

Edited by Morris D. Kerstein

Consulting Editors
Watts R. Webb
Peter V. Moulder



University Park Press • Baltimore

Management of Vascular Trauma

Edited by

Morris D. Kerstein, M.D.

Professor Department of Surgery Tulane University School of Medicine New Orleans, Louisiana

Consulting Editors
Watts R. Webb, M.D.
Professor and Chairman

Peter V. Moulder, M.D.

Professor

Department of Surgery Tulane University School of Medicine New Orleans, Louisiana



University Park Press · Baltimore

University Park Press

International Publishers in Medicine and Allied Health 300 North Charles Street Baltimore, Maryland 21201

Copyright © 1985 by University Park Press

This book is protected by copyright. All rights, including that of translation into other languages, are reserved. No part of this book may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without the prior written permission of the publisher.

Sponsoring editor: Marjorie Nelson Production editor: Michael Treadway Cover and text design by: Caliber Design Planning, Inc.

Typeset by: Bi-Comp, Inc.
Manufactured in the United States of America by: Halliday Lithograph

Library of Congress Cataloging in Publication Data

Main entry under title:

Management of vascular trauma.

Includes index.

1. Blood vessels—Wounds and injuries—Surgery.

I. Kerstein, Morris D. II. Webb, Watts R., 1922-

III. Moulder, Peter V. [DNLM: 1. Blood Vessels—injuries.

2. Vascular Surgery. WG 170 M266]

RD598.5.M34 1984 617'.413044 84-17380

ISBN 0-8391-2002-8

Management of Vascular Trauma

This book is dedicated to Margaret, Lars, Jane, and Frances, and to our students and residents who provide the joy of teaching and the satisfaction of their success.

Preface

The prevalence of trauma, both penetrating and blunt, has established the need for increasing knowledge in the management of vascular trauma. The military experience has provided the foundations upon which we have built our patterns of care, and many of the rules associated with the management of atherosclerotic vascular disease are applicable.

The problems associated with vascular trauma are often related to delays in diagnosis and therapy or to lack of understanding of the biomechanics of trauma. It is the hope of the editors and contributors that this book will provide an awareness of the gravity of the injury and the need for prompt, organized, logical intervention. The book is not intended to be a technical text on vessel repair, but rather to focus on the pathophysiology, etiology, and general guidelines in management of injuries to peripheral vessels.

Many patients with isolated vascular injuries suffer disability, amputation, or even death. A proper assessment, an organized approach, and a sensitivity to potential complications will increase our success rate in the management of vascular trauma.

Acknowledgments

The editors wish to acknowledge the dedicated editorial efforts on the part of Gae O. Decker-Garrad. Her continued friendship and contributions make her an invaluable asset.

The enormous task of typing was completed by Debbie Moreau and Kathy Hackney; our gratitude is extended to them.

The interest, support, and patience of Marjorie Nelson of University Park Press are greatly appreciated by the editors.

Contributors

Michael F. Adinolfi, M.D.

Assistant Professor Department of Surgery Tulane University School of Medicine New Orleans, Louisiana (Chapter 2; Chapter 10)

Robert C. Batson, M.D.

Professor
Department of Surgery
Chief, Section of Vascular Surgery
Louisiana State University School
of Medicine
New Orleans, Louisiana
(Chapter 3; Chapter 8)

Arthur C. Beall, Jr., M.D.

Professor Department of Surgery Baylor College of Medicine Houston, Texas (Chapter 4)

O. William Brown, M.D.

Clinical Assistant Professor Wayne State University School of Medicine Detroit, Michigan (Chapter 2; Chapter 6)

John H. Davis, M.D.

Professor and Chairman
Department of Surgery
University of Vermont School of
Medicine
Burlington, Vermont
(Chapter 5)

Michael E. DeBakey, M.D.

Professor Department of Surgery Baylor College of Medicine Houston, Texas (Chapter 4)

Robert W. Feldtman, M.D.

Chief, Department of Cardiac Surgery Memorial City Medical Center Houston, Texas (Chapter 4)

Ray J. Haddad, Jr., M.D.

Professor and Chairman
Department of Orthopedic Surgery
Tulane University School
of Medicine
New Orleans, Louisiana
(Chapter 10)

John L. Hussey, M.D.

Professor
Department of Surgery
Tulane University School
of Medicine
New Orleans, Louisiana
(Chapter 7)

Morris D. Kerstein, M.D.

Professor
Department of Surgery
Tulane University School
of Medicine
New Orleans, Louisiana
(Chapter 2; Chapter 6; Chapter 7;
Chapter 8; Chapter 9)

Martin S. Litwin, M.D.

Professor
Department of Surgery
Tulane University School
of Medicine
New Orleans, Louisiana
(Chapter 1)

Thomas D. Martin, M.D.

Resident
Department of Surgery
Baylor College of Medicine
Houston, Texas
(Chapter 12)

Kenneth L. Mattox, M.D.

Professor Department of Surgery Baylor College of Medicine Houston, Texas (Chapter 4; Chapter 12)

Norman E. McSwain, M.D.

Professor
Department of Surgery
Tulane University School
of Medicine
New Orleans, Louisiana
(Chapter 1)

Robert C. Murphy, M.D.

Professor and Chief
Department of Plastic and
Reconstructive Surgery
The Johns Hopkins University
School of Medicine
Baltimore, Maryland
(Chapter 11)

Martin C. Robson, M.D.

Professor and Chairman
Department of Plastic and
Reconstructive Surgery
Wayne State University School
of Medicine
Detroit, Michigan
(Chapter 11)

Ricardo L. Rodriguez, M.D.

Resident
Department of Surgery
Tulane University School
of Medicine
New Orleans, Louisiana
(Chapter 8)

Daniel S. Rush, M.D.

Assistant Professor Department of Surgery Tulane University School of Medicine New Orleans, Louisiana (Chapter 2; Chapter 10)

Vikrom S. Sottiurai, M.D., Ph.D.

Assistant Professor
Department of Surgery
Louisiana State University School
of Medicine
New Orleans, Louisiana
(Chapter 3)

Thomas F. Sweeney, M.D.

Clinical Assistant Professor Department of Surgery Yale University School of Medicine New Haven, Connecticut (Chapter 9)

Contents

ix Acknowledgments

Preface

	Contributors xi
1	Biomechanics of Blunt and Penetrating Vascular Trauma 1 Martin S. Litwin and Norman E. McSwain
2	Effective Triage: Diagnostic Tools for Trauma Michael F. Adinolfi, O. William Brown, Daniel S. Rush, and Morris D. Kerstein
3	Vascular Injuries to the Neck Robert C. Batson and Vikrom S. Sottiurai
4	Vascular Injuries to the Thorax 52 Michael E. DeBakey, Kenneth L. Mattox, Arthur C. Beall, Jr., and Robert W. Feldtman
5	Vascular Injuries to the Abdomen 63 John H. Davis
6	Injuries to the Peripheral Vasculature 87 O. William Brown and Morris D. Kerstein
7	Traumatic Arteriovenous Fistulas John L. Hussey and Morris D. Kerstein 97
8	Vascular Injury Associated with Drug Abuse Ricardo L. Rodriguez, Robert C. Batson, and Morris D. Kerstein
9	Antibiotics in Surgery for Vascular Trauma 120 Thomas F. Sweeney and Morris D. Kerstein
10	Fasciotomy in Vascular Surgery 128 Daniel S. Rush, Michael F. Adinolfi, and Ray J. Haddad, Jr.
	•••

viii	Contents	
11	Microvascular Surgery in Trauma 148 Robert C. Murphy and Martin C. Robson	
12	Synthetic Materials in Vascular Trauma Thomas D. Martin and Kenneth L. Mattox	174
	Index 187	

1

Biomechanics of Blunt and Penetrating Vascular Trauma

Martin S. Litwin Norman E. McSwain¹

Vascular injury of varying severity and extent can be caused either directly by penetration, or indirectly by blunt forces applied at varying distances from the point of injury.

Penetrating Injury

The most common penetrating vascular injuries are knife-inflicted stabs and gunshot wounds. Variants of knife stabs, such as those due to impalement, are the result of the same sort of physical forces and, hence, are treated in the same fashion. Gunshot wounds, however, may be caused by a host of different firearms and bullets. For this reason, treatment of the latter will vary considerably from one patient to another, depending upon the wounding agents.

Wound Ballistics

The severity of any penetrating injury is related to both the tissue penetrated and the amount of energy delivered to the tissue. The formula for kinetic energy, $KE = \frac{1}{2} mv^2$, where m is the mass of the moving object and v is the velocity of that object, indicates that because the kinetic energy is directly related

¹ The editors have solicited two distinguished surgeons to review the physics of blunt and penetrating trauma. Rather than attempt to combine their perspectives and lose their critical individuality, the editors have elected to divide the chapter into two sections, at the risk of some redundancy. The first section is Dr. Litwin's: Dr. McSwain's section begins on p. 7.

2

to the square of the velocity, it is of considerably more importance in determining the amount of energy delivered by a bullet to the tissues than is the mass, or weight, of the bullet.

In general, it may be expected that a missile must deliver 59 ft-lb of kinetic energy to produce a casualty. Missiles of various masses must travel at varying velocities to develop this minimal amount of energy necessary to cause wounding (Table 1.1).

Velocity

Low-velocity bullets are those traveling less than 1200 feet per second (fps); medium velocity, 1200–2500 fps; and high velocity, over 2500 fps.

The *muzzle*, or initial, *velocity* of a bullet is its velocity on leaving the gun barrel; it varies from bullet to bullet (Table 1.2). A high muzzle velocity ensures an *impact velocity*, or velocity at the time the bullet strikes tissue, adequate to produce wounding. The *residual velocity* of a bullet is its velocity at the time it leaves the tissue. The mass of the bullet and difference between the impact and residual velocities are factors used in the formula to determine the kinetic energy expended in producing an injury.

An impact velocity of 125–170 fps is needed for penetration of human skin. Impact velocities of less than 200 fps will cause only minor injuries, unless body cavities are entered through more vulnerable areas, or apertures, such as the eye. With low-impact velocities, the injury produced is that due to the simple tissue penetration; such gunshot wounds are comparable to stab wounds. Wounds caused by missiles of medium velocity are more severe, with considerable tissue destruction and modest "explosive" effects. High-impact velocities cause maximal tissue destruction due to extreme explosive effects, while superhigh velocities can make very small missiles very deadly (Table 1.3) (8).

TABLE 1.1 Missile velocity and weight to deliver energy for wounding

Velocity (fps)	Weight (g)
500	104.0
1000	26.1
1500	11.6
2000	6.5
2500	4.2
3000	2.9
3500	2.1
4000	1.6

TABLE 1.2 Masses and approximate muzzle velocities of frequently used bullets

Cartridge (caliber)	Bullet mass (g)	Muzzle velocity (fps)
0.22 short and long	29	1000
0.22 long ^a	40	1150
5.45 mm (AK-74—Russian)	53	2950
5.56 mm (M-16) ^a	55	3250
0.25	50	820
0.270 Winchester ^a	150	3140
0.30 Mauser ^a	86	1420
0.30-30 Winchester ^a	170	2200
0.30-06°	150	2300
0.30-(M-1 Garand)	120	2800
(M-1-A-1 carbine)	110	1975
7.62 mm (M-14) ^a	150	2500
0.32	90	800
9 mm Luger	125	1150
0.357 magnum	158	1430
0.38	150	750
0.38 special	158	870
0.38 automatic	95	970
0.44 magnum	240	1470
0.45	250	860

a Rifle.

Momentum

Even though velocity is the most important factor in giving a bullet kinetic energy, mass is also of importance. It is the mass that gives a large bullet greater momentum, or increases its tendency to keep moving. This momentum increases a bullet's effectiveness as a wounding agent.

TABLE 1.3 Kinetic energy delivered by the same missile at varying velocities

Velocity (fps)	Energy (ft-lb)
500	55
1000	222
2000	887
3000	1996
4000	3549
5000	5545

4 Litwin and McSwain

Drag

The density of the tissue in which the bullet is traveling determines the "drag," or retardation, of a bullet of known mass. The greater the "drag" or retardation, the greater the amount of kinetic energy delivered by a missile to the tissues and, hence, the worse the wound.

Shape

The shape of a particle is also of importance in determining drag, both in air and tissue. Close-range shotgun wounds are particularly severe as multiple particles with maximal drag, because of their shape and a large powder charge, deliver all of their energy to the tissues. The needle, or cylinder, presented head-on is the shape of maximal mass and minimal presentation area. Air retardation during flight is minimized. It is for this reason that bullets are shaped as they are and launched point first. The rifling of the gun barrel imparts a high rate of spin, which then stabilizes the bullet along its flight course. The shapes of small arms missiles are 1) a flat base with a rounded nose; 2) a flat base with a pointed nose; and 3) a tapered or "boat-tail" base with a pointed nose. The shape of a bullet and the increased stability of flight in a "head-first" position, imparted by the rifling in the gun barrel, lead to the best possible ballistics characteristics.

Yaw

Because of the unequal distribution of mass due to the asymmetry of all bullets, they deviate more or less from a longitudinal axis during flight (yaw). Yaw increases in proportion to the density of the retarding medium (tissue) compared to air. On entering tissue, which is a denser medium than air, the degree of yaw is markedly magnified and the area of the bullet's presentation is immediately changed. Even though a bullet may enter the tissue point-on, it may immediately be tipped as much as 90 degrees or even more. Thus the amount of kinetic energy delivered to the tissue is materially increased and the severity of the wound further exaggerated. It is also for this reason that bullets that "tumble," as do those from "burp guns" without rifling, and those that deform on striking tissue, as do "hollow points," cause more severe injuries than those that do not have these characteristics (9, 10, 12).

Clinical Significance

Simple stab wounds and wounds caused by small-caliber, low-velocity bullets cause only localized injuries; both can be treated in much the same way. Even though a permanent tract is formed by wounding with low-energy missiles, it ultimately is smaller in diameter than the wounding missile because of tissue elasticity.

As a bullet of higher velocity or greater mass passes through the tissues, it also cuts a permanent cavity or tract. At the same time, kinetic energy is trans-

ferred to the tissues in front of and beside the missile. Tissue particles, or bone fragments, set in motion by the impact act as "secondary" missiles and deliver even more energy to the tissues.

As a result, shock waves ahead of and out from the missile tract impart considerable radial motion to the tissue elements, and a large temporary cavity is produced. This cavity may go through several pulsations, alternately expanding and contracting, probably as the result of tissue elasticity. This is the internal "explosive" effect produced by high-velocity bullets. Then, as a result of tissue elastic return, the skin contracts down over the points of entry and exit. At the same time, there is still expansion of the temporary cavity, and a vacuum is created. This negative pressure sucks foreign matter, such as clothing, into the depths of the wound. The surgeon should never be misled by small entry or exit wounds; these wounds frequently give no indication whatsoever of the severity or extent of the internal injury.

The wound tract left by a medium- or high-velocity bullet is surrounded by an area of damaged tissue and filled with extravasated blood and disrupted nerves. Multiple small vessels are ruptured, and areas of hemorrhage may extend widely along fascial planes through heavily damaged muscle fibers. Unless major veins or arteries are struck directly, they may appear to be uninjured. However, after injury with a high-velocity missile, major vessels should be closely inspected for intimal injury that occurs as the result of stretching during temporary cavitation.

Such concussion injury to a peripheral artery will frequently cause an intimal flap with thrombosis and distal ischemia. When any bullet, particularly one of high velocity, passes in close proximity to both an artery and a vein, an arteriovenous fistula may result, either immediately from simultaneous direct injury to adjoining areas of both structures, or later, after necrosis of injured areas on the walls of both vessels where they lie in juxtaposition. This latter phenomenon may occur as late as 2–4 weeks after the injury (19).

It is beyond the scope of this chapter to describe in detail the appropriate surgical treatment of penetrating injury to all vascular structures. However, it is important to emphasize that prompt arteriography must be used in the diagnosis of the site of injury of peripheral arteries and that severe injury may be present even in the presence of normal distal pulses. Additionally, gunshot wounds that simultaneously penetrate both a major artery or vein and any portion of the gastrointestinal tract, particularly the colon, are especially dangerous because of the contamination produced at the vascular repair site.

Blunt Trauma

Automobile accidents constitute the most common causes of blunt vascular injury. Such injuries may be due to either sudden deceleration or acute compression. Because of the differences in their mechanism of occurrence, injuries of each sort may be expected to occur in different anatomic areas.

Deceleration

When an automobile moving at a high rate of speed collides with a more slowly moving or stationary object, its occupants will continue to move directly forward or tangentially along a vector that is in proportion to the opposing or deflecting force, respectively. With head-on collisions, the body continues to move forward at the same rate of speed as that of the vehicle in which it is riding. This forward motion continues until the occupant collides directly with a stationary part of the vehicle, or some external object if ejection occurs.

When the chest of an occupant impacts directly with the steering column, contusion of the myocardium occurs as a result of the heart continuing forward and forcefully striking the sternum. Cardiac arrhythmias of varying severity result. When sudden death does not occur, healing may be prolonged in relation to the severity of the injury. The proximal aorta is also thrown violently forward and may be damaged either at its cardiac attachments or posteriorly where it passes from a mediastinal to a retropleural position. Such injuries are of a shearing nature. In proximal injuries, the coronary arteries or the atria may be directly lacerated; under these circumstances, death is usually immediate. Injury to the aorta, however, usually only results in intima and possibly media laceration. Widening of the mediastinal shadow on chest x-ray or inequality of pulses may be noted. Subsequently, an aortic aneurysm may develop or extensive aortic dissection may occur.

When forward motion of the body is extreme and its momentum great, the posterior thorax moves forward at the same time the sternum is being pushed backward. The heart is thus compressed between the sternum and thoracic spine; it may be directly ruptured by this mechanism. Abdominal compression may also cause transmission of fluid pressure waves through the aorta and vena cava, leading to either cardiac valve rupture or papillary muscle avulsion.

In sudden deceleration, the free-hanging organs within the abdomen continue to move forward. Severe stretch and shearing injuries to the renal vascular pedicle or blood vessels in the root of the mesentery may result. Acute stretching of arteries in either area in association with deceleration results, most often, in intimal laceration, even though the muscularis and adventitia remain intact.

When the renal arteries are damaged, thrombosis quickly occurs. Only by early arteriographic diagnosis and prompt primary repair, or renal replantation, can later renal transplantation be avoided.

The base of the vascular splenic pedicle may also be partially or totally avulsed by sudden deceleration. The site most susceptible to this sort of injury is at the entry of the vascular pedicle into the splenic substance. Deceleration injuries to the liver are usually lacerations along the ligamentum teres or separation of hepatic veins from either the liver or vena cava.

In severe neck injury associated with sudden deceleration, forcible stretching of the carotid artery with compression against the vertebral column may lead to intimal disruption and thrombosis. Even though rare, such injuries must be recognized immediately if a major stroke is to be avoided.