequent visceral or abdominal wall edema, and closure of the abdomen could prompt the development of abdominal compartment syndrome. Also, before embarking on major abdominal wall reconstruction, no hint of intra-abdominal sepsis can be present (Fig. 20.1a, b). As a practical point, the presence of the so-called pinch sign (easy retraction of the skin or skin graft over the defect on examination) is a good indicator that the adhesions, which usually follow multiple laparotomies (damage control surgery), are

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_20, © Springer Science+Business Media New York 2013

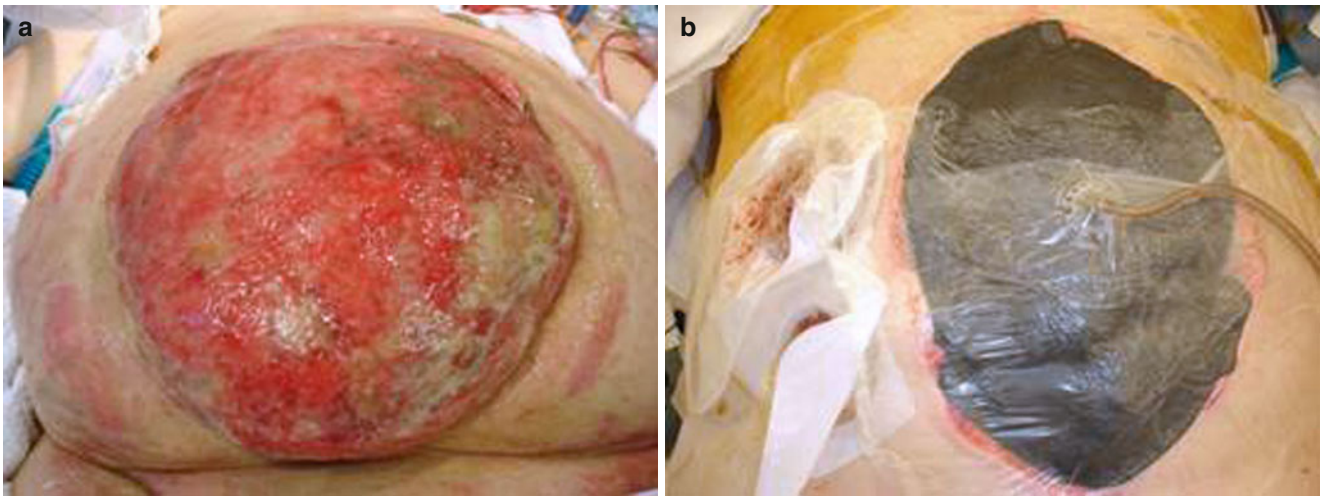


Fig. 20.1 (**a**) Frozen abdomen in acute setting. (**b**) Complex open abdomen managed by vacuum-assisted closure

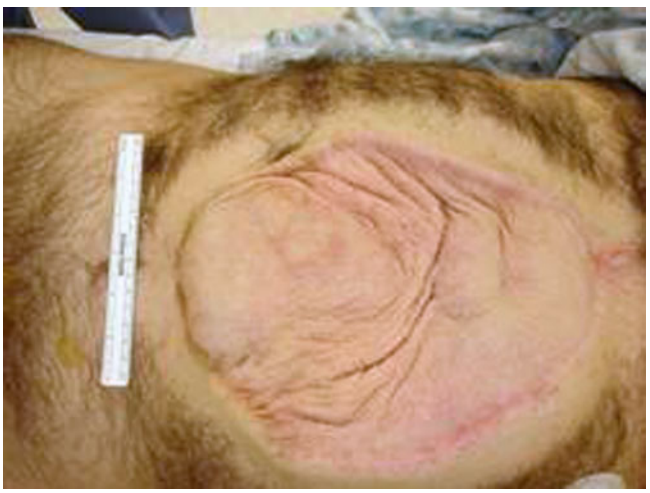


Fig. 20.2 Large abdominal wall defect managed by skin graft. Lax skin covering the fascial defect is noticed

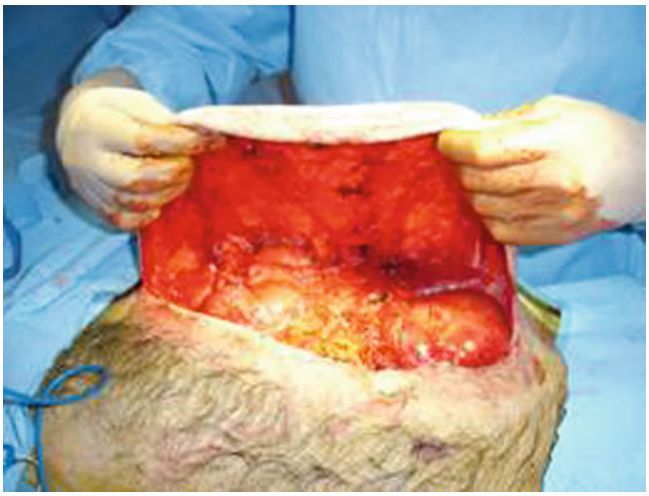


Fig. 20.3 Skin graft dissection and adhesiolysis is undertaken with care to avoid bowel injuries

subsiding, and that it is a good time to schedule the abdominal reconstruction (Fig. 20.2). The optimal timing for abdominal wall reconstruction is not clearly defined, but in our experience, we have observed that adhesions are less prominent 6–12 months after the first procedure.

Operative Approach

The component separation technique is one of the most common methods employed for the closure of complex midline abdominal wall defects. Depending on training and your institution's arrangements, the procedure is usually performed in conjunction with a plastic surgeon. The surgical approach depends on the size and location of the defect and the anatomical configuration of the remainder of the abdominal wall defect [2, 4].

The abdomen is entered via a midline incision if skin closure was achieved previously or if a temporary closure had been established. The fascial defect should be measured again at this time. Routinely, we modified this approach by entering through the edges of the coverage when a skin graft had been used previously as coverage of the skin defect. Adhesiolysis between skin coverage (skin graft or any type of temporary closure) and the bowel is achieved with care to minimize any enterotomy (Fig. 20.3).

Intraoperative Considerations

Adhesiolysis of the intra-abdominal viscera is completed (Fig. 20.4). If enterotomy occurs, it should be recognized and promptly repaired. A subcutaneous dissection is performed as laterally as possible, taking into consideration not

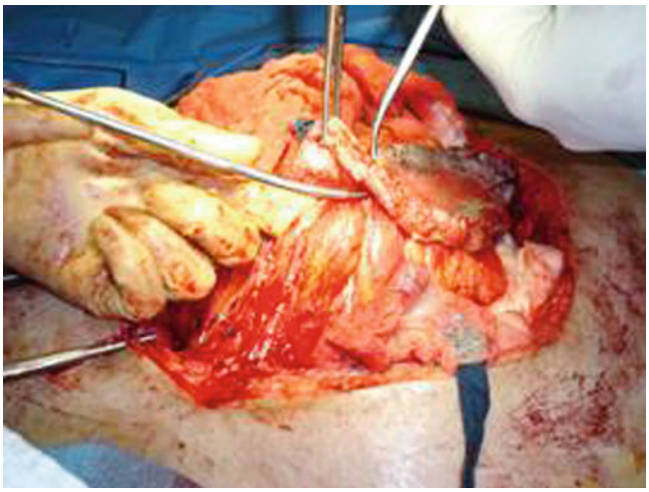


Fig. 20.4 Adhesiolysis of bowel is performed in sharp and blunt fashion. Care should be taken to avoid any enterotomies during the dissection

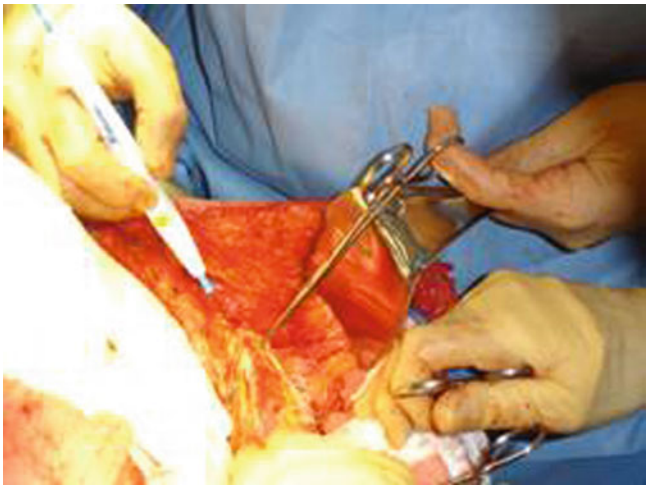


Fig. 20.5 After elevation of skin flaps in a lateral direction, the incision of the external oblique aponeurosis is divided with the electrocautery device in a longitudinal fashion, creating an approximately 10-cm advancement in the waistline

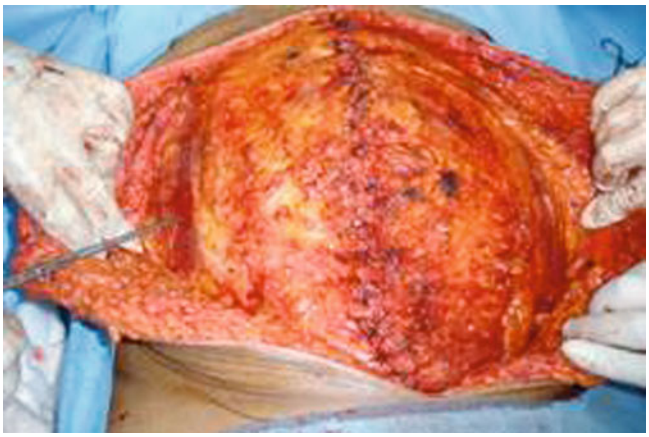


Fig. 20.6 Closure of a large abdominal wall defect using the component separation technique with approximation of the midline by suturing together the rectus abdominal muscles

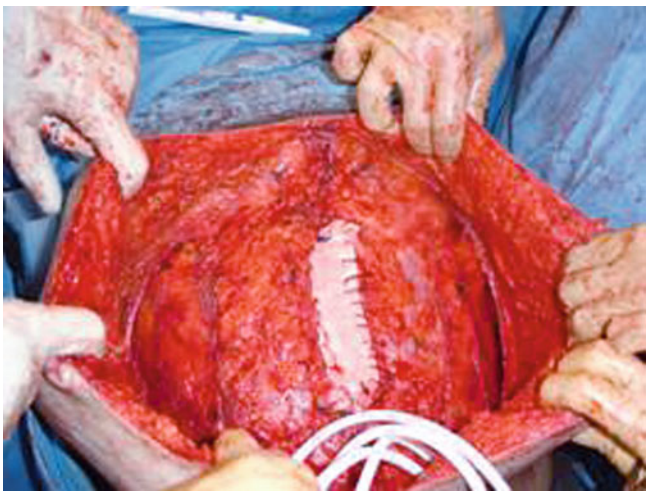


Fig. 20.7 Closure of the abdomen without tension by a partial component separation technique using biological mesh as a bridge in a 67-year-old patient after repair of a ruptured abdominal aortic aneurysm. The patient was scheduled for a planed abdominal wall reconstruction 9 months after the initial operation

to compromise the vascular supply of the flap. Attention is turned to the aponeurosis of the external oblique muscle, which is transected longitudinally about 2 cm lateral to the lateral edge of the rectus sheath (Fig. 20.5). This will give approximately 10 cm on each side, and the fascia is closed in the midline with running polydioxanone suture (PDS™-loop, Ethicon, Somerville, NJ) (Fig. 20.6). The lateral fascia gap created could be selected and reinforced using biological or synthetic mesh. If unable to approximate the fascia without tension, we prefer to use biological material, such as acellular dermal matrix (AlloDerm® or Strattice™, LifeCell, Branchburg, NJ) (Fig. 20.7). The use of prosthetic material is indicated in patients with elevated intra-abdominal pressure. Other synthetic materials (Vicryl™, Ethicon; Gore-Tex®, W.L. Gore & Associate, Newark, DE; etc,) could be used as well. Intraoperative measurement of peak inspiratory pressure is one of the most useful indicators that correlates with elevated intra-abdominal pressure; constant communication with the anesthesiologist is advised, especially at the time of closure. At the end of the procedure, the skin and subcutaneous tissue are approximated over three to four large suction drains to minimize seroma formation. The patient may be discharged home with the drains in place and should be followed as an outpatient until the drainage is less than 25 mL/day and the drains are removed (Fig. 20.8).

There are various modifications of the abdominal wall closure. One popular modification was described by the Memphis group and is termed the "separation of parts." Another variation is the "open-book" technique described by

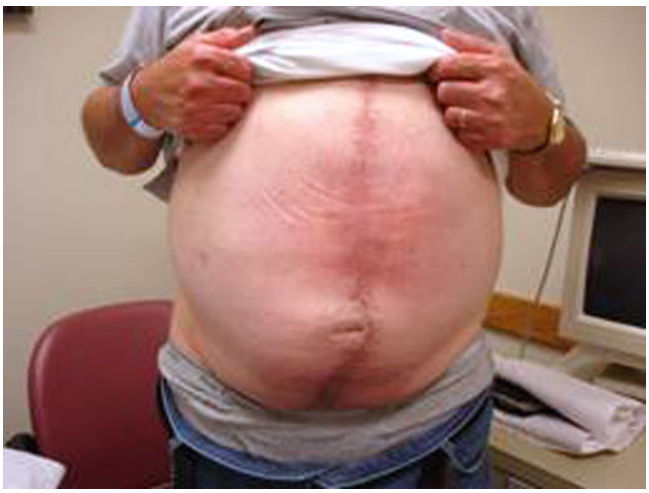


Fig. 20.8 Same patient as in Fig. 20.7, 6 months postoperatively

a group at the University of Virginia in Charlottesville; in addition to the lateral release of the external oblique, the rectus fascia is flipped into the midline using the linea alba as the fulcrum to extend the midline [2, 4, 7, 11–16]. Recently, a minimally invasive surgical technique has been employed, and the rates of recurrence were similar [17].

Other Tissue Transfer

Various tissue transfer techniques have been popularized, and these include the use of myocutaneous flap, free tissue flap transfers, and tension fascia lata, among others. Irrespective of the techniques used, vascularized flaps provide autologous tissue coverage and could avoid the use of foreign material at the closure site. In general, pedicle flaps are an alternative option for small defect repair. With large thoracoabdominal wall defects and in collaboration with our plastic surgery colleagues, coverage could be obtained in one single-stage reconstructive procedure with free flaps [9, 18–28].

Postoperative Care

All patients should have good pain management control (epidural anesthesia or patient-controlled analgesia, PCA), continued perioperative antibiotic prophylaxis for 24 h, and deep venous thrombosis prophylaxis as previously indicated. We make great efforts to encourage incentive spirometry in our patients to minimize postoperative atelectasis. The wound should be inspected daily by the operating surgeon. Deep-vein thrombosis (DVT) prophylaxis should be initiated preoperatively and continued postoperatively until the patient is fully ambulating.

Postoperative Complications

Wound complications are frequent and include hematomas, seroma formation, wound infections, and skin necrosis. Pulmonary complications include atelectasis and pneumonia. Mortality can occur, and it is due primarily to postoperative cardiovascular events. The risk of recurrence in patients who undergo abdominal wall reconstruction increases with time; therefore, we recommend long-term follow-up [18, 29].

Summary

Perioperative care of patients requiring abdominal wall reconstruction is challenging and should be performed by a multidisciplinary team led by the surgeon. Surgical technique should be individualized. Component separation is the most common procedure used by our group in the management of medium- and large-size abdominal wall defects. We prefer the use of biological mesh when prosthetic reinforcement is indicated. Various modifications of the technique have been developed. For extensive abdominal wall defects, the use of tissue transfer is important for accomplishing abdominal closure in one procedure.

References

1. Albanese AR. Eventracion mediana xifoumbilical gigante: metodo para su tratamiento. Rev Asoc Med Argent. 1951;65:376–8.
2. Ramirez OM, Ruas E, Dellon AL. "Component separation" method for closure of abdominal-wall defects: an anatomic and clinical study. Plast Reconstr Surg. 1990;86:519–26.
3. Mathes SJ, Steinwald PM, Foster RD, et al. Complex abdominal wall reconstruction: a comparison of flap and mesh closure. Ann Surg. 2000;232:586–94.
4. De Vries Reilingh TS, van Goor H, Charbon JA, et al. Repair of giant midline abdominal wall hernias: "components separation technique" versus prosthetic repair. Interim analysis of a randomized controlled trial. World J Surg. 2007;31:756–63.
5. Diaz JJ, Cullinane DC, Dutton WD, et al. The management of the open abdomen in trauma and emergency general surgery: part 1—damage control. J Trauma. 2010;68(6):1425–8.
6. Latifi R, Gustafson M. Abdominal wall reconstruction in patients with enterocutaneous fistulas. J Trauma Emerg Surg. 2011;37:241–50.
7. Diaz JJ, Dutton WD, Ott MM, et al. Eastern Association for the Surgery of Trauma: a review of the management of the open abdomen—Part 2: management of the open abdomen. J Trauma. 2011;71(2):502–12.
8. Latifi R, Turegano F. Current management of enterocutaneous fistulas. Eur J Trauma Emerg Surg. 2011;37:207–8.
9. Leppaniemi A, Tukiainen E. Planned hernia repair and late abdominal wall reconstruction. World J Surg. 2012;36:511–5.
10. Leppaniemi A. The hostile abdomen—a systematic approach to a complex problem. Scand J Surg. 2008;97(3):218–9.
11. Vargo D. Component separation in the management of the difficult abdominal wall. Am J Surg. 2004;188:633–7.

12. Fabian TC, Croce MA, Pritchard FE, et al. Planned ventral hernia. Staged management for acute abdominal wall defects. Am Surg. 1994;219:643–50.
13. Kushimoto S, Yammamoto Y, Aiboshi J, et al. Usefulness of the bilateral anterior rectus abdominis sheath turnover flap method for early fascial closure in patients requiring open abdominal management. World J Surg. 2007;31:2–8.
14. Sailes FC, Walls J, Guelig D, et al. Synthetic and biological mesh in component separation. A 10-year single institution review. Ann Plast Surg. 2010;64:696–8.
15. Espinosa-de-los-Monteros A, Franssen B, Orozco V, et al. Components-separation technique for closure of transverse non-midline abdominal wall incisional hernia. J Plast Reconstr Aesthet Surg. 2011;64:264–7.
16. Ennis LS, Young JS, Gampper TJ, et al. The "open-book" variation of component separation for repair of massive midline abdominal wall hernia. Am Surg. 2003;69(9):733–42.
17. Harth KC, Rosen MJ. Endoscopic versus open component separation in complex abdominal wall reconstruction. Am J Surg. 2010;199(3):342–6.
18. den Hartog D, Dur AH, Tuinebreijer WE, et al. Open surgical procedures for incisional hernias. Cochrane Database Syst Rev. 2008; 16:CD006438.
19. Muramatsu K, Ihara K, Taguchi T. Selection of myocutaneous flaps for reconstruction following oncologic resection of sarcoma. Ann Plast Surg. 2010;64(3):307–10.
20. Servant JM, Arnault E, Revol M, Danino A. Reconstruction of large thoracoabdominal defects using two-stage free tissue transfers and prosthetic materials. J Plast Reconstr Aesthet Surg. 2006;59(4):360–5.
21. Erni D, Harder YD. The dissection of the rectus abdominis myocutaneous flap with complete preservation of the anterior rectus sheath. Br J Plast Surg. 2003;56(4):395–400.
22. Fulda GJ, Khan SU, Zabel DD. Special issues in plastic and reconstructive surgery. Crit Care Clin. 2003;19(1):91–108.
23. Arnez ZM, Khan U, Pogorelec D, Planinsek F. Breast reconstruction using the free superficial inferior epigastric artery (SIEA) flap. Br J Plast Surg. 1999;52(4):276–9.
24. Wei CY, Chuang DC, Chen HC, et al. The versatility of free rectus femoris muscle flap: an alternative flap. Microsurgery. 1995;16(10): 698–703.
25. Hill L, Nahai F, Vasconez LO. The tensor fasciae latae myocutaneous free flap. Plast Reconstr Surg. 1978;61:517–22.
26. Nahai F, Hill L, Hester TR. Experiences with the tensor fascia lata flap. Plast Reconstr Surg. 1979;63(6):788–99.
27. Wong CH, Lin CH, Fu B, et al. Reconstruction of complex abdominal wall defects with free flaps: indications and clinical outcome. Plast Reconstr Surg. 2009;124:500–9.
28. Tukianen E, Leppaniemi A. Reconstruction of extensive abdominal wall defects with microvascular tensor fascia lata flap. Br J Surg. 2011;98:880–4.
29. Peralta R, Latifi R. Long-term outcomes of abdominal wall reconstruction. What are the real numbers? World J Surg. 2012;36:534–8.

21 Abdominal Compartment Syndrome and Hypertension in Patients Undergoing Abdominal Wall Reconstruction

Ajai K. Malhotra

Introduction

Normal pressure within the abdominal cavity varies between subatmospheric and 6.5 mmHg [1]. Intra-abdominal hypertension (IAH) occurs when the contents of the abdomen together exceed the space volume available within the abdominal cavity. It is defined as a sustained elevation of the intra-abdominal pressure (IAP) to greater than 12 mmHg on two separate measurements at least 6 h apart [2]. While transient elevations of IAP are well tolerated, sustained elevations can have significant deleterious effects on organ system function. The association of IAH and organ system dysfunction was recognized as early as the mid-nineteenth century [3]. However, the acceptance of the syndrome of IAH with organ system dysfunction as a distinct nosologic entity—abdominal compartment syndrome (ACS)—had to wait until the late twentieth century [4]. Abdominal compartment syndrome is defined as peak IAH of greater than 20 mmHg on two separate measurements at least 6 h apart in association with dysfunction of one or more organ systems that was not present previously [2]. In other words, the elevation of IAP resulted in the organ system dysfunction. Increased pressure within the abdominal cavity leads to a cascade of events that affect each organ system and tissue bed of the body. As the IAP increases, the earliest manifestations occur in the respiratory system. The diaphragm is pushed cephalad, embarrassing ventilation that affects oxygenation. At the same time, there is increased pressure over the inferior vena cava, resulting in diminished venous return to the heart, negatively impacting the cardiac output. Reduction in cardiac output affects systemic perfusion and causes tissue ischemia with generalized organ system dysfunction. The increased vena caval pressure is also transmitted via the renal veins directly affecting renal function [5]. Besides the generalized effects on every organ system as a result of reduced perfusion, there is evidence that ACS itself acts as a proinflammatory stimulus [6]. Thus, in any surgery involving the abdomen, IAH and ACS should be avoided and monitored for and, if occurring, should be rapidly diagnosed and treated to avoid poor outcomes or death.

Many of the complex abdominal wall defects that need repair were in the past probably created by attempts at either preventing the development of ACS (by not closing the musculoaponeurotic layer of the abdomen) or treating ACS after its development (by opening an intact or recently closed musculoaponeurotic layer of the abdomen and then leaving it open). Also, many, if not most, of the complex abdominal wall defects are considered complex because of the large size of the defect in the musculoaponeurotic envelope. This large size allows for a large proportion of the abdominal contents to reside outside the confines of the musculoaponeurotic layer of the abdomen. Over time, the volume available within the abdominal cavity is insufficient to accommodate all of the contents that have been residing outside. Forcing these contents back into the abdomen and thus raising the IAP to pathological levels and causing ACS will have disastrous consequences for the patient and will threaten the integrity of the repair. Hence, prior to repairing any complex abdominal wall defect, careful consideration needs to be given to avoiding this devastating complication.

Preoperative Considerations for Prevention of IAH/ACS

Patient Selection

As in all surgery, the first and foremost consideration is the general condition of the patient and whether the overall health is such that the patient can tolerate the stress of

A.K. Malhotra, MBBS (MD), MS, FRCS, DNB, FACS
Division of Trauma, Critical Care, and Emergency Surgery,
Department of Surgery, Medical College of Virginia,
Virginia Commonwealth University,
1200 E. Broad Street, West Hospital,
15th Floor, East Richmond, VA 23112, USA
e-mail: akmalhot@mcvh-vcu.edu

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_21,

anesthesia and major surgery. If a determination is made that the overall health is sufficiently good to tolerate anesthesia and major surgery, then for patients undergoing abdominal wall reconstruction for complex abdominal wall defects the next consideration would concern the possibility of developing IAH/ACS.

Morbid obesity is associated with a chronic form of IAH [7]. In such patients, even minor elevations of IAP will rapidly lead to progression of the IAH to ACS. Preoperative weight loss ameliorates the chronic IAH and thus reduces the risk of perioperative IAH/ACS. In addition, preoperative weight loss prior to complex abdominal wall reconstruction will improve the chances of a successful repair. As noted previously, the earliest manifestations of IAH/ACS concern the respiratory and cardiovascular systems; the reserve available in those two organ systems determines the ability of the individual patient to tolerate IAH. In an otherwise-healthy individual with relatively normal cardiorespiratory reserve, a 10 % drop in venous return or ventilation is tolerated without significant ill effects. However, in patients with preexisting cardiac/respiratory disease, even a reduction of less than 10 % may not be tolerated. Without performing detailed functional tests, it is not possible to quantify the state of the organ system or the reserve. However, in most patients a judgment can be made regarding whether after repair of the defect the patient will have adequate ventilation. A detailed clinical examination with manual reduction of any hernia and manually "closing" the defect are crude clinical tests that can give a fair idea about the cardiorespiratory tolerance after the final repair. Also, a tight abdominal binder can be temporarily placed to reduce the hernia, and the patient can be asked to walk around, with a clinician observing for any shortness of breath. Based on these simple tests, if it is determined the patient has adequate cardiorespiratory reserve, surgery can be undertaken. On the other hand, if either the tests cannot be performed or, after the clinical tests, it is felt that the patient has only borderline reserve, testing that is more objective with volumetric pulmonary function testing or stress tests should be considered. Each patient is truly unique and should be considered as such. It might be helpful to communicate with the patient's medical physicians to obtain as much information as possible before making a final decision about the patient's ability to undergo repair without development of IAH/ACS.

Size of Hernia: "Loss of Domain"

For patients in whom a long-standing large hernia has allowed the abdominal cavity proper to become so small that the herniated contents have lost their intra-abdominal domain, there is a high chance that the patient will develop IAH/ACS after reduction of contents and repair of defect. Even if the patient is able to tolerate the IAH and not develop ACS, the integrity of the repair will be threatened unless proper planning is performed. Here, also, simple clinical tests outlined previously allow for a determination to be made about the loss of domain. If there is doubt, computed tomographic (CT) measurements have been suggested that may aid in the determination [8]. However, CT can only perform static measurements of volume. The same available volume may suffice in a patient with laxity of muscle that allows for stretching, but that same volume may not be sufficient in another patient with an abdominal wall that is scarred and thus does not allow stretching. Again, a fair amount of judgment is necessary to adequately determine whether there is loss of domain. If there indeed is loss of domain, preoperative tissue expansion techniques (e.g., pneumoperitoneum) might be required to increase the size of the abdominal cavity. In less-severe cases, the choice of procedure (e.g., component separation) may need to be tailored to achieve a larger cavity and a more secure repair. These techniques are detailed in other chapters.

Size of Defect

Even if the size of the hernia is not large and reduction of the contents is tolerated well by the patient, it is possible that the defect in the musculoaponeurotic layer is so large that closure will lead to reduction in the volume of the abdominal cavity and IAH/ACS. After reduction of the hernia in the clinic, the edges of the defect should be brought together manually and the patient observed for signs of respiratory embarrassment. If there is no respiratory embarrassment, it is safe to presume that, after repair, ACS will not develop. On the other hand, if there is respiratory embarrassment, plans should be made accordingly for either preoperative or intraoperative expansion of the abdominal cavity.

Intraoperative Considerations

Based on the preoperative evaluations, a determination should be made prior to surgery whether the patient can tolerate any reduction in cardiac or respiratory function. As mentioned, in an otherwise-healthy adult a 10 % reduction in ventilatory capacity or venous return is usually well tolerated. When the patient is under anesthesia and being ventilated, the peak pressure (for patients on volume-controlled mode) and the tidal volume (for patients on pressure-controlled mode) serve as excellent measures of the effect of reduction and repair of the abdominal wall. A note should be made of these parameters after induction of anesthesia just prior to surgery. At the time of closure of the abdominal wall, these parameters should be monitored closely. An increase in peak pressures (for volume-controlled mode) or a decrease in tidal volume (for pressure-controlled mode) of greater

than 10 % should prompt a reevaluation of the safety of the closure. In patients with cardiorespiratory reserve that is more limited, any change should prompt a reevaluation of the closure. Close collaboration between the operating surgeon and the anesthesiologist is essential to reduce the chances of development of IAH/ACS. If unexpected changes in the parameters signal that IAH/ACS may occur, the technique of closure may need to be modified to avoid the complication. The specifics of how the abdominal cavity can be enlarged are presented in other chapters.

Postoperative Considerations

Any patient who has undergone complex repair of the abdominal wall is at increased risk of developing IAH/ACS. The condition can develop despite careful preoperative preparation and intraoperative monitoring. This happens because, in most patients undergoing complex reconstructions, extensive dissection is usually necessary both within the abdominal cavity and in the abdominal wall. Immediately after surgery, as the first phase of healing (inflammatory phase) is initiated in both these areas, the capillaries become "leaky," and "third spacing" (interstitial edema) occurs. This inflammatory swelling leads to an increase in the volume of the intra-abdominal contents, increasing IAP, which can proceed to IAH and ACS. The inflammatory phase of healing lasts 48–72 h. Following this, if healing is continuing normally, the capillaries will regain their selective function, and the interstitial edema will be resorbed and the excess fluid removed from the body by the kidneys. The end of the inflammatory phase is heralded by an increase in urine output. The conceptual understanding of this pathophysiology is important in managing the postoperative patient because, if the initial inflammatory phase can be managed without the development of organ system dysfunction, there can be a reasonable expectation of good long-term outcome. If, on the other hand, this early phase cannot be managed, it is likely that the patient will need repeat surgery with either takedown of the reconstruction or a modification of the technique so that there is more balance between the available space within the abdominal cavity and the combined volume of the contents.

Postoperative Care/Monitoring

Certain measures are applicable to all patients undergoing complex abdominal wall reconstruction. First, patients should have an indwelling urinary drainage catheter and a gastric tube on continuous low-level suction to keep these organs completely decompressed. Second, as mentioned, in the early postoperative period there is development of interstitial edema that can increase the IAP. To minimize the degree of interstitial edema, careful consideration should be given to keeping the patient euvolemic as opposed to hypervolemic. In cases of doubt, stroke volume variation or pulse pressure variation measurement offers an excellent tool for ensuring euvolemia and avoiding hypervolemia [9]. In patients with evidence of hypervolemia contributing to increased IAP, judicious use of diuretics, hemodialysis, or hemofiltration may prevent further rises in IAP. Last, even in the later stages of healing as the interstitial edema is being resorbed, promotility agents such as metoclopramide, erythromycin, neostigmine, and so on can reduce bowel distension and further rises in IAP. The process of IAH/ACS starts with an increase in IAP; hence, monitoring of this pressure is the best method for early detection and treatment of IAH/ACS. The accepted method of monitoring IAP is by bladder pressure measurements [5]. The technique is simple and noninvasive because virtually all patients who undergo complex abdominal wall reconstructions have an indwelling bladder catheter. The setup consists of a three-way stopcock connected to the following:

1. The aspiration port of the urine collection bag tube via pressure tubing and an 18-gauge needle,
2. A 50-mL syringe with sterile saline, and
3. Pressure transducer tubing.

The actual technique consists of emptying the bladder, clamping the tube of the collection bag distal to the aspiration port, and instilling 25 mL of sterile saline into the bladder. After instillation of the saline, the clamp is briefly loosened to empty the tubing of air toward the patient's side and reapplied without losing the saline. After emptying the air, the pressure within the bladder is measured and recorded. The level of the pubic symphysis is considered 0 mmHg [10]. Studies have shown excellent correlation between the true IAP and the bladder pressure measured by this technique. Similar to all techniques, however, the accuracy of the measurement depends on the meticulousness of the technique. The greatest source of error comes from incomplete emptying of the air. Air in the system anywhere from the transducer through the three-way connection into the pressure tubing, urine collection bag tubing, and the bladder catheter can dampen the pressure and give an erroneously low reading. Also, in patients with small bladders or those having bladder spasms, the pressure recording might be falsely high. If these sources of error are kept in mind and care is taken to avoid them, bladder pressure measurement is an excellent technique for monitoring patients for ACS and is by far the most common one utilized for this purpose. Even though bladder pressure monitoring is key for the early detection of IAH/ACS, organ system function monitoring is almost as important. Although the definition of ACS is an IAP greater than 20 mmHg with the development of organ system dysfunction, in reality organ system dysfunction can result from much lower IAPs as well [11]. The two most sensitive organ systems are the respiratory and renal systems.

If the patient is ventilated, changes in the peak pressure (volume-controlled ventilation) or tidal volume (pressure-controlled ventilation) will be among the earliest manifestations of IAH/ACS. Decrease in urine output in a euvolemic patient is another early manifestation of the process.

Therapy for Postoperative IAH/ACS

Therapy for postoperative IAH/ACS will depend on the severity of the syndrome and the rapidity with which IAH is increasing. In the milder form characterized by mild increases in the bladder pressure measurements or changes in ventilatory parameters, medical therapy may suffice without the need for surgical decompression. On the other hand, if the IAH is rapidly increasing and there is impending or overt ACS or there has been failure of medical management, surgical decompression will likely be the only therapy that will be effective.

Medical/Minimally Invasive Therapy

Nonoperative therapy consists of one or more of the following:

1. Neuromuscular blockade,
2. Needle/tube drainage of intra-abdominal fluid, and
3. Continuous external negative-pressure therapy by special custom-made devices.

As stated previously, a postoperative patient recuperating from complex abdominal wall reconstruction is most prone to IAH/ACS in the first 48–72 h after surgery because of interstitial edema. In this early postoperative phase, mild elevations of IAP with impending but no overt organ system dysfunction, short-term neuromuscular blockade may allow the patient to weather the inflammatory phase. The blockade is weaned once the patient is past the inflammatory phase, with resorption of the interstitial edema and overall decrease in the volume of the intra-abdominal contents [12, 13]. A small proportion of patients develops ACS not because of swelling of the viscera but rather because of accumulation of a large volume of fluid or blood within the abdominal cavity. Such patients can be treated by placing a needle or small catheter within the peritoneal cavity. In a patient who has recently undergone complex abdominal wall reconstruction, placement of a needle or catheter must be performed with extreme caution lest it injure the viscera or compromise the repair. Case reports of successful management are present in the burn literature [14]. Continuous external negative-pressure therapy is performed using custom-made devices that surround the abdomen and create a negative pressure outside the abdominal wall. Such devices have been used successfully in morbidly obese patients with chronic ACS [15, 16]. Their application in patients with acute ACS has not been reported, but in animal studies of acute ACS, they have shown potential [17]. Irrespective of which form of nonoperative therapy is utilized, ongoing monitoring is critical because if the therapy fails, surgical decompression will have to be performed to avoid a disastrous outcome.

Surgical Decompression

In patients with overt ACS or those who have failed attempts at medical management, surgical decompression will be necessary to treat the ACS. The exact technique of decompression will depend on the type of reconstruction that has been performed. In all cases, the increased pressure within the abdominal cavity has to be relieved for success. Once decompression has been achieved, an important decision will have to be made whether the patient can have a revised reconstruction at the same time. The answer will depend on the reconstructive technique utilized and if some modification can allow for increasing the size of the abdominal cavity. In any case, it is important to understand that a patient who has had ACS is likely to be in a compromised physiologic state and may not tolerate prolonged and complex surgery. It maybe wise to leave the abdomen open for 1–2 days, optimize the physiologic state, and return for a definitive repair. Even after surgical decompression, ongoing monitoring is critically important because, despite having an open musculoaponeurotic layer, the patient can still develop recurrent ACS—tertiary ACS [2].

Summary

IAH/ACS is a dangerous condition that occurs when the space available within the abdominal cavity is less than the combined total volume of the contents, leading to increased IAP, which in turn causes systemic organ system dysfunction. Any patient undergoing complex abdominal wall reconstruction is prone to develop IAH/ACS. Careful preoperative evaluation and intraoperative monitoring can provide information that the patient is likely to develop IAH/ACS. Preoperative preparation and modification of intraoperative technique can prevent the condition from occurring. Postoperative monitoring is critical for early detection and rapid therapy. Mild cases may be treated with nonoperative therapy, but for more severe cases, operative decompression with or without subsequent reconstruction has to be performed to prevent poor outcomes.

References

1. Joshi GP. Complications of laparoscopy. Anesthesiol Clin North America. 2001;19:89–105.
2. Muckart DJJ, Ivatury RR, Leppaniemi A, Smith S. Definitions. In: Ivatury RR, Cheatham ML, Malbrain MLNG, Sugrue M, editors. Abdominal compartment syndrome. Georgetown: Landes Bioscience, Eurekah.com; 2006.

3. Emerson H. Intra-abdominal pressures. Arch Intern Med. 1911;7:754–84.
4. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal reexploration. Ann Surg. 1984;199:28–30.
5. Malhotra AK, Ivatury RR. Compartment syndrome of the abdominal cavity. In: Irvin RS, Rippe JM, editors. Intensive care medicine. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2008. p. 1795–803.
6. Oda J, Ivatury RR, Blocher CR, et al. Amplified cytokine response and lung injury by sequential hemorrhagic shock and abdominal compartment syndrome in a laboratory model of ischemia-reperfusion. J Trauma. 2002;52:625–32.
7. Lambert DM, Marceau S, Forse RA. Intra-abdominal pressure in the morbidly obese. Obes Surg. 2005;15:1225–32.
8. Tanaka EY, Yoo JH, Rodriguez A, et al. A computerized tomography scan method for calculating the hernia sac and abdominal cavity volume in complex large incisional hernia with loss of domain. Hernia. 2010;14:63–9.
9. Michard F. Stroke volume variation; from applied physiology to improved outcomes. Crit Care Med. 2011;39:402–3.
10. Iberti TJ, Lieber CE, Benjamin E. Determination of intra-abdominal pressure using a transurethral bladder catheter: clinical validation of the technique. Anesthesiology. 1989;70:47–50.
11. Simon RJ, Friedlander MH, Ivatury RR, et al. Hemorrhage lowers the threshold for intra-abdominal hypertension-induced pulmonary dysfunction. J Trauma. 1997;42:398–403.
12. Macalina JU, Goldman RK, Mayberry JC. Medical management of abdominal compartment syndrome: case report and a caution. Asian J Surg. 2002;25:244–6.
13. DE Waele JJ, Benoit D, Hoste E. A role for muscle relaxation in patients with abdominal compartment syndrome? Intensive Care Med. 2003;29:332.
14. Latenser BA, Kova-Vern A, Komball D, et al. A pilot study comparing percutaneous decompression with decompressive laparotomy for acute abdominal compartment syndrome in thermal injury. J Burn Care Rehabil. 2002;23:190–5.
15. Saggi BH, Bloomfield GL, Sugerman HJ, et al. Treatment of intracranial hypertension using non-surgical abdominal decompression. J Trauma. 1999;46:646–51.
16. Sugerman HJ, Felton III WL, Sismanins A, et al. Continuous negative abdominal pressure device to treat pseudotumor cerebri. Int J Obes Relat Metab Disord. 2001;25:486–90.
17. Adams J, Osiovich H, Goldberg R, et al. Hemodynamic effects of continuous negative extrathoracic pressure and continuous positive airway pressure in piglets with normal lungs. Biol Neonate. 1992; 62:69–75.

Short-Bowel Syndrome: A Clinical Update

22

Stanley J. Dudrick, Jose M. Pimiento, and Rifat Latifi

Introduction

During the second half of the twentieth century, the basic laboratory development and subsequent successful clinical application of the techniques of total parenteral nutrition (TPN) had a transformative effect on the modern practice of medicine, surgery, pediatrics, and many of their subspecialties. Arguably, none of the benefits of this technique has been more fundamental and lifesaving than the resultant developments and advances in the metabolic management, nutritional support, innovative operative procedures, and pathophysiologic understanding of patients with the short-bowel syndrome (SBS) following massive intestinal resection. Furthermore, primarily because of the remarkable salvage of most of these patients with this critically severe life-threatening situation, it has eventually been recognized that an even broader spectrum of disorders of alimentary tract functions could be identified in addition to the dramatic endgame of SBS; it has been recognized that the patients with these various intestinal dysfunctions deserve our special basic and clinical attention, investigations, and attempts to prevent, ameliorate, or cure them. As a result, the concept of intestinal failure inevitably and logically arose and continues to evolve as knowledge and experience regarding these often-complex alimentary tract problems and their management are generated or acquired [1].

Intestinal failure has had a multitude of definitions, which will likely undergo additional revisions as knowledge of this deceptively simple but tremendously complex and adaptable organ system and the variations in the types, extents, and degrees of failures of its multiple components accumulates with further study. Simply stated, intestinal failure is a condition characterized by deficient, inadequate, ineffective, or absent performance of the appropriate and expected intestinal functions essential for the efficacious and optimal absorption of the fluids and nutrients required to maintain the normal physiologic activities of the body cell mass. However, intestinal failure encompasses a broad spectrum of variety and severity of signs, symptoms, presentations, and responses to therapeutic interventions; its precise definition is difficult and virtually impossible to standardize to "one-size-fits-all" situations. Moreover, its clinical description usually has more practical relevance and usefulness for specific optimal management than does its broad definition. In this regard, intestinal failure is analogous, for example, to cardiac failure, pulmonary failure, renal failure, circulatory failure (shock), and other organ/system failures in that it can present, advance, respond, and adapt in myriad ways to challenge both the patient and the caregivers attempting to ameliorate, manage, and support the patient throughout the various stages of intestinal failure. Attempts to define intestinal failure more precisely by a single, comprehensive, and uniformly accurate statement is, in reality, a futile academic endeavor of limited utility to the practitioner. A summative description of the clinical picture and the relevant laboratory data in each individual patient will ordinarily be of the most value in formulating a management plan specifically best suited for each case. These complex problems are not routine or common, and their management and resolution require persistent, conscientious, dedicated, intensive attention to detail, together with skill, knowledge, experience, judgment, wisdom, and resilience if optimal outcomes are to be achieved [1].

S.J. Dudrick, MD, FACS (✉)
Department of Surgery, Yale University School of Medicine,
40 Beecher Street, Naugatuck, CT 06770, USA
e-mail: sjdudrickmd@comcast.net

J.M. Pimiento, MD
Department of Surgery, Michigan State College of Human Medicine Lacks Cancer Center/Saint Mary's Health Care,
250 Cherry Street SE, Grand Rapids, MI 49503, USA
e-mail: pimiento@trinity-health.org

R. Latifi, MD, FACS
Department of Surgery, University of Arizona,
1501 N. Campbell Ave., 245063, Tucson, AZ 85724, USA

Department of Surgery, Weill-Cornell University, Doha, Qatar

Department of Surgery, Hamad Medical Corporation, Doha, Qatar
e-mail: latifi@surgery.arizona.edu

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_22,

Short-bowel syndrome is a form of intestinal failure usually consisting of an inadequate length of intestine that results following massive bowel resection. SBS is a clinical entity characterized primarily by intractable diarrhea, steatorrhea, dehydration, malnutrition, weight loss, and malabsorption of fats, minerals, and other macronutrients and micronutrients and not a situation merely defined anatomically by a specific length of remaining functioning small intestine. Subsequent adverse consequences of SBS include hypovolemia, hypoalbuminemia, hypokalemia, hypocalcemia, hypomagnesemia, hypozincemia, hypocupricemia, essential fatty acid and vitamin deficiencies, anemias, hyperoxaluria, and metabolic acidosis. The formation of kidney stones or gallstones can also often accompany SBS. The actual clinical presentation and progression of the patient with SBS depends on several factors, including the following:

1. The extent of the bowel resection;
2. The site(s) of the resection;
3. The presence or absence of the ileocecal valve;
4. The residual function of the remaining small bowel, stomach, pancreas, biliary tree, and colon;
5. The capacity or potential of the intestinal remnant for adaptation;
6. The primary nature and status of the disease, disorder, or trauma that precipitated the loss of the small bowel;
7. The type, extent, location, and activity of any residual disease in the intestinal remnant; and
8. The general condition of the organ systems and body cell mass of the patient [2–8].

The minimum length of small bowel sufficient for adequate digestion and absorption is controversial. Standardization of the adaptive potential of the residual bowel is difficult because of the variable absorptive capacity of the remaining remnants, the wide variation in the length of the normal small intestine, and the difficulty in obtaining reproducible measurements of the length of the remaining bowel following massive resection. The nutritional and metabolic status, overall general health and function, and age of the patient are important collateral factors. Depending on the state of contraction or relaxation of the intestinal musculature, intraoperative estimates of the length of the normal, intact, small intestine in the adult vary from 260 to 800 cm (approximately 8–26 ft). On the other hand, the mean length of normal small intestine measured during life is 350 cm (11–12 ft), and postmortem it is 600 cm (20 ft) [6]. Because of this large variability, it is virtually impossible to determine the exact initial length of the remaining small bowel, and it is difficult to estimate the percentage of the total length of small bowel represented by the segment remaining following massive intestinal resection. Moreover, many surgeons often only measure the length of the resected small bowel, rather than also measuring the length of the remaining intestinal segment, which is the critically important functional and prognostic measurement. In addition, they then often fail to describe accurately the nature, condition, and extent of the remaining small bowel in the patient's medical record for future reference. Furthermore, because inflamed intestine generally shortens after operation, the absorptive functions following massive small bowel resection often do not correlate well with the original intraoperative estimated or measured length of the remaining intestine [6–8].

Because of the rather ample functional reserve capacity of the small bowel, short segmental resections of the small intestine usually do not result in significant problems with digestion and absorption [8–10]. Indeed, resection of as much as 40 % of the small intestine is usually well tolerated, provided that the duodenum, the distal half of the ileum, and the ileocecal valve are spared [11]. On the other hand, resection of 50 % or more of the small intestine usually results in significant malabsorption initially but can be tolerated eventually without extraordinary pharmacological or parenteral or enteral nutritional support. However, resection of 75 % or more of the small intestine usually leaves the patient with 70–100 cm (2–3 ft) of remaining intestine, resulting in a degree of SBS that can significantly impair the ability of the patient to maintain normal nutrition and metabolism. Such patients will likely require special nutritional management on a long-term or permanent basis, especially with the loss of the terminal ileum and the ileocecal valve, if normal body cell mass and function are to be preserved or restored [7].

The severity of symptoms and signs following massive small bowel resection is related both to the extent of the resection and the specific anatomic sites of the resected small bowel [12]. However, the minimal residual small intestinal absorptive surface required to sustain life without permanent parenteral nutritional support appears to vary somewhat with each patient [13, 14]. Development of effective TPN has revolutionized the treatment of SBS by allowing maintenance of adequate nutrition indefinitely or until the remaining bowel can adapt maximally to oral or enteral feeding, thus reducing the morbidity and mortality significantly [15–20]. Prolonged survival has now been achieved in a number of patients having only an intact duodenum and 15 cm (6 in.) of residual jejunum, with or without all or part of the colon [4, 10, 21]. If approximately 60 cm (2 ft) of jejunum or ileum remain functional in addition to the entire duodenum, survival has been the rule rather than the exception [21].

Preservation of the ileocecal valve is of paramount importance during massive small bowel resection and, by significantly increasing the duration of the intestinal transit time, allows a longer exposure time of the intestinal chyme to the residual absorptive surface of the mucosa. Salvage of the ileocecal valve, whenever possible, has the clearly beneficial effect of increasing the absorptive capacity of the remaining small bowel to approximately twice that anticipated for the same length of comparable small bowel

without an intact ileocecal valve. Primarily as a result of mucosal hyperplasia and villous hypertrophy, absorption by the residual intestinal segments of patients with SBS can increase as much as fourfold. Therefore, in a patient with an intact ileocecal valve, the total cumulative absorptive capability of the remaining bowel potentially can be increased maximally about eightfold. This amount of adaptive absorptive recovery function often approaches normal intestinal capacity [7, 21].

The most common clinical conditions that precipitate massive small bowel resections are those that compromise the vascular supply of the small intestine [22–24]. These include venous thrombosis and arterial occlusion as a consequence of primary vascular disease, heart failure with attendant mesenteric low-flow state, various coagulopathies, volvulus, malrotation of the gut, and internal or external herniation of the bowel with strangulation. SBS can also occur as a result of necrotizing enterocolitis or massive atresia of the small intestine in newborn infants, at times associated with gastroschisis or omphalocele. Inflammatory bowel disease involving large segments of the small bowel, or recurrent exacerbations of inflammatory bowel disease over a long period of time, can eventually result in SBS secondary to massive or multiple intestinal resections. Excision of retroperitoneal malignancies that involve the celiac or superior mesenteric vessels can mandate secondary resection of most or the entire small bowel to accomplish palliation or cure. Major abdominal blunt or sharp trauma involving transection, disruption, or avulsion of the mesenteric vasculature can also result in ischemic necrosis of large segments of the small bowel, resulting in SBS. Postirradiation or postoperative complications such as extensive severe radiation enteritis, multiple small bowel fistulas, multiple bowel obstruction procedures, and intestinal gangrene can also result in irreversible SBS.

Some of these conditions or situations are accompanied by, result in, or result from complex abdominal wall defects. For example, in neonates, gastroschisis is a congenital anomaly that not only is comprised of a defect in the closure of the abdominal wall but also is frequently associated with other developmental intestinal deformities, such as extensive or multiple small bowel atresias or mesenteric vascular abnormalities that result in the "apple peel" or "Christmas tree" mesentery anomalies. Omphaloceles, sometimes ruptured during the birthing process, are accompanied not only by an underdeveloped and contracted peritoneal cavity causing a "loss of domain" of the extra-abdominal small intestine, but also by atretic segments of bowel and an abdominal wall defect in the region of the umbilical cord. Surgical correction of these problems is obviously required, and the extent and nature of the procedure or procedures vary with the magnitude and complexity of each individual situation, ranging from simple closure of the abdominal wall defect, with or without resection of an accompanying atretic segment of bowel, to a compound or composite operative and nonoperative management plan of a multifaceted or variegated nature to restore both the integrity of the abdominal wall and the anatomical and functional continuity of the intestinal tract. The most difficult or complex of these conglomerate situations not only can pose formidable challenges to the neonatology and pediatric surgery teams but also can represent the highest level of personal and professional accomplishment when optimal outcomes result from their combined skills, efforts, and acumen. Obviously, nutritional and metabolic management and support must be intricately and masterfully interwoven judiciously with surgical operative talent, ingenuity, and timing; it must be continued, persistently and conscientiously, throughout the recovery and rehabilitative periods until optimal organ, system, and body cell mass functions are achieved or restored for the patient.

In adults, the recent era of abdominal compartment syndrome and the treatment or decompression of intraperitoneal hypertension by "open abdomen" measures or temporary intestinal coverage by various reconstructive operative techniques, using native tissues or various artificial or despeciated substitute products for abdominal closure, has been accompanied by a significant incidence of fistula formation, bowel obstructions, herniations, recurrent operations, and so on. At times, the prolonged treatment periods necessary to salvage and rehabilitate these patients, together with the multiple associated complications, not only have challenged surgeons technically to restore abdominal wall integrity but also have required their understanding of the physiologic and metabolic states of the patients that will enable them to restore and maintain intestinal continuity and function. This occurs while dealing with multiple enteroatmospheric fistulas ("the surgeon's nightmare"), multiple intestinal resections, functional or anatomical intestinal failure or SBS, combined with the ever-present need to maintain optimal nutritional status to promote immunocompetence; combat infection; heal anastomoses and wounds; support normal organ, system, and body cell mass functions; and preserve life itself [25, 26]. The problems for most such patients result from acute major traumatic injuries, in which portions of the abdominal wall might be lost, destroyed, or devitalized, in addition to injuries to other organ systems. However, these complex abdominal wall/SBS catastrophic situations can also arise following nontraumatic gastrointestinal (GI) tract perforations secondary to a variety of inflammatory or neoplastic disorders, mesenteric infarctions of the intestine, anastomotic leaks, various intraperitoneal abscesses, abdominal wound disruptions, and so on, often coupled with or compounded by hypoproteinemic malnutrition as a contributing, precipitating factor, as a comorbidity, or as a secondary complication of SBS or other intestinal failure [26].

Pathophysiology of Short-Bowel Syndrome

The intestinal absorption of water, electrolytes and other specific nutrients is dependent primarily on the extent and site of the small bowel resection. The intestinal phase of digestion occurs initially in the duodenum, where pancreatic enzymes and bile acids promote digestion of all nutrients and enhance fat absorption. It is highly uncommon for the duodenum to be resected together with extensive segments of the small bowel, primarily because of the differences in blood supply; however, total duodenectomy, when it occurs, leads to malabsorption of calcium, folic acid, and iron [2]. Proteins, carbohydrates, and fats are absorbed virtually completely in the 150 cm of the jejunum; therefore, only small quantities of these nutrients or their derivatives ordinarily reach the ileum [27].

The small intestine acquires and handles a total of about 8 L of fluid daily, including dietary ingestion and endogenous secretions. Normally, approximately 80 % of the intraluminal water transported is absorbed in the small bowel, leaving approximately 1.5 L of fluid to traverse the colon. The colon usually absorbs about 1–2 L of fluid, having maximal absorptive capacity of approximately 6 L of fluid per day [28]. Because the ileum and colon have a large capacity for absorbing excess fluid and electrolytes, proximal small bowel (jejunal) resections only rarely result in diarrhea. On the other hand, extensive or total resection of the ileum results in a greater potential for malabsorption and resultant diarrhea. Not only will such resections increase the volume of fluid reaching the colon, but also, depending on the length of ileum resected, bile salt diarrhea (cholorrhea) or steatorrhea may ensue, with subsequent losses of essential fatty acids and fat-soluble vitamins. If the ileocecal valve has been resected, transit time is likely to decrease, and bacterial colonization of the small bowel will eventually be more likely to occur, further aggravating cholorrhea and steatorrhea.

As the length of ileal or colonic resections increases, essential absorptive surface area is lost, resulting in proportionally increased dehydration, hypovolemia, and electrolyte derangements. If the colon remains in continuity with the remaining small bowel following massive intestinal resection, malabsorbed bile salts can be deconjugated by colonic bacteria, stimulating increased colonic fluid secretion and further compounding existing diarrhea. Following extensive ileal resection, the enterohepatic circulation is interrupted, and irreversible loss of bile salts results, with or without the colon in continuity. Although the excess fecal losses stimulate hepatic synthesis of bile salts, a higher incidence of cholelithiasis occurs in these patients. Because the transit time in the ileum is usually slower than in the jejunum, residual intestinal transit is slowed, and fecal output is diminished as the length of remaining ileum increases.

Following extensive small bowel resections, intestinal lactase activity might decrease, resulting in lactose intolerance [29]. The presence of unhydrolyzed lactose causes increased hyperosmolality in the intestinal lumen. Moreover, fermentation of lactose by colonic bacteria produces a large amount of lactic acid, which can further aggravate osmotic diarrhea [2]. The water-soluble vitamins (vitamin B complex and C) and minerals (Ca^{2+}, Fe^{3+}, Cu^{2+}) are absorbed in the proximal small intestine, whereas magnesium diffuses passively throughout the entire small bowel [2]. On the other hand, the ileum is the only absorption site for vitamin B_{12} and bile salts. Resection of the jejunum with preservation of the ileum produces no permanent impairments of protein, carbohydrate, and electrolyte absorption [30]. The ileum can compensate for most absorptive functions, but not for the secretion of jejunal enterohormones. Following jejunal resections, diminished secretions of cholecystokinin and secretin decrease gallbladder contraction and emptying and pancreatic exocrine secretions. In addition, after jejunal resection, gastric hypersecretion is greater than after ileal resection. This results from the loss of inhibitory hormones such as gastric inhibitory polypeptide (GIP) and vasoactive intestinal polypeptide (VIP), which are secreted in the jejunum, thus causing gastrin levels to rise, stimulating gastric hypersectretion [31]. Significant gastric hypersecretion can be documented within 24 h postoperatively, and the gastric and small bowel mucosa can be injured by the accentuated high gastric acid output, causing gastritis, ulceration, and bleeding. Subsequently, the high salt and acid load secreted by the stomach, together with the inactivation of digestive enzymes by the inordinately low intraluminal intestinal pH, serves to compound the other causes of diarrhea associated with SBS.

Ordinarily, the colon is a major site of water and electrolyte absorption, and as the ileal effluent increases, the colon may increase its absorptive capacity to three to five times normal [32]. Moreover, the colon has a moderate capacity to absorb other nutrients, and concomitant colon resections can adversely affect the symptomatic and nutritional courses of patients with massive small bowel resections. Malabsorbed carbohydrates that reach the colon are fermented there by indigenous bacteria to yield short-chain fatty acids, principally acetate, butyrate, and propionate [33, 34]. These short-chain fatty acids can be absorbed by the colon in quantities representing up to 500 cal per day and can enter the portal circulation to serve as a fuel source [35, 36]. Although retention of the colon is highly desirable during massive bowel resections, its presence can be associated with potential complications. In addition to cholorrheic diarrhea, a patient with a massive small bowel resection and an intact colon often develops hyperoxaluria and a tendency to form calcium oxalate renal stones. These

result from the increased absorption of dietary oxalate, which is normally rendered insoluble by binding with calcium in the intestinal lumen and therefore is ordinarily unabsorbable. However, in patients with SBS and steatorrhea, intestinal calcium ion is bound preferentially to the increased quantities of unabsorbed fatty acids, leading to decreased binding, and thus an increased colonic absorption of unbound oxalate [12].

Finally, preservation of the ileocecal valve is important in preventing abnormal metabolic sequelae because the ileocecal valve not only slows intestinal transit and passage of chyme into the colon but also to a large extent prevents bacterial reflux and passage from the colon into the small bowel. Nutrients that reach the colonic lumen, especially vitamin B_{12}, become substrates for bacterial metabolism rather than being absorbed into the circulation by the mucosa [2]. Furthermore, bacterial overgrowth in the small bowel in patients with SBS appears to increase the incidence of liver dysfunction [37].

Nutritional and Metabolic Management of Short-Bowel Syndrome

In the metabolic and nutritional management of patients with SBS, three different but overlapping therapeutic periods having rather distinctive characteristics can be designated arbitrarily (Table 22.1) [38]. During the first 2 months (immediate and early postoperative period), the clinical picture and course are dominated by problems related to fluid and electrolyte balance; adjustments of organ blood flow patterns, especially the portal venous flow; and other effects of the major operative insult and its accompanying specific and general complications. During the second period, from about 2 months up to 2 years postoperatively (bowel adaptation period), efforts are directed toward defining maximum oral feeding tolerances for various nutrient substrates, encouraging and maximizing intestinal and bowel adaptation, and determining and formulating the most effective patient-specific feeding regimens. Usually within 2 years, 90–95 % of the bowel adaptation potential has been accomplished, and only 5–10 % further improvement in absorption and bowel adaptation can be anticipated. The third period (long-term management period) constitutes the period after 2 years, when nutritional and metabolic stability have ordinarily occurred. By this time, the patient has either adapted maximally so that nutrition and metabolic homeostasis can be achieved entirely with oral feeding, or the patient is committed to receiving specialized supplemental or complete nutritional support for the remaining life span, either by ambulatory home TPN or specially prepared enteral or oral feedings [7].

Table 22.1 Synopsis of short-bowel syndrome management [7, 21, 38]

Immediate postoperative period (First 2 months)
Fluid and electrolyte replacement
Lactated Ringer's solution
Dextrose 5 % in water
Human serum albumin (low salt)
K^+, Ca^{++}, Mg^{++} supplementation
Strict intake and output
Daily body weight
Graduated metabolic monitoring
Antacid therapy (optional prn)
(30–60 mL via nasogastric [N-G] tube q 2 h;
clamp N-G tube 20 min)
Mylanta liquid
Camalox suspension
Amphogel suspension
Gelusil liquid
Antisecretory/antimotility therapy
Cimetidine, 300 mg IV q 6 h
Ranitidine, 150 mg IV q 12 h
Famotidine, 20 mg IV q 12 h
Pantoprazole, 40 mg IV daily
Codeine, 60 mg IM q 4 h
Loperamide, 4–16 mg po daily
Lomotil, 20 mg po q 6 h
Hyoscyamine sulfate, 0.125 mg sc q 4 h
Cholestyramine 4 g po q 8 h
Total parenteral nutrition
1 L on second postoperative day
Gradually increase dosage as tolerated
Supplemental fluids, electrolytes, and colloids as needed
Bowel adaptation period (First 2 years)
Progression of oral diet
Water, tea, broth
Simple salt solutions
Simple sugar solutions
Combined salt/sugar solutions
Dilute chemically defined diets
High carbohydrate, high protein
Modified fiber, low-fat diet
Near-normal, normal diet
Enteral supplementation
Coconut oil, 30 mL po tid
Safflower oil, 30 mL po tid
Multiple vitamins, 1 mL bid
Ferrous sulfate, 1 mL tid
Ca gluconate, 6–8 g/day
Na bicarbonate, 8–12 g/day
Parenteral supplementation
Electrolytes, trace elements
Divalent cations (Mg, Zn, Cu, Se)
Vitamin B_{12}, Vitamin K, folic acid
Albumin, packed red cells
Fat emulsion

(continued)

Table 22.1 (continued)

Antisecretory/antimotility (Refer to items for the first 2 months for additional agents)
Famotidine, 20 mg po q 12 h
Pro-Banthine, 15 mg po q 4–6 h
Dicyclomine, 20 mg po q 6 h
Omeprazole, 20 mg po q day
Deodorized tincture of opium, 10–30 gtts q 4 h
Codeine, 30–60 mg po q 4 h
Paregoric, 5–10 mL po q 4 h
Growth hormone/glutamine [54, 55]
Long-term management (After 2 years)
Apply previous principles
As indicated individually
Ambulatory home TPN (total parenteral nutrition)
Supplemental or total continuous, cyclic or intermittent
Surgical management
Treat operative complications
Drain abscesses
Resect fistulas
Lyse adhesions
Reduce obstructions
Restore bowel continuity
Probable cholecystectomy
Intestinal lengthening [70–75]
Intestinal transplantation [77–81]

Immediate Postoperative Period

During the immediate postoperative period, for up to 2 months, virtually all nutrients, including water, electrolytes, fats, proteins, carbohydrates, and all vitamins and trace elements, are absorbed from the GI tract poorly, unpredictably, or not at all [38]. Fluid losses via the GI tract are greatest during the first few days following massive small intestinal resection, and anal or stomal effluent frequently reaches volumes in excess of 5 L per 24 h. To minimize life-threatening dehydration, hypovolemia, hypotension, electrolyte imbalances, and other related potential problems, vigorous fluid and electrolyte replacement therapy must be instituted promptly and judiciously. Frequent measurements of vital signs, fluid intake and output, and central venous pressure, together with regular determinations of hematologic and biochemical indices, are mandatory in monitoring the patient during this period of rapid metabolic change and instability. All patients with SBS exhibit some abnormalities in their liver profiles, and the vast majority of them experience at least transient hyperbilirubinemia [38]. This has been advocated by some to be secondary to the translocation of microorganisms or their toxins through the ischemic or gangrenous intestinal mucosa into the portal vein and thence to the liver [39, 40]. Others attributed the hyperbilirubinemia to impaired blood flow to the liver through the portal vein by as much as 50 % as a result of greatly diminished mesenteric venous return secondary to the massive small bowel resection [41]. Still others attributed this phenomenon to a combination of both factors or other etiologies [42]. Broad-spectrum anaerobic and aerobic antibiotic therapy should be instituted empirically and maintained for several days to 1 week following massive intestinal resection.

Typical patient management efforts during this period are directed toward achievement of four primary goals: fluid and electrolyte replacement, antisecretory/antimotility therapy, antacid therapy, and TPN. During the first 24–48 h, replacement therapy usually consists of 5 % dextrose in lactated Ringer's solution administered intravenously concomitantly with appropriate amounts of potassium chloride or acetate, calcium chloride or gluconate, magnesium sulfate, and fat- and water-soluble vitamins. If there is no evidence of sepsis, low-salt human albumin (12.5–25 g) usually is added exogenously to the intravenous regimen every 8 h for the first 24–48 h postoperatively to maintain normal plasma albumin concentrations and normal plasma colloid oncotic pressure. It is our opinion and experience that maintenance of optimal intravascular colloid osmotic pressure with normal albumin and erythrocyte concentrations reduces intestinal mucosal edema and enhances fluid and nutrient absorption while reducing losses as diarrhea. In patients with severe diarrhea, zinc losses can increase to as much as 15 mg/day, and appropriate aggressive parenteral replacement is required [43].

Antiacid therapy can reduce the increased tendency for peptic ulceration, which commonly occurs following massive small bowel resection. Antacids are given through a nasogastric tube, if one is in place, every 2 h in doses of 30–60 mL, and the tube is then clamped for 20 min before reapplying suction. Alternatively, or concomitantly, liquid sucralfate can be given by mouth or via the nasogastric tube in a dose of 1 g every 6 h, clamping the tube for 20 min after each dose. To counteract the hypergastrinemia and associated gastric hypersecretion that follows massive small bowel resection in the majority of patients, an H_2 receptor blocker is infused intravenously [44]. The intravenous administration of 300–600 mg of cimetidine every 6 h can have a profound effect on reducing gastric acid and intestinal fluid production. Alternatively, 150 mg ranitidine can be given intravenously every 12 h, 20 mg famotidine can be given intravenously every 12 h, or an intravenous form of a proton pump inhibitor, pantoprazole, can be given daily in 40-mg doses. In selected patients with short bowel, somatostatin analog (octreotide) has reduced fecal losses when administered in a dosage of 50–150 μg IV or subcutaneously every 6 h [45, 46]. If diarrhea persists despite these measures, an opiate can be prescribed. Preferably, codeine is given intramuscularly in doses of 60 mg every 4 h. Improvement in fluid and electrolyte management can also be achieved in selected patients with stomal access to a distal

defunctionalized bowel loop by reinfusing the chyme from the proximal stoma into the distal bowel segment [47]. Later in the course of the postoperative period, when the patient is tolerating liquids by mouth, antimotility therapy can be achieved by giving 4–16 mg loperamide orally in divided doses daily, 4 g cholestyramine every 4–8 h, or 20 mg diphenoxylate every 6 h. Codeine (30–60 mg), 5–10 mL paregoric, or 10–30 drops deodorized tincture of opium (DTO) every 4 h orally can be used to impede bowel motility. The major advantages of DTO are that it is readily absorbed by the upper alimentary tract, and the patient's bowel hypermotility and diarrhea can be titrated to tolerable therapeutic levels by adjusting the dosage up or down a few drops at a time to optimize dose effectiveness and to minimize undesirable side effects [7, 21].

By the second or third postoperative day, the patient's cardiovascular and pulmonary status have usually stabilized sufficiently to allow TPN to be initiated [7, 21]. The average adult patient can usually tolerate 2 L of TPN solution daily administered by central vein. By titrating levels of plasma glucose and glycosuria, the daily nutrient intake can be increased gradually to desired levels or to patient tolerance. In a patient with diabetes mellitus or who is glucose intolerant, crystalline regular human insulin is added to the TPN solution in dosages up to 60 units per 1,000 cal as needed. Following an operation of the magnitude of massive small bowel resection, patients may require up to 3,000 mL of TPN solution (about 3,000 cal) per day initially for a few days to maintain nutritional and metabolic homeostasis. Supplemental fluid and electrolyte infusions might be necessary for several days or weeks to replace excessive losses as diarrhea. The patient is offered a clear liquid diet as soon as the postoperative condition is stabilized, and fecal output is controlled with antidiarrheal medications. It may take several days to several weeks before the patient is able to discontinue TPN support in favor of oral or enteral feedings. It is essential to provide adequate nutritional supplementation with TPN for as long as the patient requires such support to maintain optimal nutritional status. The TPN ration is reduced gradually in an equivalent reciprocal manner as oral intakes and intestinal absorption of required nutrients are increased. The patient's diet is advanced slowly and gradually to a low-lactose, low-fat, high-protein, high-carbohydrate composition according to individual tolerances to the nutrient substrates and to the water volume and osmolality of the dietary regimen [7, 21, 48].

Bowel Adaptation Period

During the period of bowel adaptation from 2 months to 2 years postoperatively, the patient is allowed to consume increasing amounts of water, simple salt solutions, and simple carbohydrates [7, 21]. Various fruit and other flavorings can be added to 5 % dextrose in lactated Ringer's solution as a relatively inexpensive and practical oral nutrient and fluid replacement solution. Gradually, dilute solutions of chemically defined diets containing simple amino acids and short-chain peptides are given as tolerated in increasing volumes and concentrations as bowel adaptation progresses toward a normal or near-normal diet consisting of high carbohydrate, high protein, and low fat and comprised of food most preferred by the patient as the next stage of nutritional rehabilitation. Alternatively, the major nutrients can be provided as required in commercially prepared modular feedings tailored to the needs of individual patients until ordinary food is well tolerated. All essential vitamins, trace elements, essential fatty acids, and minerals are initially supplied in the patient's balanced intravenous nutrient ration. Subsequently, the oral diet may be supplemented most economically by short- and medium-chain triglycerides in the form of coconut oil, 30 mL two or three times daily; essential fatty acids as safflower oil, 30 mL two or three times daily; multiple fat- and water-soluble vitamins in pediatric liquid form, 1 mL twice daily; vitamin B_{12}, 1 mg intramuscularly every 4 weeks; folic acid, 15 mg intramuscularly weekly; and vitamin K, 10 mg intramuscularly weekly. Some patients may require supplemental iron, which can be administered initially by deep intramuscular injection as iron dextran according to the recommended patient-specific dosage schedule or as an intravenous infusion after testing the patient for sensitivity [7, 21]. Alternatively, an oral liquid iron preparation can be given one to three times daily, while closely monitoring iron indices and liver function tests.

A strong tendency for patients with SBS to develop metabolic acidosis usually requires the use of sodium bicarbonate tablets, powder, wafers, or liquid in doses of 8–12 g/day for as long as 18–24 months, but usually not for fewer than 6 months [7, 21]. It is often helpful to alternate the form of sodium bicarbonate prescribed to encourage maximal patient compliance. Because of the difficulty in absorbing adequate dietary calcium, supplemental calcium gluconate should also be prescribed as tablets, wafers, powder, or liquid in doses of 6–8 g/day. As bowel adaptation progresses, the doses of sodium bicarbonate and calcium gluconate can be decreased concomitantly or discontinued as restorative goals are attained. However, such oral supplements might be necessary for as long as 2 years or more in some patients to maintain homeostasis. Occasionally, on the other hand, a patient may become severely acidotic (pH 7.0–7.2) as a result of obviously copious diarrhea, but sometimes more subtly, and may require urgent or emergency intravenous infusion of sodium bicarbonate. Usually, the patient responds promptly to the therapy within a few hours and without untoward sequelae. Rarely, calcium gluconate must be given intravenously as a supplement to correct recalcitrant

hypocalcemia (<8.0 mg/dL). It is important to maintain normal serum albumin levels in patients with hypocalcemia. Dietary advancement and nutrient supplementation must obviously be individualized for each patient, and an effective nutrition support team can be helpful in maintaining and monitoring these complex patients. When solid foods are given, they should be dry and followed 1 h later with isotonic fluids, rather than giving solids and liquids together at the same time. This practice is followed to minimize diarrhea and to improve nutrient absorption. Lactose intolerance should be anticipated and treated as required with a low-lactose diet or 125–250 mg lactase by mouth. Clearly, milk products should be avoided as much as possible if intolerance persists [7, 21].

As progress occurs during the bowel adaptation period of management of the SBS, fat can be increased in the diet as tolerated, and supplementation with short- and medium-chain triglycerides and essential fatty acids may no longer be necessary [7, 21]. Serum-free fatty acid levels and triene-to-tetraene ratios are monitored periodically to determine the efficacy of treatment and the need for supplementation. Contrary to early reports, high-fat diets apparently are comparable to high-carbohydrate diets when evaluated in reference to calories absorbed, blood chemistries, stool or stomal output, urine output, and electrolyte excretions [47]. However, it has been suggested that enteral intake of fat should approach 50–100 % greater than expected goals to compensate for malabsorbed nutrients [43]. Patients who cannot tolerate or utilize a normal oral diet should be given a trial of continuous administration of enteral formula. Low-residue, polymeric, chemically defined, or elemental diets offer the putative advantage of high absorbability in the patient with a short bowel. However, some investigators have recently shown no differences in caloric absorption, stomal output, or electrolyte loss among elemental, polymeric, and normal diets in patients with SBS [7, 21, 49–51].

Depending on the results of periodic hematologic and biochemical studies, adjustments are made in the patient's intake of sodium, potassium, chloride, and calcium [52]. In addition, intermittent supplemental infusions of solutions containing magnesium, zinc, copper, and selenium might be required. As malabsorption and diarrhea become less troublesome, the vitamin and trace element requirements may be satisfied by multivitamin capsules, tablets, or chewable tablets containing therapeutic doses of vitamins or minerals, one dose twice daily. Relatively large amounts of magnesium, zinc, vitamin C, and vitamin B complex can be administered in the form of several commercially available therapeutic vitamin and mineral preparations [7, 21, 38]. It is especially important to avoid thiamine deficiency (Wernicke's syndrome).

In some patients, it might be necessary periodically to correct individual nutrient substrate deficiencies intramuscularly or intravenously for prolonged periods of time. Intermittent infusions of human serum albumin and packed erythrocytes might be required to treat recalcitrant hypoalbuminemia and anemia and to restore the plasma albumin level and the hematocrit to normal. Cholestyramine can be administered to counteract bile salt diarrhea if indicated, but intraluminal cholestyramine itself can cause or aggravate diarrhea. Fatty acid, electrolyte, trace element, vitamin, and acid–base imbalances must be promptly corrected enterally or parenterally as required when manifested clinically or by laboratory assessment. Serum vitamin B_{12} levels must be monitored and vitamin B_{12} deficiency corrected immediately. Hyperoxaluria should be assessed regularly, and if documented, foods containing high levels of oxalate, such as chocolate, spinach, celery, carrots, tea, and colas, should be restricted [7, 21].

In patients with severe forms of SBS, in whom little or no small intestine is present distal to the duodenum or in whom the remaining small intestine has residual disease, hypermotility and recalcitrant or intractable diarrhea may require continuous long-term antimotility/antisecretory treatment with oral or parenteral forms and dosages of the previously described pharmaceutical agents. Additional oral medications that have been helpful in selected patients include omeprazole, 20 mg daily; propantheline bromide, 15 mg every 4–6 h; dicyclomine hydrochloride, 20–40 mg every 6 h; and hyoscyamine sulfate, 0.125–0.250 mg every 4–6 h as needed [7, 21].

Long-Term Management Period

Long-term management of SBS can be accomplished successfully in most patients by conscientious attention to the principles and practices outlined previously. However, in a few patients who have undergone massive small bowel resection, TPN or supplemental parenteral nutrition must be provided in a continuous or cyclic manner for extended periods of time and sometimes for life. The metabolic management and nutritional therapy of patients with SBS must be tailored specifically to each patient, and the clinical responses following massive intestinal resections depend on many and varied factors. Patients with SBS pass through several stages of nutritional and metabolic support during their recovery, convalescence, and rehabilitation. Most of them can ultimately be maintained on a normal or near-normal diet. However, depending on the adaptability of their remaining bowel, they may have to settle for receiving their nutritional requirements by one or more of the following options:

1. A modified oral diet;
2. An oral diet supplemented with intravenous fluid or electrolytes;

3. An oral diet supplemented with enteral feedings;
4. An enteral diet entirely;
5. An oral diet supplemented with enteral feedings and parenteral nutrition;
6. An enteral diet supplemented with oral feedings;
7. An oral diet supplemented with parenteral nutrition;
8. An enteral diet supplemented with parenteral nutrition;
9. An enteral diet supplemented with parenteral nutrition and oral feedings;
10. A primarily parenteral nutrition regimen supplemented with variable oral or enteral diets; and
11. Total parenteral nutrition virtually entirely, but with trophic oral feedings as tolerated to stimulate intestinal adaptation and immunocompetence.

Almost every patient with SBS eventually develops gallstones, most usually requiring cholecystectomy within 2 years following massive intestinal resection if the gallbladder had not been previously removed. Indeed, the high propensity of patients who have undergone massive intestinal resection to develop stones in the gallbladder has stimulated some physicians to advocate cholecystectomy prophylactically at the time of bowel resection [53]. However, gallstone formation in the common bile duct and elsewhere in the biliary tree is also increased in these patients even after cholecystectomy. Therefore, long-term surveillance with periodic abdominal ultrasonography might be useful in identifying and monitoring echogenic changes in the gallbladder and biliary tree in patients with a short bowel [7, 21].

Finally, some otherwise-stable patients occasionally develop recalcitrant diarrhea secondary to colonization or bacterial overgrowth of the residual small bowel segment, requiring periodic stool culture and bacterial antigen studies followed by parenteral treatment with appropriate antibiotics [7, 21].

Growth Hormone, Glutamine, and Modified Diet

An extensive study has been completed to determine if growth hormone or nutrients, given alone or together, could enhance absorption from the small bowel after massive intestinal resection, especially in patients who continue to experience malabsorption and require long-term parenteral nutrition [54]. The effects of a high-carbohydrate, low-fat diet, the amino acid glutamine, and growth hormone, administered alone or in combination, were studied in 47 adult patients with SBS who were dependent on TPN to some extent for an average of 6 years. The average age of the patient was 46 years; and the average residual small bowel length was 50 cm in those with all or a portion of the colon remaining, and it was 102 cm in those with no colon remaining. During the 28 days of therapy, recombinant growth hormone was given by subcutaneous injection at a dose ranging from 0.03 to 0.14 mg/kg/day (average dose 0.11 mg/kg/day). Supplemental glutamine was provided by both the parenteral and enteral routes. The parenteral glutamine dosage averaged 0.6 g/kg/day, whereas a standard daily dose of 30 g glutamine was administered orally in six equal portions of 5 g mixed with a hypotonic cold beverage. In addition to the growth hormone and glutamine, all patients underwent extensive diet modification and nutritional education, the details of which have been reported extensively elsewhere [55]. On completion of the 4-week protocol, growth hormone was discontinued, and the patients were discharged home on 30 g/day oral glutamine and the modified oral diet [7, 21].

The initial balance studies indicated improvement in absorption of protein by 39 %, accompanied by a 33 % decrease in stool output with the regimen. In evaluation of the long-term results, averaging 1 year and extending as long as 5 years, 40 % of those studied remained off TPN, and an additional 40 % reduced their TPN requirements, with no change in TPN requirements in the remaining 20 %. These changes had occurred in a subset of patients that had previously failed to adapt to the provision of enteral nutrients, and this therapy may offer an alternative to long-term dependence on TPN for some patients with severe SBS. Subsequently, a more comprehensive clinical study of greater than 300 patients has been reported by the same group of investigators [56, 57]. However, growth hormone alone has not been shown to be beneficial consistently in other randomized, blind, placebo-controlled, crossover studies, and the Bryne et al. study results have not been reproduced by other investigators [58–60]. These conflicting data emphasize the need for further clinical studies to evaluate the effects of trophic agents on intestinal adaptation [61]. Both growth hormone and glutamine are available for clinical use, but growth hormone generally is not used routinely or often because of its high cost, side effects, and questionable efficacy [58, 62].

A recent review article on the management options in SBS reported that administration of glucagon-like peptide-2 (GLP-2) to patients following major small bowel resection improved intestinal adaptation and nutrient absorption [63]. Teduglutide, an enzyme-resistant GLP-2 analog, has shown promise in preventing intestinal injury, restoring mucosal integrity, increasing villous height, enhancing intestinal absorptive function, and increasing lean body mass, based on data from ongoing clinical trials in patients with SBS [64–69]. However, further studies and the completion of current phase III trials are necessary to determine the appropriate dosage (high vs. low) and length of treatment required for these patients to gain optimal benefits from the administration of this agent [63, 64].

Surgical Considerations

Total parenteral nutrition is the mainstay of early and sometimes late management of SBS [56]. Prior to the widespread use of TPN, patients often survived the initial surgical insult of massive small bowel resection and its early complications only to die ultimately of fluid, electrolyte, and nutritional imbalances. Today, however, patients can usually be managed successfully and often rehabilitated with the judicious use of TPN. In this regard, the surgeon is required to insert, maintain, and supervise a temporary and subsequently a permanent indwelling central venous catheter or catheter port for administration of TPN solutions [7, 21].

Massive small bowel resection is associated with a prompt and inordinate increase in the secretion of gastrin and gastric acid. The resulting hypersecretion can readily cause or aggravate existing gastritis, ulceration, bleeding, diarrhea, and fluid and electrolyte depletion. Because the hypersecretion is thought to be mediated hormonally, truncal vagotomy and pyloroplasty have been performed in human beings with good results [2]. Now that effective H_2 receptor blockers have been developed for clinical use, the surgical treatment of hypersecretion is seldom indicated or required. Currently, vagotomy or other acid-reducing operations should be reserved only for those SBS patients who develop complicated peptic ulceration problems resistant to conservative medical therapy. Partial or total gastric resections in patients with SBS should be avoided assiduously.

In patients with SBS following massive intestinal resection, parenteral nutrition should be given for at least 6–12 months to ensure that optimal bowel adaptation has occurred before contemplating the use of any surgical procedures to increase absorption of nutrients [39]. In most SBS patients, sufficient bowel adaptation occurs during the first year following massive intestinal resection so that parenteral nutrition can be discontinued, and contemplated surgical interventions can be avoided [7, 21].

Thompson has recently reviewed his extensive operative experience with adjunctive management of SBS patients [58]. He posited that if an adult with SBS develops intestinal dilation, it usually is secondary to obstruction, either secondary to recurrent intra-abdominal adhesions or at the site of a previous anastomosis. Bacterial overgrowth often develops in dilated, relatively hypotonic bowel and compounds the malabsorption secondary to SBS. Although conservative management is preferable initially, surgery is usually required to relieve intestinal obstruction, which may include lysis of adhesions, stricturoplasty, or minimal segmental resections only as absolutely necessary [58, 70]. Dilation of the intestinal remnant occurs more frequently in children than in adults and appears to have a basis that is more adaptive in nature than pathologic [58, 71]. In patients with adequate bowel length, longitudinal taper enteroplasties have been used to restore the dilated lumen diameter toward normal. Tapering enteroplasties may be either resective or imbricating, with no significant differences between the approaches [58, 71]. Lengthening procedures are not performed on obstructed bowel in an effort to "create length," but rather to relieve the functional obstruction and to allow the bowel transit to return toward normal. To restore luminal diameter, Thompson and others have found the so-called intestinal-lengthening procedures to be the optimal treatment [58, 71]. Although easiest to describe as lengthening, Thompson stated that these procedures actually more truly represent an attempt to optimize the ratio of volume to surface area of the intestine to improve contact time between luminal contents and absorption surface [58]. The initial operative approach was longitudinal lengthening via the Bianchi procedure, which involves meticulous dissection of the mesentery of the bowel segment to allocate terminal blood vessels to either side of the bowel wall [58, 71–73]. Longitudinal transection of the bowel is then performed, usually with a stapling device, which creates two parallel vascularized limbs of a smaller caliber, which can then be anastomosed effectively to lengthen the intestinal remnant through which the chyme must flow [58, 71, 72]. More than 100 cases have been reported, mostly in children, with overall improved nutrition in approximately 80 % of patients [58, 71]. Complications have been reported after 20 % of procedures, with the complications not surprisingly including ischemia, anastomotic leaks, and recurrent dilation [58, 71]. However, follow-up for up to 10 years suggests that long-term benefits occurred in 50 % of patients, while 10 % ultimately underwent intestinal transplantation [58, 71].

An alternative method of lengthening, serial transverse enteroplasty (STEP), has been introduced, consisting of repeated applications of a linear stapling device from opposite directions in a zigzag fashion, which divides the bowel about 50 % of its diameter from either the mesenteric and antimesenteric sides or transversely [58, 73, 74]. Thompson indicated that this procedure ideally involves complete release of adhesions from the duodenum to the colon, and then a combination of tapering enteroplasties or STEP enteroplasties restore a uniform bowel lumen appropriate for the size of the patient. He typically required a bowel diameter of at least 4 cm before performing a STEP enteroplasty to maintain a subsequent lumen diameter of about 2 cm [58]. Motility can be somewhat slow to return, and in general, the full benefit of a STEP taper procedure is not often realized until 8–12 weeks after surgery [58]. More than 70 cases of STEP have been reported in the literature, with clinical improvement in 80 % of patients; 5 % underwent subsequent intestinal transplantation [71].

Thompson summarized his experience with these procedures as follows:

> Our experience with the STEP technique has been quite favorable, and it has now become our procedure of choice [58, 73, 75]. We found that 58 % of 64 patients undergoing either the Bianchi procedure or STEP were able to discontinue Parenteral Nutrition (PN). This correlated with the length gained and total length after the procedure. Overall clinical outcome is similar with STEP and Bianchi procedures. STEP avoids the difficult dissection along the mesenteric border required of the Bianchi procedure and the end to end anastomosis. While bowel may have to be more dilated to use this technique, it is more feasible in challenging areas such as near the ligament of Treitz. There are no prohibitions to performing either repeat STEP procedures or tapering enteroplasties at later operations [58].

Attempts to ameliorate the untoward effects of SBS surgically by interposing isoperistaltic or antiperistaltic bowel segments, intestinal valves, or recirculating loops; by pacing the intestine electrically; by growing new intestinal mucosa; and by transplanting small intestine have been of limited additional value to date [76]. Therefore, no operative procedure for adjunctive management of SBS currently is sufficiently safe and effective to recommend its routine use [58, 73]. Long-term parenteral nutrition remains the cornerstone of successful management of SBS, and its judicious use is recommended in appropriate amounts and formulations for as long as needed not only to ensure maximal GI adaptation and nutritional rehabilitation of the patient but also to support the optimal size and function of the body cell mass [7].

Intestinal Transplantation in Patients with Short-Bowel Syndrome

Since the early 2000s, intestinal transplantation has been increasingly applied as a rescue therapy for patients with life-threatening complications of SBS and other forms of intestinal failure [63]. When the complications include portal hypertension or progressive liver failure, patients with SBS become candidates for combined liver/small intestine transplantation [58, 77]. The generally accepted indications for intestinal transplantation include recurrent sepsis, loss of central venous access, and development of progressive liver disease. Intestinal transplants have also been used following extensive resection of retroperitoneal neoplasms such as desmoids, fibromas, and neuroendocrine tumors, during which the superior mesenteric artery and its dependent bowel are sacrificed in deference to potential cure [58].

To date, almost 2,000 intestinal transplants have been performed in the United States, approximately 75 % of which have been in recipients under 18 years of age [58, 77]. One-year graft survival rates are currently as high as 89 % in adults aged 18–34 and as low as 64 % in children under 1 year of age [58, 77]. Graft survival drops at 5 years, with published rates ranging as low as 31 % in children under 1 year of age to as high as 69 % in children aged 6–10 years of age [58, 77]. Patient survival rates are similar at 1 and 5 years after transplant [58, 77–79]. Chronic rejection and infectious complications remain important determinants of survival, and improvements in outcomes since the mid-1990s have in large part been related to improved pediatric critical care and to judicious management of immunosuppression to reduce the incidence of opportunistic infections and post-transplant lymphoproliferative disorder [58, 79]. Overall, it is increasingly being recognized that the treatment of intestinal failure involves both nutritional and metabolic rehabilitation and transplantation, and that these approaches are complementary rather than competitive or contradictory [58].

Information regarding long-term nutritional outcome and quality of life (QOL) is continually emerging [58, 77, 80, 81]. Approximately one-third of patients undergoing intestinal transplantation require parenteral nutrition at discharge; however, at 1 year, 90 % are independent of it [58, 77]. QOL has been improved in almost all areas, but particularly related to digestive function, vocational abilities, medical compliance, optimism, and energy [58, 80]. On the other hand, this should be interpreted cautiously in view of more recent studies suggesting that QOL in SBS transplant patients remains lower than in nontransplant controls [58, 81].

Of all of the surgical approaches to SBS, intestinal transplantation has the greatest potential for treating selected patients with SBS, in terms of both the number of patients who might benefit and the functional improvement achieved [58]. With greater experience and improved results, it is hoped that this therapy can be extended to a larger number of patients with SBS [58, 63]. Thompson recommended that patients with high-risk complications of intestinal failure be referred early to a center specializing in intestinal transplantation so that patients might be carefully managed and monitored by an experienced team and, if needed, listed for transplant prior to development of complications that preclude the operation [58].

Summary and Conclusions

Short-bowel syndrome is a form of intestinal failure following massive intestinal resection for a variety of conditions in which the remaining length of small bowel has inadequate capabilities for the absorption of the required water,

macronutrients, and micronutrients to support optimal health, functions, and performance of the body cell mass. Some of these conditions or situations are accompanied by, result in, or result from complex abdominal wall defects. Notable are the clinical scenarios that often accompany the treatment of abdominal compartment syndrome by the various "open abdomen" techniques. The complex pathophysiology of SBS was summarized together with its clinical consequences. Nutritional and metabolic management of SBS can be characterized arbitrarily by three overlapping periods of therapy, which were discussed in some detail and have withstood the tests of time for a few decades. This was followed by a summation of the more recent efforts to enhance intestinal absorption by incorporating the use of growth hormone, teduglutide, glutamine, and other nutraceuticals, in combination with dietary modifications, in attempts to reduce or obviate the use of long-term parenteral nutrition in selected patients while promoting maximal adaptation of the intestine. Surgical considerations in the adjunctive management of SBS were discussed as potential means of enhancing intestinal absorption. Increasing the exposure of the intestinal chyme to the mucosal enterocytes by decreasing intestinal transit and overcoming functional bowel obstructions with a variety of specialized surgical procedures has been helpful in appropriate patients. Of all of the surgical approaches to SBS management, intestinal transplantation may well have the greatest promise in terms of restoring GI tract function to normal as this field of endeavor continues to advance and improve its long-term outcomes. Finally, parenteral nutrition remains the cornerstone of optimally successful management of SBS, and its judicious use and monitoring by expert, experienced, dedicated nutrition support teams can ensure safe, effective, and maximal GI adaptation and nutritional rehabilitation of the patient while maintaining the optimal size and function of the body cell mass.

References

1. Dudrick SJ. Foreword. In: Langas A, Goulet E, Tappenden K, editors. Intestinal failure: diagnosis, management and transplantation. Oxford: Blackwell; 2008.
2. Allard J, Jeejeebhoy K. Nutritional support and therapy in the short bowel syndrome. Gastroenterol Clin North Am. 1989;18:589–601.
3. Deitel M, Wong KH. Short bowel syndrome. In: Deitel M, editor. Nutrition in clinical surgery. 2nd ed. Baltimore: Williams & Wilkins; 1985. p. 255–75.
4. Dudrick SJ, Jackson D. The short bowel syndrome and total parenteral nutrition. Heart Lung. 1983;12:195–201.
5. Goutrebel M, Saint-Aubert B, Astre C, et al. Total parenteral nutrition needs in different types of short bowel syndrome. Dig Dis Sci. 1986;31:713–23.
6. Weser B. Nutritional aspects of malabsorption. Short gut adaptation. Clin Gastroenterol. 1983;12:443–61.
7. Dudrick SJ, Abdullah F, Latifi R. Nutrition and metabolic management of short bowel syndrome. In: Lafiti R, Dudrick S, editors. The biology and practice of current nutritional support. 2nd ed. Georgetown: Landes Bioscience; 2003. p. 261–74.
8. Tilson MD. Pathophysiology and treatment of short bowel syndrome. Surg Clin North Am. 1980;60:1273–84.
9. Dudrick SJ, Englert DM. Management of the short bowel syndrome. In: Miller TA, Dudrick SJ, editors. The management of difficult surgical problems. Austin: University of Texas Press; 1981. p. 225–35.
10. Dudrick SJ, O'Donnell JJ, Englert DM. Ambulatory home parenteral nutrition for short bowel syndrome and other diseases. In: Deitel M, editor. Nutrition in clinical surgery. 2nd ed. Baltimore: Williams & Wilkins; 1985. p. 276–87.
11. Trier JS, Lipsky M. The short bowel syndrome. In: Sleidenger MH, Fordtran JS, editors. Gastrointestinal disease: pathophysiology, diagnosis, management. 4th ed. Philadelphia: WB Saunders; 1989. p. 1106–12.
12. Weser B, Fletcher JT, Urban E. Short bowel syndrome. Gastroenterology. 1979;77:572–9.
13. Wilmore DW, Dudrick SJ. Effects of nutrition on intestinal adaptation following massive small bowel resection. Surg Forum. 1969;20:398–400.
14. Wilmore DW, Holtzapple PG, Dudrick SJ, et al. Transport studies, morphological and histological findings in intestinal epithelial cells following massive bowel resection. Surg Forum. 1971;22:361–3.
15. Conn HJ, Chavez CM, Fain WR. The short bowel syndrome. Ann Surg. 1972;175:803–14.
16. Dudrick SJ. A clinical review of nutritional support of the patients. Am J Clin Nutr. 1981;34:1191–8.
17. Sheldon DF. Role of parenteral nutrition in patients with short-bowel syndrome. Med J Aust. 1979;67:1021–9.
18. Stewart GR. Home parenteral nutrition for short-bowel syndrome. Med J Aust. 1989;2:317–9.
19. Wilmore DW, Johnson DJ. Metabolic effects of short bowel reversal in treatment of short bowel syndrome. Arch Surg. 1968;97:784–91.
20. Wilmore DW, Dudrick SJ, Daly JM, et al. The role of nutrition in the adaptation of small intestine after massive resection. Surg Gynecol Obstet. 1971;132:673–80.
21. Dudrick SJ, Latifi R, Fosnocht D. Management of the short bowel syndrome. Surg Clin North Am. 1991;71:625–43.
22. Garcia VF, Templeton JM, Eichelberger MR, et al. Colon interposition for the short bowel syndrome. J Pediatr Surg. 1981;16:994–5.
23. Levine GM. Short gut syndrome and intestinal adaptation. In: Kurtz RD, editor. Nutrition in gastrointestinal disease. New York: Churchill Livingstone; 1981. p. 101–11.
24. Thompson JS, Rikkers LF. Surgical alternatives for the short bowel syndrome. Am J Gastroenterol. 1987;22:97–105.
25. Schecter WP, Hirshberg A, Chang DS, Harris HW, Napolitano LM, Wexner SD, et al. Enteric fistulas: principles of management. J Am Coll Surg. 2009;209(4):484–91.
26. Schecter WP. Management of entercutaneous fistulas. Surg Clin North Am. 2011;91(3):481–91.
27. Borgstrom B, Dahlquist A, Lundh G, et al. Studies of intestinal digestion and absorption in the human. J Clin Invest. 1957;36:1521–36.
28. Debongnie J, Philips S. Capacity of the human colon to absorb fluid. Gastroenterology. 1978;74:698–703.
29. Ricotta J, Zuidema FD, Gadacz RT, et al. Construction of an ileocecal valve and its role in massive resection of the small intestine. Surg Gynecol Obstet. 1981;152:310–4.
30. Wright HK, Tilson MD. Short gut syndrome: pathophysiology and treatment. Curr Probl Surg. 1971;8:1–51.
31. Strause E, Gerson E, Yalow RS. Hypersecretion of gastrin associated with the short bowel syndrome. Gastroenterology. 1974;66:175–80.
32. Philips SF, Giller J. The contribution of the colon to electrolyte and water conservation in man. J Lab Clin Med. 1973;81:733–46.

33. Bond JH, Currier BE, Buchwald H, et al. Colonic conservation of malabsorbed carbohydrates. Gastroenterology. 1980;78:444–7.
34. Bond JH, Levitt MD. Fate of soluble carbohydrate in the colon of rats and humans. J Clin Invest. 1976;57:1158–64.
35. Haverstad T. Studies of short-chain fatty acid absorption in man. Scand J Gastroenterol. 1980;21:257–60.
36. Pomare EW, Branch WJ, Cummings JH. Carbohydrate fermentation in the human colon and its relation to blood acetate concentration in venous blood. J Clin Invest. 1985;75:1148–54.
37. Capton JP, Gineston JL, Herve MA, et al. Metronidazole in prevention of cholestasis associated with parenteral nutrition. Lancet. 1983;1:446–7.
38. Dudrick SJ, Latifi R. Management of patients with short-bowel syndrome. In: Kirby DF, Dudrick SJ, editors. Practical handbook of nutrition in clinical practice. Boca Raton: CRC Press; 1994. p. 215–25.
39. Barnett WO, Oliver RI, Elliot RL. Elimination of the lethal properties of gangrenous bowel segments. Ann Surg. 1968;167:912–9.
40. Bounous G, McArdle AH. Release of intestinal enzymes in acute mesenteric ischemia. J Surg Res. 1968;9:343–8.
41. Ratych RE, Smith GW. Anatomy and physiology of the liver. In: Zuidema GD, editor. Shackleford's surgery of the alimentary tract. 3rd ed. Philadelphia: WB Saunders; 1991. p. 273–86.
42. Sarr WG, Tito WA. Intestinal obstruction. In: Zuidema GD, editor. Shackleford's surgery of the alimentary tract. 3rd ed. Philadelphia: WB Saunders; 1991. p. 372–413.
43. Woolf GM, Miller C, Kurian R, et al. Nutritional absorption in short bowel syndrome: evaluation of fluid, calorie and divalent cation requirements. Dig Dis Sci. 1987;32:8–15.
44. Cortot A, Fleming CR, Malagelada JR. Improved nutrient absorption after cimetidine in short bowel syndrome with gastric hypersecretion. N Engl J Med. 1979;300:79–80.
45. Ladefoged K, Christensen K, Hegnhoi J, et al. Effect of long-acting somatostatin analog SMS 201-995 on jejunostomy effluents in patients with severe short bowel syndrome. Gut. 1989;30:943–9.
46. Nightingale J, Walker E, Burnham W et al. Short bowel syndrome. Digestion. 1990;45(Suppl 1):77–83.
47. Levy E, Frileux P, Sandrucci S, et al. Continuous enteral nutrition during the early adaptive stage of the short bowel syndrome. Br J Surg. 1988;75:549–53.
48. Ovesen L, Chu R, Howard L. The influence of dietary fat on jejunostomy output in patients with severe short bowel syndrome. Am J Clin Nutr. 1983;38:270–7.
49. Woolf GM, Miller C, Kurian R, et al. Diet for patients with short bowel: high fat or high carbohydrate? Gastroenterology. 1983;84:823–8.
50. McIntyre P. The short bowel. Br J Surg. 1985;72:893–9.
51. McIntyre P, Fitchew M, Lennard-Jones J. Patients with a high jejunostomy do not need a special diet. Gastroenterology. 1986;91:25–33.
52. Ladefoged K. Intestinal and renal loss of infused minerals in patients with severe short bowel syndrome. Am J Clin Nutr. 1982;36:59–67.
53. Thompson JS. Surgical considerations in the short bowel syndrome. Surg Gynecol Obstet. 1993;36:59–67.
54. Byrne TA, Persinger RL, Young LS, et al. A new treatment for patients with short-bowel syndrome. Ann Surg. 1995;222:243–55.
55. Byrne TA, Morrissey TB, Nattakorn TV, et al. Growth hormone, glutamine and a modified diet enhance nutrient absorption in patients with severe short bowel syndrome. J Parenter Enteral Nutr. 1995;19:296–302.
56. Wilmore DW, Byrne TA, Persinger RL. Short bowel syndrome: new therapeutic approaches. Curr Probl Surg. 1997;34:389–444.
57. Wilmore DW. Growth factors and nutrients in the short bowel syndrome. J Parenter Enteral Nutr. 1999;23:S117–20.
58. Thompson JS, Weseman R, Rochling FA, Mercer DF. Current management of the short bowel syndrome. Surg Clin North Am. 2011;91(3):493–510.
59. Scolapio JS, Camilleri M, Fleming CR, et al. Effect of growth hormone, glutamine, and diet on adaptation in short bowel syndrome: a randomized controlled trial. Gastroenterology. 1997;115:1075–81.
60. Szkudlarek J, Jeppesen PB, Mortensen PB. Effect of high dose growth hormone with glutamine and no change in diet on intestinal absorption in short bowel patient: a randomized double blind, crossover, placebo controlled study. Gut. 2000;47:199–205.
61. Platell CFE, Coster J, McCauley RD, Hall JC. The management of patients with the short bowel syndrome. World J Gastroenterol. 2002;8(1):13–20.
62. Wales PW, Nasr A, de Silvea N et al. Human growth hormone and glutamine for patients with short bowel syndrome. Cochrane Database of Syst Rev. 2010;issue 6. Art No. CD006321, doi: 10.1002/14651858.CD006321.pub2
63. Seetharam P, Rodrigues G. Short bowel syndrome: a review of management options. Saudi J Gastroenterol. 2011;17:229–35.
64. Ferone M, Scolapio JS. Teduglutide for the treatment of short bowel syndrome. Ann Pharmacother. 2006;40:1105–9.
65. Mardini HE, de Villiers WJ. Teduglutide in intestinal adaptation and repair: light at the end of the tunnel. Expert Opin Investig Drugs. 2008;17(6):945–51.
66. Jeppesen PB, Gilroy R, Pertkiewicz M, et al. Randomized placebo-controlled trial of teduglutide in reducing parenteral nutrition and/or intravenous fluid requirements in patients with short bowel syndrome. Gut. 2011;60(7):902–14.
67. Mouksassi MS, Marier JF, Cyran J, Vinks AA. Clinical trial simulations in pediatric patients using realistic covariates: application to teduglutide, a glucagon-like peptide-2 analog in neonates and infants with short bowel syndrome. Clin Pharmacol Ther. 2009;86(6):667–71.
68. Wallis K, Walters JR, Gabe S. Short bowel syndrome: the role of GLP-2 on improving outcome. Curr Opin Clin Nutr Metab Care. 2009;12(5):526–32.
69. Yazbeck R. Teduglutide, a glucagon-like peptide-2 analog for the treatment of gastrointestinal diseases, including short bowel syndrome. Curr Opin Mol Ther. 2010;12(6):798–809.
70. Thompson JS, Langnas AN. Surgical approaches to improving intestinal function in short bowel syndrome. Arch Surg. 1999;134:706–71.
71. Thompson JS, Sudan DA. Intestinal lengthening in the short bowel syndrome. Adv Surg. 2008;42:49–61.
72. Bianchi A. Experience with longitudinal intestinal lengthening and tailoring. Eur J Pediatr Surg. 1999;9:256–9.
73. Sudan D, Thompson JS, Botha J, et al. Comparisons of intestinal lengthening procedures for patients with short bowel syndrome. Ann Surg. 2007;246:593–604.
74. Kim H, Fauza D, Garza J, et al. Serial transverse enteroplasty (STEP): a novel bowel lengthening procedure. J Pediatr Surg. 2003;38:425–9.
75. Yannam G, Sudan D, Grant W, et al. Intestinal lengthening in adults with short bowel syndrome. J Gastrointest Surg. 2010;14:1931–6.
76. Thompson JS, Sudan DA, Gilroy R. Predicting outcome of procedures to slow intestinal transit. Transplant Proc. 2006;38:1838–9.
77. Mazariegos GV, Steffick DE, Horslen S, et al. Intestinal transplantation in the United States 1999–2008. Am J Transplant. 2010;10:1020–34.
78. Grant D, Abu-Elmagd K, et al. 2003 report of the Intestine Transplant Registry. Ann Surg. 2005;241:607–13.
79. Abu-Elmagd K, Costa G, Bond GJ, et al. Five hundred intestinal and multivisceral transplantations at a single center. Ann Surg. 2009;250:567–81.
80. O'Keefe SID, Emerling M, Kovitsky D, et al. Nutrition and quality of life following small intestinal transplantation. Am J Gastroenterol. 2007;102:1093–100.
81. Sudan D. Long term outcomes and quality of life after intestinal transplantation. Curr Opin Organ Transplant. 2010;15:357–60.

Nutritional Management of Gastroenterocutaneous Fistulas

23

Albert Chi, Michael Ditillo, and Bellal Joseph

Introduction

The etiology, epidemiology, and classification of gastrointestinal (GI) fistulas are complex. The majority of fistulas develop as a complication of abdominal surgery, trauma, Crohn's disease, intra-abdominal abscess, malignant disease, or radiotherapy. Enterocutaneous fistulas (ECFs) are abnormal connections between two epithelialized surfaces, generally from the bowel to skin, through which enteric contents pass. Fistulas in the GI system are classified on the basis of the site of origin and termination, volume of drainage, and etiology. Each component of the classification is important in that all parts have treatment implications. In general, medical treatment and stabilization precede attempts at surgical intervention. In patients with all forms of enteric fistulas, sepsis is a major cause of mortality and must be treated aggressively. Surgical treatment is reserved for patients whose fistulas do not resolve with medical and nonsurgical therapy.

Gastrointestinal fistulas may occur after surgery or spontaneously. An estimated 80 % of GI fistulas occur as complications after abdominal surgery, with an estimated overall incidence of 0.8–2 % [1]. Fistula-associated morbidities include malnutrition, electrolyte imbalances, skin excoriation, abscess formation, and sepsis. The development of ECFs in trauma patients has been shown to increase length of stay an average of 21 days in the intensive care unit (ICU) and 66 days in the hospital. Mortality rates range from 5 to 20 %, and are the result of sepsis, electrolyte imbalance, and malnutrition [2].

Understanding the pathophysiology of, as well as the risk factors for, ECFs should help reduce their occurrence. Their care and management still present a considerable medical and surgical challenge. Moreover, the well-established treatment guidelines for ECFs, along with some newer treatment options, should help clinicians achieve better outcomes. That said, there are few data in the management of patients with ECFs, and their management is challenging.

The anatomic classification of enteric fistulas is based on the segment of bowel from which they originate (i.e., enterocutaneous, gastrocutaneous, colocutaneous, etc.). The etiologic classification is based on the underlying disease process (i.e., postoperative, trauma, foreign body, Crohn's disease, diverticulitis, tuberculosis, malignancy). The most important physiological determinant of a fistula is the daily output of intestinal fluid. Fistula output is a predictor of morbidity and mortality, and although not an independent indicator of spontaneous closure, 24-h output generally decreases prior to closure. While fistula mortality rates have decreased over the past few decades from as high as 40–65 to 5.3–21.3 %, high-output fistulas continue to have a mortality rate of approximately 35 % [3].

The physiologic classification is based on the volume of fistula output:

- A low-output fistula drains less than 200 mL/day.
- A moderate-output fistula drains between 200 and 500 mL/day.
- A high-output fistula drains more than 500 mL/day and up to 3,000 mL or more of fluid daily.

Classification is key in understanding the management and treatment options. The difficulties presented by a high-output fistula with such massive losses of water, electrolytes, and nutrients are daunting. There is a significant but lesser degree of malnutrition with moderate-output fistulas; low-output fistulas have a much lower incidence of associated malnutrition.

A. Chi, MD
Department of Surgery, Johns Hopkins Hospital,
1800 Orleans Street, Baltimore, MD 21287, USA
e-mail: achi3@jhmi.edu

M. Ditillo, DO
Department of Surgery, Yale School of Medicine,
330 Cedar Street BB 310, New Haven, CT 06519, USA
e-mail: michael.ditillo@yale.edu

B. Joseph, MD (✉)
Department of Surgery, Division of Trauma, Critical Care,
and Emergency Surgery, University of Arizona/University
Medical Center, 1501 N. Campbell Ave., 245063,
Tucson, AZ 85724, USA
e-mail: bjoseph@surgery.arizona.edu

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2_23,

Management of Enterocutaneous Fistulas

Gastrointestinal fistula exudate is typically comprised of a rich mixture of sodium, potassium, chloride, and bicarbonate ions; proteins; and other components. Large volumes of GI secretions might be lost through fistulas, which potentially results in profound disturbances in fluid and electrolyte levels, leading to dehydration, hyponatraemia, hypokalemia, and metabolic acidosis. The degree of the deficit caused by the fistula is directly proportional to volume and composition. Initial treatment of an ECF focuses on the correction of fluid and electrolyte imbalance, abscess drainage and treatment of infection, correction of malnutrition, and meticulous fistula control and skin care. Approximately one-third of ECFs will heal spontaneously with these measures within 5–6 weeks. Patients with a fistula should not be allowed to eat (should be NPO [nil per os]) during the initial stage of treatment. The NPO status means absolutely nothing should be allowed by mouth, even ice chips, if the goal of minimizing output is to be achieved.

Nutritional support should be initiated after correction of fluid, electrolyte, vitamin, blood volume, and clotting deficits. Gastric acid secretion and intestinal and pancreatic secretion are initially inhibited by intravenous H_2 receptor blockers and parenteral somatostatin. Enteral feedings are preferable because of the positive effects on immunologic and hormonal gut function but are often impractical because of feeding intolerance, lack of access to the GI tract, or high-output fistula losses. The caloric intake is calculated at 25–30 Kcal/kg body weight per day. It is important to note usually only one-third to one-half of the caloric ration is given as dextrose on the first day. After tolerance and utilization of the dextrose are established, the concentration and dosage are gradually increased over the next few days to meet full caloric requirements. In general, patients with low-output fistulas should receive the full basal energy requirement and between 1 and 1.5 g of protein per kilogram body weight every day, with a minimum of 20 % of the caloric intake supplied as lipid. The primary role of the fat emulsion is to prevent essential fatty acid deficiency, although this is still a controversial issue. With high-output fistulas, patients should receive 1.5–2 times their basal energy expenditure plus 1.5–2.5 g of protein per kilogram body weight per day. This nutritional regimen should also include twice the recommended daily allowance (RDA) for vitamins and trace minerals, up to ten times the RDA for vitamin C, and zinc supplements.

The role of artificial nutrition, provided as either total parenteral nutrition (TPN) or enteral nutrition (EN), is primarily that of supportive care to improve the malnourished status of the patient and provide GI tract rest. In some cases, parenteral nutrition does not need to be total, as patients can have oral intake. Nutritional support is associated with a decrease in fistula output, appears to modify the composition of GI and pancreatic secretions, and therefore may be considered to have a primary therapeutic role. Indeed, TPN has been the mainstay of conservative management of GI fistulas throughout the last three decades. Conservative treatment with TPN has been shown to reduce the maximal secretory capacity of the GI tract by 30–50 %, induce protein synthesis, and promote favorable conditions for closure. However, the use of TPN can be associated with potentially serious complications, such as bacterial translocation, superinfection of central venous access, and metabolic disorders as a result of fistula losses. Generally, TPN is indicated in patients with gastroduodenal, pancreatic, or jejunoileal fistulas, and EN is provided for fistulas of the esophagus, distal ileum, and colon. TPN might also be beneficial if fistula output is increased or patients are intolerant of EN.

Total Parenteral Nutrition

Since the 1970s, the mainstay of treatment has been supportive, with initiation of an NPO regimen and intravenous (parenteral) nutrition with the aim of stabilizing the patient and inducing GI tract rest. In 1967, Dudrick et al. [4] described the growth of intravenously fed beagle puppies that experienced normal weight gain and normal growth as compared with their orally fed counterparts. Major achievements by Dudrick then brought this new therapy from the laboratory to the clinical bedside; the technique was refined so that it could be applied with low morbidity. Early nutritional support via TPN has the potential to reduce disease severity, diminish complications, and decrease the ICU length of stay. When EN is not possible, TPN gives clinicians the ability to fulfill patients' ongoing requirement parenterally for calories, protein, electrolytes, vitamins, minerals, trace elements, and fluids. TPN use has been studied in patients with a wide array of clinical conditions, such as trauma, cancer, inflammatory bowel disease, short-gut syndrome, radiation enteritis, poor wound healing, and GI fistula. Yet, few well-designed, randomized, controlled trials of the efficacy of TPN in critically ill and injured patients have been conducted. It is well known that 20–40 % of critically ill and injured patients exhibit some form of malnutrition. Of that subgroup, 85–90 % can be treated with EN. In the remaining 10–15 %, EN is contraindicated; TPN, delivered intravenously, provides the only support.

Role of Somatostatin

The pharmacological agents somatostatin-14 and its analogue octreotide have been used in addition to artificial nutrition because of their inhibitory effects on GI secretions. There is evidence to suggest that the greater the fistula output, the more effective octreotide is in reducing the volume of output. The dose of somatostatin-14 used for digestive fistulas is an initial bolus of 250 μg plus a continuous intravenous

infusion of 250 μg/h until closure, followed by 3 mg/day (125 μg/h) for 48 h to protect against fistula recurrence. It is important that continuous infusion of somatostatin-14 is not interrupted. If continuous infusion is interrupted, a rebound effect might be seen, during which time GI secretions can increase, and this may lead to reduced efficacy. However, this can be avoided if the infusion is reinstated as soon as possible with another bolus of 250 μg.

Somatostatin-14 and its analogues are not intended as a replacement for conservative treatment. Instead, when used in combination, somatostatin-14 and TPN appear to exert a synergistic effect on the reduction of GI secretions and improve fistula closure rates. Unlike TPN, somatostatin-14 totally inhibits basal exocrine GI secretions and suppresses the possibility of exogenous stimuli. The dual therapy combines the effects of TPN on protein synthesis induction with total inhibition of fistula losses by somatostatin-14, which is the primary condition for spontaneous closure. The information currently available seems to suggest a beneficial effect of somatostatin-14 when administered in association with standard conservative treatment, although current data are insufficient to draw firm conclusions. However, outcomes with respect to reduction in time to spontaneous closure are particularly promising and certainly warrant further investigation in well-controlled blinded studies.

Enteral Nutrition

Over the 2000s decade, there has been increasing interest in the use of specially formulated enteral and parenteral feedings, with the goal of influencing and altering the body's immune response to injury and critical illness. There is an increasing body of literature that shows a potential benefit of these specialty formulas in the management of malnourished and critically ill patients. As such, supplements such as glutamine, arginine, and omega-3 fatty acids may play a role in immunomodulation as well as make a contribution to overall GI function in patients with inflammatory bowel disease and short-gut syndrome. To date, there have been no studies that examined the roles of these formulas in the treatment of ECFs; however, they may benefit through their overall immunomodulating effect and contribution to GI health, as well as overall improvement in nutrition.

Enteral nutrition, when compared to parenteral nutrition, has fewer serious complications and is less expensive. EN formulas differ in their protein and fat content and can be classified as elemental (monomeric), semielemental (oligomeric), polymeric, or specialized. Elemental formulas contain individual amino acids and glucose polymers and are low fat, with only about 2–3 % of calories derived from long-chain triglycerides. Semielemental formulas contain peptides of varying chain length, simple sugars, glucose polymers or starch, and fat, primarily as medium-chain triglycerides. Polymeric formulas contain intact proteins, complex carbohydrates, and mainly long-chain triglycerides. Specialized formulas contain biologically active substances or nutrients such as glutamine, arginine, nucleotides, or essential fatty acids. Although elemental and semielemental formulas cost about 400 % more than polymeric formulas, they are still widely used because they are believed to be better absorbed, less allergenic, and better tolerated in patients with malabsorptive states and to cause less exocrine pancreatic stimulation. Although there have been no cases that looked at the affects of the formulas, there has been one case series by Teubner et al. that looked at patients with ECFs and their ability to tolerate polymeric formulas [5]. No other case series or studies in patients with ECFs compared different types of formulas, and there were no studies that reported the use of pancreatic enzymes to avoid the need for semielemental or elemental formulas in these patients.

Immune-Modulating Nutritional Supplementation

Glutamine, although not recognized as an essential amino acid, is considered conditionally essential during periods of metabolic stress and illness [6]. Glutamine acts as an energy and nitrogen source for intestinal mucosa and lymphocytes. It also serves as a respiratory substrate for enterocytes and other rapidly dividing cells, such as endothelial cells and proliferating cells in wounds and areas of inflammation [7]. After surgery, an increase in glutamine utilization as a primary fuel source by enterocytes as well as other rapidly dividing cells has been identified. Supplementation of glutamine has been shown to have a trophic effect in intestinal mucosa.

A recent prospective, double-blind, randomized trial of patients with major burns (>50 % body surface area [BSA]) demonstrated that supplemental intravenous glutamine infused continuously over 24 h provided significantly better support than isonitrogenous enteral or parenteral amino acid solutions without glutamine. In that trial, 26 severely burned patients (i.e., full thickness burns 25–90 % BSA) were randomized. The group randomized to glutamine containing nutrition had a lower incidence of gram-negative bacteremia as well as significant improvements in serum transferrin and prealbumin 14 days after injury. Furthermore, in the glutamine group, a trend toward lower mortality rate, a decreased incidence of bacteremia, and less antibiotic use were noted [8]. Decreased concentrations of glutamine are associated with immune dysfunction and increased rates of complications [9]. In a meta-analysis by Novak et al., the use of glutamine supplementation in critically ill patients resulted in a reduction in infectious complications (relative risk [RR] 0.08; 95 % confidence interval [CI] 0.64–1.00); however, this was not associated with a decrease in mortality [10].

Arginine is considered to be a nonessential amino acid in the diet of healthy adults but has been identified as a conditionally

essential amino acid in the critically ill patient. Arginine stimulates the release of growth hormone and prolactin, induces the release of insulin, improves weight gain, and increases wound healing. It has also been shown to accelerate wound healing, and it has a trophic effect on the immune system. The potential benefit of arginine in critically ill patients includes enhanced protein metabolism, improved microcirculation and organ function, augmented immune function, increased antibacterial effects, improved gut function, and possible antioxidant effects [11–13].

An often-overlooked part of artificial nutrition is the role of micronutrient supplementation. Micronutrients include vitamins, minerals, and trace elements. The majority of water-soluble vitamins are absorbed via the proximal small GI tract. Fat-soluble vitamins are absorbed in the mid- to distal ileum because of their dependence on bile and pancreatic lipase. Digestion of food needs to be accomplished before trace elements become bioavailable. Zinc, iron, and selenium are mainly absorbed by the duodenum and jejunum, whereas chromium and copper are absorbed by the ileum [14].

Micronutrient deficiencies are based on inadequate or inappropriate administration during artificial nutrition or as a consequence of increased requirements or bodily losses associated with critical illness [15]. The exact requirements of micronutrients in critically ill patients are unknown. Abnormally low levels may reflect redistribution rather than a true deficiency. Based on the understanding that micronutrients play a role in the maintenance of the body's defensive and reparative processes, the U.S. Food and Drug Administration (FDA) in 1984 made recommendations on the dosage of parenteral vitamin supplementation [16]. Although the FDA has not made similar recommendations for trace elements, the American Society for Parenteral and Enteral Nutrition established guidelines in 2002 [17]. This being said, to date there are no studies that focused on the role that micronutrients play in the treatment of fistulas. Having said this, by their nature, ECFs disrupt the anatomical sites of normal micronutrient absorption, and loss of enteric content leads to loss of both vitamins and trace elements. As such, understanding the anatomy/location of the fistula plays a key role in anticipating the loss of key micronutrients and leads to the prevention of deficiencies either by "refeeding" GI losses distal to the fistula or via parenteral supplementation [18].

Conclusion

Nutritional management of patients with ECFs is challenging. The management requires patient- as well as fistula-specific factors to be considered for optimizing the best treatment regimen. Currently, there are no well-established, evidence-based clinical guidelines for managing the medications and nutrition care of these patients. Malnutrition is common, and adequate nutritional provision is essential. Although it is often difficult and sometimes impossible to provide adequate EN in the presence of an ECF, it should be implemented whenever possible. Supplemental parenteral nutrition is often required for high-output small bowel fistulas. The role of immunonutrition at this point is unknown; however, it appears to be beneficial. In general, medical treatment and stabilization precede attempts at surgical intervention. In patients with all forms of enteric fistulas, sepsis is a major cause of mortality and must be treated aggressively. Surgical treatment is reserved for patients whose fistulas do not resolve with medical and nonsurgical therapy.

References

1. Wainstein DE, Fernandez E, Gonzalez D, Chara O, Berkowski D. Treatment of high-output enterocutaneous fistulas with a vacuum-compaction device. A ten-year experience. World J Surg. 2008;32:430–5.
2. Evenson AR, Fischer JE. Current management of enterocutaneous fistula. J Gastrointest Surg. 2006;10:455–64.
3. Makhdoom ZA, Komar MJ, Still CD. Nutrition and enterocutaneous fistula. J Gastroenterol. 2003;31:195–204.
4. Dudrick SJ. Early developments and clinical applications of total parenteral nutrition. J Parenter Enteral Nutr. 2003;27:291.
5. Teubner A, Morrison K, Ravishankar HR, et al. Fistuloclysis can successfully replace parenteral feeding in the nutritional support of patients with enterocutaneous fistula. Br J Surg. 2004;91(5):625–31.
6. Lacey JM, Wilmore DW. Is glutamine a conditionally essential amino acid? Nutr Rev. 1990;48:297–309.
7. Souba WW, Klimberg VS, Plumley DA, Salloum RM, Flynn TC, Bland KI, et al. The role of glutamine maintaining a healthy gut and supporting the metabolic response to injury and infection. J Surg Res. 1190;48:383–91.
8. Wischmeyer PE, Lynch J, Liedel J, Wolfson R, Riehm J, Gottlieb L, et al. Glutamine administration reduces gram-negative bacteremia in severely burned patients: a prospective, randomized, double-blind trial versus isonitrogenous control. Crit Care Med. 2001;29:2075–80.
9. Newsholme P. Why is L-glutamine metabolism important to cells of the immune system in health, postinjury, surgery or infection? J Nutr. 2001;131(suppl):2515S–22S.
10. Novak F, Heyland DK, Avenell A, Drover JW, Su X. Glutamine supplementation in serious illness: a systemic review of the evidence. Crit Care Med. 2002;30:2022–9.
11. Barbul A. Arginine and immune function. Nutrition. 1190;6:53–62.
12. Luiking YC, Poeze M, Ransay G, Deutz NEP. The role of arginine in infection and sepsis. J Parenter Enteral Nutr. 2005;29(suppl): S70–4.
13. DeBiasse MA, Wilmore DW. What is the optimal nutritional support? New Horiz. 1994;2:122–30.
14. Berger MM, Shenkin A. Vitamins and trace elements: practical aspects of supplementation. Nutrition. 2006;22:952–5.
15. Krishnan S, Lonchyna VA. Micronutrient supplementation in adult nutrition therapy: practical considerations. J Parenter Enteral Nutr. 2009;33(5):548–62.
16. Elia M. Changing concepts of nutrient requirements in disease. Implications for artificial nutritional support. Lancet. 1995;5:1279–84.
17. A.S.P.E.N. Board of directors and the Clinical Guidelines Taskforce. Guidelines for the use of parenteral and enteral nutrition in adult and pediatric patients. J Parenter Enteral Nutr. 2002;26(suppl):22SA–4SA. Errata 2002;26(2):144.
18. Joseph B, Wynne J, Dudrick S, Latifi R. Nutrition in trauma and critically ill patients. Eur J Trauma Emerg Surg. 2010;1:25–30.

Index

R. Latifi (ed.), *Surgery of Complex Abdominal Wall Defects*,
DOI 10.1007/978-1-4614-6354-2, © Springer Science+Business Media New York 2013