Modern Vascular Surgery

Volume 5

Modern Vascular Surgery

Volume 5

Edited by
John B. Chang

Springer-Verlag
New York Berlin Heidelberg London Paris
Tokyo Hong Kong Barcelona Budapest

John B. Chang, M.D., F.A.C.S.
Long Island Vascular Clinic
Roslyn, NY 11576 USA

With 194 Illustrations in 245 Parts

Library of Congress Cataloging-in-Publication Data

Modern vascular surgery, volume 5 / [edited by] John B. Chang.
p. cm.
Includes bibliographical references.

1. Blood-vessels—Surgery—Congresses. I. Chang, John B.
II. Title: Modern vascular surgery, volume five.
[DNLM: 1. Vascular Surgery—congresses. WG 170 M68953]
RD598.5.M6375 1992
617.4'13—dc20
DNLM/DLC
for Library of Congress 92-2307

Printed on acid-free paper.

Production coordinated by Chernow Editorial Services, Inc., and managed by Christin R. Ciresi; manufacturing supervised by Jacqui Ashri.
Typeset by Asco Trade Typesetting Ltd., Hong Kong

9 8 7 6 5 4 3 2 1

ISBN-13: 978-1-4612-7731-6 e-ISBN-13: 978-1-4612-2946-9
DOI: 10.1007/ 978-1-4612-2946-9

This book is dedicated to my dear mother, Jung Soon Chang, who has taught me so much about love and life. My mother has been an example of sincere graciousness; she has been a caring mother and was a supportive wife to my father, Bin Shik Chang, M.D. With my deepest and heartfelt appreciation, I dedicate this book to you, Mother, for all that you have given to me, with true love and affection, always,
From your little son,

John

Contents

Contributors

Bret C. Allen, MD, Department of Pathology, University of Mississippi Medical Center, Jackson, Mississippi, USA

Yaron Almagor, MD, National Institutes of Health, National Heart, Lung, and Blood Institute, Bethesda, Maryland, USA

Thomas Barnett, MD, Resident in General Surgery, Department of Surgery, The Lankenau Hospital, Philadelphia, Pennsylvania, USA

Antonio L. Bartorelli, MD, National Institutes of Health, National Heart, Lung, and Blood Institute, Bethesda, Maryland, USA

Richard M. Basile, MD, FACS, Assistant Professor in Surgery, Berkshire Medical Center, Pittsfield, Massachusetts, USA

Ramon Berguer, MD, PhD, Professor of Surgery, Chief, Division of Vascular Surgery, Wayne States University, and Harper Hospital, Detroit, Michigan, USA

Philippe G. Bull, MD, Second Department of Surgery, Wilhelminenspital, Vienna, Austria

Allan D. Callow, MD, PhD, Research Professor of Surgery, Department of Surgery, Washington University, St. Louis, Missouri, USA

Rudolph C. Camishion, MD, FACS, Professor of Surgery, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School at Camden, Head, Division of General Surgery, Cooper Hospital/University Medical Center, Camden, New Jersey, USA

J.D. Carmichael, Department of Surgery, Baptist Medical Center, Princeton, New Jersey, USA

Aurel C. Cernaianu, MD, FICS, Assistant Professor of Surgery, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School at Camden, Director of Research Programs, Division of Cardiothoracic Surgery, Cooper Hospital/University Medical Center, Camden, New Jersey, USA

Benjamin B. Chang, MD, FACS, Vascular Surgery Section, Albany Medical College, Albany, New York, USA

John B. Chang, MD, Associate Professor of Clinical Surgery, Albert Einstein College of Medicine, Bronx, New York, USA

Fuzhen Chen, MD, Vascular Surgery Unit, Zhongshan Hospital, Shanghai Medical University, Shanghai, PR China

Dolores F. Cikrit, MD, Indiana University Medical Center, Indianapolis, Indiana, USA

Jonathan H. Cilley, Jr., MD, FACS, Assistant Professor of Surgery, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School at Camden, Attending, Division of Cardiothoracic Surgery, Cooper Hospital/University Medical Center, Camden, New Jersey, USA

Jon R. Cohen, MD, Department of Surgery, Division of Vascular Surgery, Long Island Jewish Medical Center, New Hyde Park, New York; Associate Professor of Surgery, Albert Einstein College of Medicine, Bronx, New York, USA

Frank J. Criado, MD, FACS, Director, Maryland Vascular Institute at Union Memorial Hospital, Baltimore, Maryland, USA

Ronald L. Dalman, MD, Division of Vascular Surgery, Department of Surgery, The Oregon Health Sciences University, Portland, Oregon, USA. Present: Chief, Vascular Surgery Service Assistant Professor of Surgery, Palo Alto Veterans Administration Medical Center, Stanford University School of Medicine, Palo Alto, California, USA

Michael C. Dalsing, MD, Director, General Vascular Surgery and Associate Professor of Surgery, Indiana University Medical Center, Indianapolis, Indiana, USA

Anthony J. DelRossi, MD, FACS, Associate Professor of Surgery, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School at Camden, Chairman, Department of Surgery, Head, Division of Cardiothoracic Surgery, Cooper Hospital/University Medical Center, Camden, New Jersey, USA

Helmuth Denck, MD, First Department of Surgery, Krankenhaus Wien-Lainz, Vienna, Austria

Edward B. Diethrich, MD, Medical Director, Arizona Heart Institute and Foundation; Director and Chairman, Department of Cardiovascular Services; Director and Chief, Cardiovascular Surgery and Heart and Heart/Lung Transplantation, Humana Hospital-Phoenix, Phoenix, Arizona, USA

Wei Du, MD, Vascular Research, Tufts University, Boston, Massachusetts, USA

Karen O. Ehrman, MD, Department of Surgery and Radiology, Indiana University Medical Center, Indianapolis, Indiana, USA

Calvin B. Ernst, MD, Clinical Professor of Surgery, University of Michigan Medical School; Head, Division of Vascular Surgery, Henry Ford Hospital, Detroit, Michigan, USA

Kensuke Esato, MD, FACA, First Department of Surgery, Yamaguchi University School of Medicine, Ube, Yamaguchi, Japan

Youxian Feng, MD, Vascular Surgery Unit, Zhongshan Hospital, Shanghai Medical University, Shanghai, PR China

Frederick I. Field, MD, Division of Vascular Surgery, Loma Linda University Medical Center, Loma Linda, California, USA

Weiguo Fu, MD, Vascular Surgery Unit, Zhongshan Hospital, Shanghai Medical University, Shanghai, PR China

Kentaroh Fujioka, MD, First Department of Surgery, Yamaguchi University School of Medicine, Ube, Yamaguchi, Japan

H. Gabbert, Institute of Pathology, University Clinics of Mainz, Mainz, Germany

T.A. Gaskin, Department of Surgery, Baptist Medical Center, Princeton, New Jersey

Marvin L. Gliedman, MD, Professor of Surgery, Albert Einstein College of Medicine of Yeshiva University, Montefiore Medical Center, New York, New York, USA

John A. Griswold, MD, Department of Surgery, Division of Trauma and Critical Care, University of Mississippi Medical Center, Jackson, Mississippi, USA

Helmuth Gruber, MD, Department of Anatomy, University of Vienna Medical School, Vienna, Austria

Zhuo-yun Gu, Department of Surgery, Chinese PLA General Hospital, Beijing, China

Robert Guidoin, PhD, Department of Experimental Surgery, Université Laval, Quebec, Canada

Sushil K. Gupta, MD, Division of Vascular Surgery, Montefiore Medical Center/Albert Einstein College of Medicine, Bronx, New York, USA

Yoshiyuki Haga, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

U. Hake, Division of Cardiothoracic and Vascular Surgery, University Clinics of Mainz, Mainz, Germany

D.P. Halpin, Department of Surgery, Baptist Medical Center, Princeton, New Jersey, USA

Nobuo Hatori, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

Larry H. Hollier, MD, FACS, FACC, Chairman, Department of Surgery, Ochsner Clinic and Alton Ochsner Medical Foundation; Clinical Professor of Surgery, Louisiana State University and Tulane University Medical Center, New Orleans, Louisiana, USA

Anthony M. Imparato, MD, Professor of Surgery, New York University Medical Center, New York, USA

J.H. Isobe, Department of Surgery, Baptist Medical Center, Princeton, New Jersey, USA

S. Iversen, Division of Cardiothoracic and Vascular Surgery, University Clinics of Mainz, Mainz, Germany

H. Jakob, Division of Cardiothoracic and Vascular Surgery, University Clinics of Mainz, Mainz, Germany

T.R. Kahn, Department of Surgery, Baptist Medical Center, Princeton, New Jersey, USA

Ronald N. Kaleya, MD, Assistant Professor of Surgery, Albert Einstein College of Medicine of Yeshiva University, Montefiore Medical Center, New York, New York, USA

Richard F. Kempczinski, MD, Professor of Surgery, Chief, Vascular Surgery, University of Cincinnati Medical Center, Department of Surgery, Division of Vascular Surgery, Cincinnati, Ohio, USA

Wolff M. Kirsch, MD, Professor of Neurological Surgery, Loma Linda University School of Medicine, Department of Surgery, Division of Neurosurgery, Loma Linda, California, USA

Harry B. Kram, MD, Division of Vascular Surgery, Montefiore Medical Center/Albert Einstein College of Medicine, Bronx, New York, USA

Takayuki Kuga, MD, First Department of Surgery, Yamaguchi University School of Medicine, Ube, Yamaguchi, Japan

Stephen G. Lalka, MD, Department of Surgery and Radiology, Indiana University Medical Center, Indianapolis, Indiana, USA

Robert P. Leather, MD, Chief, Division of General Surgery, Department of Surgery, Vascular Surgery Section; Professor of Surgery, Albany Medical College, Albany, New York, USA

Martin B. Leon, MD, National Institutes of Health, National Heart, Lung, and Blood Institute, Bethesda, Maryland, USA

Guangdi Li, MD, Beijing Heart Lung Blood Vessel Medical Center-An Zhen Hospital, Beijing, China

Anita K. Lindsey, MD, Department of Surgery, University of Mississippi Medical Center, Jackson, Mississippi, USA

Frank W. LoGerfo, MD, Associate Professor of Surgery, Harvard Medical School; Chief, Division of Vascular Surgery, Harvard-Deaconess Surgical Service, New England Deaconess Hospital, Boston, Massachusetts, USA

J.L. Mathews, Department of Surgery, Baptist Medical Center, Princeton, New Jersey, USA

Jose Mena, MD, Department of Surgery, Ochsner Clinic and Alton Ochsner Medical Foundation, New Orleans, Louisiana, USA

Atsuhiro Mitsumaru, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

Peter Neglèn, MD, Visiting Assistant Professor of Surgery, University of Mississippi Medical Center, Jackson, Mississippi, USA

Richard F. Neville, MD, National Institutes of Health, National Heart, Lung, and Blood Institute, Bethesda, Maryland; Section of Vascular Surgery, UMDNJ-New Jersey Medical School, Newark, New Jersey, USA

H. Oelert, Division of Cardiothoracic and Vascular Surgery, University Clinics of Mainz, Mainz, Germany

Masaki O-hara, MD, First Department of Surgery, Yamaguchi University School of Medicine, Ube, Yamaguchi, Japan

Eriya Okuda, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

Miralem Pasic, MD, Clinic for Cardiovascular Surgery, University Hospital Zurich, Zurich, Switzerland

L. Beaty Pemberton, MD, FACS, Professor and Chief, Department of Surgery, Truman Medical Center, St. Louis, Missouri, USA

Michael Perlman, BS, National Institutes of Health, National Heart, Lung, and Blood Institute, Bethesda, Maryland, USA

Frank B. Pomposelli, Jr, MD, Assistant Professor of Surgery, Harvard Medical School, Division of Vascular Surgery, Harvard-Deaconess Surgical Service, New England Deaconess Hospital, Boston, Massachusetts, USA

John M. Porter, MD, Professor of Surgery, Division of Vascular Surgery, Department of Surgery, The Oregon Health Sciences University, The Oregon Health Sciences University School of Medicine, Portland, Oregon, USA

Liqun Pu, MD, CVT Lab, McGill University, Montreal, Canada

Arshad Quadri, MD, Resident in Surgery, Berkshire Medical Center, Pittsfield, Massachusetts, USA

Seshadri Raju, MD, Professor of Surgery, University of Mississippi Medical Center, Jackson, Mississippi, USA

James Reilly, MD, Resident in General Surgery, Department of Surgery, The Lankenau Hospital, Philadelphia, Pennsylvania, USA

Robert S. Rhodes, MD, Department of Surgery, University of Mississippi Medical Center, Jackson, Mississippi, USA

Calvin A. Ridgeway, MD, Chief Resident, Department of Surgery, Truman Medical Center, St. Louis, Missouri, USA

S. Riggins, Department of Surgery, Baptist Medical Center, Princeton, New Jersey, USA

Parviz Sadhigi, MD, FACS, Professor in Surgery, Program Director, Berkshire Medical Center, Pittsfield, Massachusetts, USA

Alan P. Sawchuk, MD, Indiana University Medical Center, Indianapolis, Indiana, USA

Steven P. Schmidt, MD, Division of Vascular Surgery, Loma Linda University Medical Center, Loma Linda, California, USA

W. Schmiedt, Division of Cardiothoracic and Vascular Surgery, University Clinics of Mainz, Mainz, Germany

Dhiraj M. Shah, MD, Vascular Surgery Section, Albany Medical College, Albany, New York, USA

Masafumi Shimizu, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

Richard K. Spence, MD, FACS, Associate Professor of Surgery, University of Medicine and Dentistry of New Jersey, Robert Wood Johnson Medical School at Camden, Head, Division of Vascular Surgery, Cooper Hospital/University Medical Center, Camden, New Jersey, USA

D.E. Strandness, Jr, MD, Professor, Department of Surgery, University of Washington School of Medicine, Seattle, Washington, USA

Hiroaki Takenaka, MD, First Department of Surgery, Yamaguchi University School of Medicine, Ube, Yamaguchi, Japan

Susumu Tanaka, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

Lloyd M. Taylor, MD, Division of Vascular Surgery, Department of Surgery, The Oregon Health Sciences University, Portland, Oregon, USA

David F.J. Tollefson, MD, Senior Vascular Surgery Fellow, Henry Ford Hospital, Detroit, Michigan, USA

Yozo Uriuda, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

Frank J. Veith, MD, Chief, Vascular Surgical Services, Montefiore Medical Center; Professor of Surgery, Albert Einstein College of Medicine, New York, New York, USA

Renu Virmani, MD, Armed Forces Institute of Pathology, Washington, DC, USA

Dajie Wang, MD, Department of Surgery, University of Virginia, Charlottesville, Virginia, USA

Yuqi Wang, MD, MS, Vascular Surgery Unit, Zhongshan Hospital, Shanghai Medical University, Shanghai, PR China

Zhonggao Wang, MD, FICA, Professor and Chief, Department of Vascular Surgery, Beijing Heart Lung Blood Vessel Medical Center-An Zhen Hospital, Beijing, China

Rodney A. White, MD, Chief, Vascular Surgery, Harbor-UCLA Medical Center; Associate Professor of Surgery, UCLA School of Medicine, Torrance, California, USA

Anthony D. Whittemore, MD, Chief, Division of Vascular Surgery, Brigham and Women's Hospital; Associate Professor of Surgery, Harvard Medical School, Boston, Massachusetts, USA

Robert D. Williams, MD, FACS, Cardiovascular and Thoracic Surgeon, Department of Surgery, Truman Medical Center, St. Louis, Missouri, USA

Jidong Wu, MD, Beijing Heart Lung Blood Vessel Medical Center-An Zhen Hospital, Beijing, China

Xiuling Yao, Vascular Surgery Unit, Zhongshan Hospital, Shanghai Medical University, Shanghai, PR China

Jianrong Ye, MD, MS, Vascular Surgery Unit, Zhongshan Hospital, Shanghai Medical University, Shanghai, PR China

Hiroshi Yoshizu, MD, Department of Surgery II, National Defense Medical College, Tokorozawa, Saitama, Japan

Jian Yu, MD, Beijing Heart Lung Blood Vessel Medical Center-An Zhen Hospital, Beijing, China

Keuk Yum, MD, Clinical Assistant Professor, Thomas Jefferson University; Department of Surgery, The Lankenau Hospital, Philadelphia, Pennsylvania, USA

Nobuya Zempo, MD, First Department of Surgery, Yamaguchi University School of Medicine, Ube, Yamaguchi, Japan

Hai Zhang, MD, Transplantation and Vascular Surgery, Johns Hopkins University, Baltimore, Maryland, USA

Kuo-hua Zhang, Department of Surgery, Chinese PLA General Hospital, Beijing, China

Yong Hua Zhu, MD, Assistant Research Professor, Loma Linda University School of Medicine, Department of Surgery, Division of Neurosurgery, Loma Linda, California, USA

Introduction

This book is based on the research and clinical work presented at the Fifth International Symposium, Vascular Surgery, 1991. Since the inception of the first symposium in May 1983, the past symposia have been extremely successful in gathering outstanding scholars and scientists from around the world to an academic podium. During the 1991 symposium, we had outstanding young investigators, who participated with other renowned clinicians and researchers.

It was my honor to present an Outstanding Vascular Surgeon's Award to my long-time friend and pioneer in the field of vascular surgery, Dr. Anthony M. Imparato. In the past, Dr. H.H.G. Eastcott, Dr. Michael E. DeBakey and Dr. D. Emerick Szilagyi have been recipients of this award.

To make these academic endeavors successful, I have been fortunate to be associated with many wonderful individuals at my institution, the Long Island Jewish Medical Center. I have a deep sense of appreciation of Dr. Robert K. Match, President, Dr. Seymour Cohen, Vice President for Education and Research, and Dr. Leslie Wise, Chairman, Department of Surgery, for the sincere support and loyal friendship they have given me through the years.

My special thanks to Ms. Ann J. Boehme and the staff of the Office for Continuing Education, Long Island Jewish Medical Center, for their support and outstanding effort in preparation of this major event, the Fifth International Vascular Symposium.

I extend my appreciation to the publishing company and editorial members who have performed an excellent job assembling the manuscripts into textbook style.

To my staff at the Long Island Vascular Center: I thank you for your continuing efforts to support my academic endeavors. To my executive secretary, Ms. Eileen Moran Zanini, I sincerely appreciate your untiring effort to accomplish this difficult task of editing.

To my wonderful sons, Victor and Robert: I am very fortunate to be your father. You will always be a joy to me.

My eternal gratitude and love goes to my lifetime friend and wife, Lucy Chang, M.D. I thank you for the love, understanding and unselfish support you continue to give me.

About the Editor

John B. Chang, M.D., F.A.C.S.

Former and Founding Director
Division of Vascular Surgery
Department of Surgery
Long Island Jewish Medical Center
New Hyde Park, New York

Consultant
Department of Surgery
Long Island Jewish Medical Center
New Hyde Park, New York

Chairman
Department of Surgery
Deepdale General Hospital
Little Neck, New York

Director
Long Island Vascular Center
New Hyde Park, New York

Associate Professor of Clinical Surgery
Albert Einstein College of Medicine
Bronx, New York

1
Current State of Noninvasive Vascular Study: Carotid Artery

D.E. STRANDNESS, JR.

When ultrasonic duplex scanning was introduced in 1974, the site to which it was first applied was the carotid artery.[1] This was due to the fact that the artery is close to the skin, is frequently involved by atherosclerosis, and commonly studied by arteriography. It was the ideal testing ground for the feasibility of this new technology. The application involved development of velocity criteria for grading the degree of stenosis and its verification by arteriography.[2] This process produced several interesting results that are of great importance and include the following: (1) stenoses and occlusions could be detected and graded with a high sensitivity and satisfactory positive predictive value; (2) arteriography, while considered to be the gold standard, was shown to be less than perfect in documenting the degree of involvement; (3) ultrasonic duplex scanning was usable by technologists once they became familiar with imaging and Doppler methods; (4) the test was sufficiently sensitive and specific to permit its use for a large and diverse group of patients.

The patients who deserve study by duplex scanning include the following:

1. The asymptomatic patient found to have a cervical bruit.
2. Patients with transient ischemic attacks and strokes.
3. Patients who are to undergo major cardiac and noncardiac operative procedures and found to have a bruit in the neck.
4. Patients who present with symptoms and/or signs of vertebro-basilar insufficiency.
5. Patients who have undergone carotid endarterectomy and present with new symptoms and/or signs.
6. Patients with documented carotid artery atherosclerosis who are being assessed for progression of disease.

Diagnostic Criteria

The evolution of duplex scanning to its current status required the development of velocity criteria for normal carotid bifurcation and when it became involved by atherosclerosis. It is important to keep in mind that the carotid bulb is a very unique bifurcation. It is the only place in the arterial system where there is a dilatation at a branch point such as the carotid bifurcation. This is important for two reasons: (1) it produces unusual flow patterns that must be recognized since they can be misinterpreted as abnormal; and (2) it is in the postero-lateral aspect of the bulb where atherosclerosis first develops.

As flow enters the carotid bulb into the dilated region, flow will be very complex with an area of recirculation that develops in the postero-lateral aspect.[3] Flow in this region will actually reverse. This is referred to as the region of boundary layer separation. Flow is antegrade near the medial wall and the flow divider. The area of flow separation will change in size during each heart cycle. These complex flow patterns must be recognized as a normally occurring phenomenon.

The flow patterns in the bulb can be recognized in two ways. If the sample volume of the pulsed Doppler is placed near the flow divider, flow will be antegrade during the entire pulse cycle. However, when the sample volume is moved a few millimeters into the zone of boundary layer separation, the forward-reverse flow patterns can easily be discerned. For the experienced technologist, this is not difficult to document. With the availability of color, this can be more easily demonstrated. Since the convention is to have flow in arteries be depicted in red and veins in blue, the flow during systole near the flow divider will be red, but in the separated zone it will be blue. When these flow patterns are observed, it is sufficient to rule out the presence of atherosclerosis in the bulb. This is very important since the presence of bounding layer separation in the bulb of patients who present with ischemic events essentially rules out this region as a cause of the problem.

When atherosclerosis develops, it will start in the bulb. If it remains confined to that region, it is usually benign since it is usually a fibrous plaque with a smooth intact covering. However, when the plaque progresses the lesion undergoes degenerative changes to become a complicated lesion. These advanced lesions can lead to the serious complications that we are familiar with and include transient ischemic events, stroke, and total occlusion of the internal carotid artery.

When the disease develops in the bulb, the unusual flow patterns associated with boundary layer separation will not be observed. This is a sign that the bulb is occupied by atheroma. If the lesion does not extend beyond the limits of the bulb, flow to the brain via the internal carotid artery is not impaired, and there will be very little to no flow disturbances produced.

These lesions are rarely of clinical significance since it is unusual for this degree of involvement to lead to a clinical event.

When the plaque begins to extend beyond the limits of the bulb and impinge on the region where the antegrade flow to internal carotid artery normally occurs, flow disturbances will be produced and recognized by the presence of spectral broadening.[4] If the peak systolic frequency recorded does not exceed 4 MHz (using a 5 MHz pulsed Doppler at a 60 degree angle), the lesion is classified as a 16–49% stenosis. If one is using angle adjusted velocity as the parameter, the frequency shift would be equivalent to 125 cms/sec.

When the plaque progresses further, producing increased narrowing, the peak systolic velocity will correspondingly increase above the 4 MHz frequency shift and the 125 cms/sec velocity value.[4] The stenosis will then be classified into a 50–79% category. With this degree of narrowing the end-diastolic frequency remains below 4.5 KHz. This would correspond to 145 cms/sec when angle adjusted velocity is used. This is considered to be a pressure and flow reducing lesion but is not generally considered to be a pre-occlusive lesion.

With a continued enlargement in the size of the plaque, there is a further increase in both the systolic and end-diastolic velocity.[5] For the 80–99% stenosis category, the major criterion used is the end-diastolic velocity which exceeds 4.5 KHz or 145 cms/sec in absolute terms. These lesions are often referred to as pre-occlusive since they frequently lead to total occlusion.

The totally occluded internal carotid artery is relatively easy to detect if simple rules are followed during the course of the examination. Normally, 70–80% of the flow in the common carotid goes to the brain, which is a low resistance organ. As a result, the end-diastolic velocity in the common carotid artery will normally be above zero. When the internal carotid artery becomes occluded, the resistance now "seen" by flow in the common carotid artery is much higher, and end-diastolic flow will now go to zero. This should be the first hint that the internal carotid artery may be totally occluded. Further confirmation can be obtained by the failure to detect any flow in the internal carotid artery. Color may be of some assistance here but it must be remembered that the residual channel in a very tight stenosis may be difficult to find and demonstrate. It is important for the examiner to look beyond the bulb to determine if any flow at all is present. Other indicators that can be of value include an apparent increase in flow in the contralateral carotid artery. This occurs because this artery is now serving as the major supplier of blood to both hemispheres.

When the results of duplex scanning have been compared against arteriography, the sensitivity has consistently been above 90%. The specificity in the earliest studies was as low as 37%.[6] This was due to the fact that the unusual flow patterns associated with boundary layer separation were

not recognized as being normal. Once we determined the basis of these flow changes, the specificity improved so that at the present time it is also greater than 90%. This level of sensitivity and specificity makes duplex scanning a good method for studying populations with a low prevalence of disease as well as one where the prevalence is expected to be quite high.

Role of Color Doppler

The latest innovation in the field of duplex scanning has been the addition of color to the systems. Color has appeal since it permits the observer to immediately assess the location of the vessels of interest. Another advantage is that variations in the anatomy of the vessels and their position relative to each other will be immediately evident. For example, coils and kinks may be readily seen, which makes the interpretation of the velocity changes more certain. In the current systems a change in velocity is noted by a change in the hue of the color. By convention, it is also possible to determine the direction of flow since flow in one direction is assigned one color while the reverse flow component is assigned another. It should not be unexpected that arterial flow would be presented in red with venous flow in blue. However, one must be careful in interpreting color changes, since any change in the angle of incidence of the sound beam with the vessel will also result in a frequency shift change that does not reflect a true change in velocity. This can be subtle and the observer must be aware of this potential problem. It is also known that aliasing can occur with color as it can with standard pulsed Doppler technique when the Nyquist limit has been exceeded.

Our current use of color in the carotid artery is to use it as the road map for the vessels but to continue to use the standard velocity criteria for making a diagnosis of the degree of stenosis as noted above. Until such time that precise color criteria are developed to replace the standard method, we strongly urge users to stick with the time-proven approaches that have been validated against contrast arteriography.

Applications in Practice

The theoretical advantage of an excellent screening test is that it might be used to identify those patients who are at risk for developing an ischemic cerebrovascular episode. Hopefully, therapy could then be applied to prevent such an event from occurring. The most controversial patients are those who are asymptomatic but are found to have a bruit in their neck. What does one do with these patients? Should one be aggressive or simply ignore the finding? If one adopts an aggressive stance, the future course of

events depends entirely upon the extent of involvement in one or both carotid arteries. If duplex scanning were not available, the only method of determining the degree of involvement with certainty would be arteriography. This would result in a large number of unnecessary studies being performed, since at least 90% of those performed would lead to the discovery of lesions that should be treated conservatively. The only lesions that deserve careful consideration for operation in asymptomatic patients are those with a >80% diameter reducing stenosis.[7] When these are found there is evidence that progression to total occlusion is common within the first few months after discovery of the lesion. If the internal carotid artery does occlude, the chance of the patient sustaining a stroke is in the range of 25%.

Plaques that narrow the artery by less than 80% are safe to follow because the event rate is small, being about 4% per year.[7] The one situation that remains worrisome is the patient with an occlusion of one internal carotid artery and a 50–79% stenosis of the other carotid artery. The problem here is that the natural history of this combination of lesions is poorly understood. In some cases, it might be preferable to proceed directly with carotid endarterectomy to preserve the patency of the internal carotid artery. On the other hand, if a conservative course is followed, I would strongly recommend that repeat duplex scans be done every three months to determine stability of the lesion. If progression to an 80–99% stenosis occurs, the patient should be considered a candidate for prophylactic carotid endarterectomy.

For plaques that narrow the internal carotid artery by <50%, it is very safe to simply follow the patients. An unanswered question relates to how often these patients should be followed with repeat duplex scans. My practice at the moment is to do a repeat study every 12 months. This would appear to be cost-effective and safe in terms of a progression rate from a <50% to a >50% stenosis, which is 8% per year.

For symptomatic patients, the need for duplex scanning is often raised, since many of these patients will likely undergo arteriography anyway. Duplex scanning can be useful for the following reasons: (1) it provides useful information for the neuroradiologist in planning the study (for example, if very little disease is found on the duplex scan, it may be helpful to obtain multiple views of the bulb and internal carotid artery for better visualization); (2) the duplex scan will provide the necessary baseline data for follow-up studies, since it is clear that arteriography cannot be used for such purposes; and (3) the duplex study may provide enough information to forestall the need for arteriography. For example, if the internal carotid artery were found to be occluded, there would be no need to proceed with the contrast study. However, if there is any question at all about the status of the internal carotid artery in terms of patency, it is important to verify its status with the contrast study.

The role of carotid endarterectomy has been a source of great concern and controversy.[8] While numerous surgical series have been published showing that the operation can be performed with a perioperative stroke and mortality rate that is under 5%, this has not been accepted by many in the neurological community.[9] This is due to the fact that some community experiences and the report of the RAND Corporation have shown that the perioperative morbidity and mortality rates were too high, making the role of the operation more suspect as a method of preventing stroke. In addition, since only historical controls were available, the possible benefit of the operation was uncertain.

This skepticism led to the mounting of several randomized clinical trials, which included the Veterans Administration Asymptomatic trial (VAAT), the North American Symptomatic Carotid Endarterectomy trial (NASCET), and the trial with asymptomatic patients (ACAS). The VAAT trial has completed all phases except data analysis and follow-up, scheduled for completion in the spring of 1991.

The NASCET trial has been under way for three years. Recently, it was determined by interim analysis that the subset of symptomatic patients with a 70% diameter reducing stenosis or greater had a much better prognosis when treated by operation. In fact, this part of the trial has now been terminated and has led to the distribution of a national medical alert by the NIH announcing this finding.[10] This will have a profound effect in this country and has special importance to those who use duplex scanning as a method of screening symptomatic patients.

There are two aspects of the trial findings that can be usefully applied in the practice of stroke prevention. If a patient is symptomatic with a TIA or stroke, the finding of a high grade stenosis on duplex scanning will require arteriography and subsequent carotid endarterectomy. On the other hand, the finding of a lesser lesion will in all likelihood be followed by treatment with aspirin until such time that operation has been proven to be of greater benefit in this subset of patients. It is unlikely that contrast arteriography will be required in this group if the accuracy of the testing falls into the acceptable range as noted above.

There are other implications for the group with lesions that are less than 70%. This group should be followed, since if they progress to a greater degree of stenosis, then operation would appear to be justified. This will require repeat duplex scans to be done since arteriography cannot and should not be used on a repetitive basis to make this determination. The follow-up interval remains to be determined, but based upon the initial findings and the degree of narrowing, it is possible to make some tentative recommendations. For lesions narrowing the artery by >50%, there should probably be a restudy at least every six months to assess the stability of the lesion. For lesions less than 50%, it is probably safe to restudy the patients every 12 months. However, if the patient remains symptomatic or has recurrent events on aspirin therapy, one should adopt an aggressive

stance with regard to use of endarterectomy to remove the focus of the problem.

Another group of patients that have been the subject of some controversy are those who are to undergo major cardiac or noncardiac surgery and who are noted to have a bruit in their neck.[11] These patients are a problem because of the perceived increase in risk of a stroke when they undergo their major operative procedure. The studies done on these patients would suggest that they are not at greater risk for a stroke and that their primary reason for operation should be taken care of first, with one exception, and that is when the patient is also symptomatic from their carotid artery atherosclerosis.

In the case of coronary artery disease, the greatest risk for life is with the coronary arteries and not the carotid arteries. Thus, even if one were aggressive in treating a carotid lesion in this setting, it would be important to still treat the heart disease first and the carotid artery at a later date. Thus, a preoperative duplex scan is helpful since it will provide the necessary information upon which to plan the course of therapy. It must be remembered that there is no evidence that the coronary artery bypass grafting has an adverse effect on carotid artery atherosclerosis that could or should increase the risk of stroke. When stroke occurs in the setting of coronary bypass surgery, it is likely to be secondary to emboli that develop during the clamping of the aorta for placement of the vein grafts.

One area which remains very difficult to evaluate with duplex scanning is vertebrobasilar insufficiency. While there is no doubt that subclavian stenosis and/or occlusion can be detected with a high degree of certainty along with reverse flow in the vertebral artery, this is commonly observed in patients who are free of symptoms.[12] It is also difficult to determine with a high degree of certainty whether the symptoms that occur are in fact due to ischemia of the posterior fossa.

References

1. Barber FE, Baker DW, Nation AWC, et al. Ultrasonic duplex echo Doppler scanner. IEEE Trans Biomed Engin 1974;21:109–113.
2. Langlois YE, Roederer GO, Strandness DE Jr. Vascular ultrasound. Ultrasonic evaluation of the carotid bifurcation. Echocardiography 1987;4.141–159.
3. Phillips DJ, Greene FM, Langlois YE, et al. Flow velocity patterns in the carotid bifurcations of young presumed normals. Ultrasound Med Bio 1983, 9:39–49.
4. Langlois YE, Roederer GO, Chan AW, et al. Evaluation of carotid artery disease. The concordance between pulsed Doppler/spectrum analysis and angiography. Ultrasound Med Biol 1983,9:51–63.
5. Roederer GO, Langlois YE, Jager KA, et al. A simple spectral parameter for accurate classification of severe carotid disease. Bruit 1984,3:174–178.
6. Fell G, Phillips DJ, Chikos PM, et al. Ultrasonic duplex scanning for disease of the carotid artery. Circulation 1981;64:1191–1194.

7. Roederer GO, Langlois YE, Jager KA, et al. The natural history of carotid arterial disease in asymptomatic patients with cervical bruits. Stroke 1984;15:605–613.
8. Barnett HJM, Plum F, Walton JN. Carotid endarterectomy. An expression of concern. Stroke 1984;15:948–950.
9. Healy DA, Clowes AW, Zierler RE, et al. Immediate and long-term results of carotid endarterectomy. Stroke 1989;20:1138–1142.
10. Clinical Alert. Benefit of carotid endarterectomy for patients with high-grade stenosis of the internal carotid artery. Issued by the National Institute of Neurological Disorders and Stroke, February, 1991.
11. Ivey TD, Strandness DE Jr, Williams DB, et al. Management of patients with carotid bruit undergoing cardiopulmonary bypass. J Thorac Cardiovasc Surg 1984;87:183–189.
12. Bornstein NM, Norris JW. Subclavian steal. A harmless haemodynamic phenomenon? Lancet 1986;2:303–305.

2
Current Status of Noninvasive Studies for Extremity Arterial and Venous Disease

RICHARD F. KEMPCZINSKI

Despite more than 30 years of clinical experience, noninvasive testing of patients with extremity arterial disease remains controversial. Some have suggested that noninvasive testing may be no more accurate than an experienced clinician in diagnosing arterial occlusive disease and therefore is an unnecessary addition to the already high cost of patient care and a disincentive to the development of clinical acumen by surgical trainees.[1]

Although an experienced examiner can usually document the presence of extremity arterial occlusion, clinical examination alone may be misleading in certain situations; e.g., when there is concomitant neuropathy.[2] Furthermore, since many patients with extremity pains are initially seen by physicians who may be inexperienced in managing vascular disease, the noninvasive laboratory can provide objective confirmation of their clinical impression and assure prompt and appropriate triage of such patients. In much the same way that measurement of brachial blood pressure has become invaluable in the detection and management of hypertension, measurement of lower extremity systolic pressures provides the clinician with objective documentation of the regional hypotension that is the hallmark of arterial occlusive disease. Certainly, few would question the importance of noninvasive testing in the diagnosis of extremity deep venous thrombosis, an area where clinical diagnosis is notoriously inaccurate.

Apart from merely documenting the presence of vascular disease, noninvasive testing is useful in relating an anatomical lesion to its resulting functional derangement. This is especially important in patients with diabetes mellitus where hemodynamic data are critical in categorizing the severity of resting extremity symptoms. It may also be valuable when there are multiple, in-series occlusions whose relative hemodynamic significance must be assessed to plan appropriate arterial reconstruction.

Noninvasive tests may also be used to help focus subsequent angiography. Knowing in advance the location of hemodynamically significant lesions, the radiologist can alter his planned approach to better visualize the areas of clinical interest. By contrast, failure to demonstrate a lesion

consistent with the patient's symptoms may avoid unnecessary arteriograms.[3]

Patients can also be followed more carefully in the vascular laboratory than is possible with clinical examination alone. The results of nonoperative therapy can thus be objectively documented and potential disease progression detected even before the patient appreciates clinical deterioration. Furthermore, since distal pulses may be absent even after a successful proximal reconstruction, and incisional pain or coexistent neuropathy may create a confusing clinical picture, the need for hemodynamic parameters is especially important in the perioperative assessment of patients.

Extremity Arterial Disease

Although there is no consensus regarding which procedures are most appropriate for the evaluation of extremity arterial insufficiency, a few generalizations appear warranted. As in other areas, a combination of tests is often more sensitive than any single test used alone, and therefore most laboratories generally use at least two of the procedures described below. However, when a single test clearly establishes the diagnosis, additional tests in such cases are redundant and decrease specificity. The practice of routinely performing a battery of tests, regardless of the clinical situation, is not cost effective and should be condemned.

In addition, noninvasive tests may be classified as *Indirect* if they identify the presence of disease by detecting some physiologic or hemodynamic alteration that is the result of the arterial lesion, or *Direct* if they interrogate the lesion itself, characterizing its extent and appearance. Over the last several years, there has been a general movement away from indirect tests with a proportionly greater reliance on direct studies.

As credentialing agencies have begun to look at the Noninvasive Vascular Diagnostic Laboratory, they have identified certain *Primary* test, which they expect to be available in any laboratory wishing to study patients with certain type of vascular disease, and *Secondary* tests, which might add important physiologic information to the assessment but which, by themselves, are not sufficiently accurate to establish the diagnosis.

Finally, since chronic arterial occlusions may be very well compensated under resting conditions, persons with exercise-related complaints should usually undergo some form of stress testing to confirm and accurately quantitate the extent of their disability.

Doppler Arterial Survey

By surveying an accessible peripheral artery with a simple, hand-held, continuous wave Doppler velocity meter, an experienced examiner can often derive a surprising amount of useful information. Although a multiphasic

Doppler signal in a distal vessel strongly suggests that the proximal arterial tree is unobstructed, such information is very qualitative and quite subjective. However, the presence of such a signal at the ankle in a patient with equivocal symptoms often prompts us to shorten our formal extremity evaluation to a simple measurement of the ankle/brachial index (ABI). Furthermore, the presence of an audible Doppler signal in any vessel *de facto* confirms its patency and should warrant an aggressive, radiologic effort to document its presence even when it may not have been seen on routine angiography. Although this technique is direct, its subjectivity make it a secondary procedure that should never be the sole basis for diagnosis.

Segmental Limb Pressures

The measurement of multilevel, segmental, systolic pressures is probably the most generally accepted and widely applied technique for documenting extremity arterial occlusive disease. Although indirect, it is simple, reproducible, inexpensive, and well accepted by patients. More data have been reported using this technique than virtually any other in the vascular diagnostic armamentarium, yet it is subject to numerous shortcomings.

Although it is usually accurate in establishing the presence of arterial occlusion, it is much less satisfactory for localizing the responsible lesions. Aortoiliac disease is particularly difficult to identify and quantitate in the presence of concomitant superficial femoral artery occlusion. Even when separate, narrow, high and low thigh cuffs are used, the location of such multisegmental lesions is, at best, correctly predicted in only 78% of the extremities studied.[4] Brener et al. found that 55% of patients with angiographically proved aortoiliac stenosis had normal proximal thigh indices, while 31% of those with occlusion of the superficial femoral artery, but no iliac disease, had a false positive thigh pressure, thus prompting them to conclude that segmental limb pressures (SLP) were incapable of accurately localizing lower extremity arterial occlusive disease.[5] In a similar study, Flanigan et al. compared direct intra-arterial measurement of femoral artery pressure to that obtained using wide and narrow thigh blood pressure cuffs. They found that overall accuracy in diagnosing aortoiliac disease was poor for both the wide (52%) and narrow (73%) cuff technique.[6] More recently, Lynch et al. documented a 97% sensitivity to aortoiliac disease and an 89% sensitivity to isolated femoropopliteal disease. However, in the presence of combined disease, the specificity to aortoiliac disease decreased from 70% to 41% and the sensitivity to femoropopliteal disease fell from 89% to 55%.[7]

The distal monitoring site also influences the accuracy of thigh SLP when concomitant arterial occlusive disease is present below the groin. In 23% of patients with such multilevel disease, the upper thigh pressure was more than 15 mm Hg lower when a pedal artery was used to monitor return of

TABLE 2.1 Correlation between ankle systolic pressure (SLP), ankle/brachial index (ABI) and clinical severity of extremity ischemia.

	SLP (mm Hg)	ABI
Claudication	70–100	0.5–0.8
Rest pain	<50	0.3
Gangrene	0–30	<0.2

blood flow compared to a similar measurement using the popliteal artery.[8] Furthermore, in patients with diabetes mellitus or chronic renal failure, the vessels may be so heavily calcified that they are virtually incompressible, thus making the measurement of SLP meaningless.[9] Measurement of *toe pressures* in such patients may occasionally circumvent this problem. Even when measurements of ankle pressure can be made, there is significant variability between consecutive measurements in the same patient. Baker and Dix found a mean variation in the ABI of 0.18 and suggested that multiple baseline determinations should be obtained and that the index must change at least 0.15 before it can be considered significant.[10]

Because of these limitations, we do not use SLP to localize lower extremity arterial occlusive disease but rather to categorize the degree of the patient's ischemia (Table 2.1). If the examiner remembers that such pressure changes may represent the sum of more than one lesion, misinterpretation is less likely to occur. Although the limitations of SLP can be minimized by interpreting them in the light of the patient's history and physical findings, an additional diagnostic modality, such as wave form analysis, should be a part of every complete extremity arterial evaluation.

Wave Form Analysis

Two types of wave form analysis are presently in widespread use: *Doppler velocity* and *pulse volume*. Most laboratories that measure arterial velocity wave forms use a directional continuous wave Doppler equipped with a zero-crossing, frequency-to-voltage converter to produce an electric signal, which is then transcribed on a standard strip-chart recorder. In the lower extremity, tracings are usually obtained from the common femoral, popliteal, and posterior tibial arteries. Using different dimensional parameters for the resulting wave forms, various authors have calculated the proximal damping quotient,[11] wave form index,[12] pulse rise time,[13] or pulsatility index[14] in an attempt to derive quantitative data. However, Flanigan et al compared the accuracy of femoral pulsatility index (FPI) to direct intra-arterial femoral pressure measurements in diagnosing aortoiliac disease and found that FPI had a sensitivity of only 62% and a specificity of 69%. They concluded that "FPI is not sufficiently accurate for its previously suggested use as a clinical tool to differentiate hemodynamically signif-

icant from hemodynamically insignificant aortoiliac occlusive disease."[15] Aukland and Hurlow compared the pulsatility index in the pedal arteries to arteriography in 29 patients with symptomatic lower extremity ischemia and concluded that "absolute values of the pulsatility index in the pedal arteries indicate the cumulative effect of occlusive disease in the whole limb, and therefore are of little value in grading the popliteal-tibial segment."[16]

Processing the Doppler velocity signal through a zero-crossing detector can introduce artifacts into the velocity profile and is susceptible to distortion from radio frequency interference, probe movement, and flow in adjacent veins.[17] Although all these concerns are theoretically valid, in the hands of an experienced technician this approach provides surprisingly useful data in most clinical situations. It also has the advantages of being readily available in most institutions, applicable to a wide variety of clinical problems, and one of the least expensive of the noninvasive techniques presently offered.

Nevertheless, if truly quantitative information is required, the Doppler signal must be processed by some other method, such as real-time frequency analysis. Using this technique, Johnston has been able to localize aortoiliac and superficial femoral artery occlusive disease with sensitivity greater than 80% and specificity greater than 95%. However, this approach remains relatively complicated and has not been widely accepted.

Regardless of the method used to process the Doppler velocity signal, several limitations persist: (1) considerable technical skill is required to produce consistent, artifact-free tracings; (2) the presence of excess fat, hematoma, or scar tissue around the vessel may significantly interfere with ultrasound transmission; (3) atherosclerotic plaque on the anterior wall of the vessel may make it difficult to obtain a diagnostically useful tracing; (4) in the presence of severe occlusive disease, the signal-to-noise ratio is low and one may be unable to obtain interpretable tracings, especially distal in the extremity; and (5) it is usually impossible to penetrate prosthetic grafts and obtain representative wave forms.

In view of these limitations, we rely on pulse volume wave forms (PVRs) to help localize extremity arterial occlusions. They provide a better assessment of segmental limb perfusion, are less technician dependent, are not limited by vessel wall calcification, are readily and rapidly obtained using the same cuffs already in place for SLPs, and can be analyzed qualitatively with considerable accuracy.[18,19]

Stress Testing

Although resting measurement of SLPs and PVRs usually provides clinically useful information, confusion arises when patients give a history suggestive of intermittent claudication but have borderline or even normal resting values. Furthermore, although such measurements may document

the presence and location of hemodynamically significant arterial lesions, they do not quantitate the resulting functional disability—information that may be critical in selecting patients for revascularization. When one extremity is afflicted with symptomatic arterial occlusive disease, resting pressure measurements in the contralateral, asymptomatic limb may be deceptively normal. Laing and Greenhalgh studied 100 such limbs and found that 78% had normal resting ankle pressures. However, after a brief period of treadmill exercise, 41% of these "normal" limbs had a definitely abnormal pressure response to exercise.[20]

Reactive hyperemia can be induced using a variety of techniques. Although we generally prefer treadmill exercise, we also use temporary, pneumatic cuff occlusion of segmental limb blood flow in the following clinical situations: (1) when walking is precluded by the patient's overall condition or by the presence of a previous amputation or foot lesion; (2) when one extremity is so severely symptomatic that the contralateral "normal" leg cannot be sufficiently stressed; or (3) when an upper extremity vessel is being evaluated as a possible donor source for an extra-anatomic bypass graft. In the latter case, exercise is difficult to standardize and cuff-induced ischemia is equally effective in assessing the vessel's potential to deliver the additional blood flow.

Except in the above situations, we study all persons who have exercise related lower extremity complaints before and after standardized treadmill walking. Depending on the patient's age and agility, the treadmill is set at a speed of $1\frac{1}{2}$ or $2\frac{1}{2}$ miles/hr and an incline of 10 degrees. Patients are exercised for five minutes or until they are forced to stop because of local or general symptoms. Ankle pressures and PVRs are then recorded as quickly as possible to confirm a drop in perfusion pressure compatible with the patient's symptoms.

We prefer exercise-induced reactive hyperemia because it duplicates the clinical setting in which the patient normally becomes symptomatic, it provides objective quantitation of his disability, and it permits assessment of his total response to exercise, thus facilitating identification of patients who are more limited by musculoskeletal or cardiopulmonary disease than by their claudication.

Duplex Scanning

Although indirect hemodynamic tests are useful in identifying the presence of arterial occlusive disease and roughly estimating its severity, they do not provide any of the anatomic information that will be needed in planning its correction, and they may not be sufficiently sensitive to predict impending failure of infrainguinal arterial reconstructions. However, the duplex scanner, which combines the anatomical data of a real-time, B-mode imager with the hemodynamic information derived from spectral analysis of the pulsed Doppler velocity wave form (Table 2.2), may prove more useful,

TABLE 2.2 Duplex scan criteria for estimating the severity of peripheral arterial stenoses.[21]

Grade	Criteria
Normal	Triphasic waveform
	No spectral broadening
1–19% Stenosis	Slight spectral broadening
	PSV <30% greater than adjacent proximal segment
20–49% Stenosis	No spectral "window"
	PSV <100% greater than adjacent proximal segment
50–99% Stenosis	PSV >100% greater than adjacent proximal segment
	Reverse velocity component absent
	Distal signal monophasic with reduced PSV
Occlusion	No flow in imaged artery
	Monophasic "thump" proximal to lesion
	Monophasic waveforms and decreased PSV in distal artery

PSV = peak systolic velocity

especially in evaluating the aortoiliac segment.[21] Its ability to distinguish high grade stenosis from occlusion and to detect hemodynamically insignificant disease is unique among noninvasive tests and represents the first practical means for longitudinally following discrete arterial lesions.

Angiography has long been considered the "gold standard" in the detection of extremity arterial occlusive disease. Yet, because it provides only two-dimensional information, it can be significantly misleading and it provides no physiologic data on the functional significance of the various lesions seen. Furthermore, it is expensive, invasive, poorly tolerated by patients and not suitable for long-term follow-up. Duplex scanning may potentially overcome all of these limitations. In a recent report, color flow duplex scanning was used to "map" the iliofemoral and femoral popliteal segments in 61 patients being evaluated for laser angioplasty.[22] When the findings on duplex were compared with traditional angiograms as the "gold standard," specificity and sensitivity were excellent. (Table 2.3)

Duplex scanning has proved to be an accurate and reliable noninvasive method for following infrainguinal autogenous bypass grafts. Green et al. followed 177 patients with infrainguinal grafts and considered a duplex scan of the bypass *abnormal* if peak systolic flow velocity (PSV) was >120 cm/sec or <40 cm/sec.[23] No patient in this study with abnormal ABI and duplex scan suffered sudden graft occlusion prior to the next study. If the duplex scan was abnormal but the ABI was normal, the incidence of sudden graft occlusion was 4%. By contrast, if both the duplex scan and ABI were abnormal, the risk of graft occlusion was 66%. Sladen et al. also found that PSVs >300 cm/sec or three times the velocity in the adjacent normal graft were very sensitive in identifying the failing graft.[24] Mills et al. performed a similar study on 379 infrainguinal reversed vein grafts and

TABLE 2.3 Comparison of traditional angiography and color flow duplex scanning when used to "map" the lower extremity arterial tree.[22]

	Sensitivity	Specificity
Normal vs abnormal	96%	83%
50% Stenosis	99%	87%
Occlusion	99%	81%

found that only 2.1% of grafts with a PSV >45 cm/sec in the graft failed within the next six months.[25] By contrast, 12.6% of grafts with PSV <45 cm/sec were found to have a stenotic lesion when studied with angiography. Somewhat surprisingly, only 29% of the grafts identified by duplex scan as *failing* were associated with a reduction of ABI >0.15. This study emphasizes the importance of a routine surveillance protocol utilizing duplex scan for all infrainguinal bypass grafts.

The amplitude and the quality of the velocity waveform obtained in any given vascular graft will vary depending on the site where the recording is made, the presence or absence of significant tapering or narrowing along the length of the conduit, and the outflow resistance of the runoff vessels.[26] For example, an *in-situ* vein graft usually tapers from proximal to distal. Accordingly, blood flow can be expected to accelerate along the length of such a graft and the highest flow velocities will be expected distally. By contrast, a reversed saphenous vein graft will generally show a decrease in flow along its length as the graft becomes progressively larger. Prosthetic grafts are isodiametric and PSV should be constant in such a graft.

In the period following a successful lower extremity bypass, a hyperemic flow pattern with forward end-diastolic flow velocity (EDV) should be observed due to the low peripheral vascular resistance usually present. Within one to two months of operation, normal peripheral vascular resistance should return and a triphasic waveform should be seen. However, despite these differences in EDV, PSV should not change significantly.

Typically, PSV is highest in femoropopliteal grafts with good runoff, intermediate in femorotibial grafts, and least in grafts to isolated popliteal segments or femorotibial grafts with poor runoff. Despite the numerous factors that can influence PSV in successful vascular grafts, flow velocity is typically >45 cm/sec.[26] However, a graft flow velocity between 35–45 cm/sec has been described in some successful large diameter (>6 cm) *in-situ* grafts with very limited runoff.

Transcutaneous pO_2 Measurement

Transcutaneous measurement of tissue oxygen content ($TcpO_2$) adds yet another dimension to the noninvasive evaluation of extremity arterial disease, since it is capable of documenting the hypoxia that is the hallmark

of ischemic tissue and can thus provide information to supplement the hemodynamic parameters presently generated by most vascular laboratories. Although it has been used to classify patients into various clinical categories based on the severity of their disease, this information is already available from hemodynamic studies. The greatest utility of $TcpO_2$ presently appears to be in the assessment of diabetic patients with incompressible vessels and in predicting the healing potential of ischemic lesions or amputations. However, additional experience will be required to determine not only the optimal technique for measuring $TcpO_2$ but also its clinical applications,[27] although early experience suggests that it may become an important addition to most vascular laboratories.

Conclusions

Our routine evaluation for extremity arterial insufficiency includes: (1) resting SLPs and PVRs at three or more levels in each extremity; and (2) treadmill exercise in patients with exercise related complaints. For patients with a functioning infrainguinal bypass graft, the entire graft is initially scanned prior to discharge from the hospital. On subsequent follow-up visits, flow velocities are only monitored in the proximal, mid and distal thirds of the graft. Presently, we are not routinely scanning the native arterial tree at the time of initial evaluation. $TcPO_2$ measurement is reserved for patients being evaluated to predict the healing potential of extremity ulcers or planned amputations if the hemodynamic data is equivocal.

Extremity Venous Disease

Deep Venous Thrombosis

The diagnosis of acute deep venous thrombosis (DVT) based on clinical criteria alone is notoriously inaccurate.[28] Haeger[29] found that 46% of patients with the clinical diagnosis of DVT had normal ascending venograms. For many years, the gold standard for diagnosis of DVT was ascending venography. However, the expense, discomfort, and lack of repeatability of venography, coupled with its potential for initiating thrombosis, limits its usefulness as a diagnostic procedure.[30,31] Given the frequent need for screening asymptomatic high-risk patients, an ideal noninvasive method of detecting acute DVT should be rapid, repeatable, well tolerated by patients, and have high positive and negative predictive values.

Most of the initial noninvasive tests for DVT were indirect and were based on the detection of hemodynamic alterations in the extremity distal to a more proximal thrombus. Although they represented a real advance compared to venography and were widely adopted, they were only moderately sensitive, especially to partially occluding thrombi, and were unable

to provide any anatomic information regarding the extent or the age of the thrombi. The recent availability of duplex venous scanning has overcome most of these objections and has led to broad acceptance of this technique for the diagnosis of DVT and the surveillance of high risk patients. In fact, the technique has been so well accepted by patients and physicians that few venograms are any longer ordered in most institutions where high quality, duplex venous scanning is available, thus making it difficult for many vascular laboratories to obtain sufficient numbers of venograms to validate the accuracy of duplex scanning in their own institution and to prospectively monitor the quality of their laboratory.

Impedance Plethysmography

Impedance plethysmography (IPG) has been reported to be a sensitive indicator of hemodynamically significant venous outflow obstruction.[32] The acknowledged limitations of IPG are both anatomic (inaccurate in the diagnosis of thrombi in calf veins) and patient dependent (high false positive rate with obesity, congestive failure, external venous compression, and chronic DVT). Despite these recognized limitations, IPG has been the principal technique used for the diagnosis of acute DVT in most vascular laboratories. Even following the development of venous duplex scanning, there were theoretical reasons to believe that it would continue to be a useful adjunct to this direct technique.

Impedance plethysmography depends upon changes in electrical impedance in an extremity following inflation and release of a proximal venous tourniquet to diagnose the presence of compromised venous outflow.[33] Thus, it provides indirect, hemodynamic data rather than anatomic evidence of venous obstruction. False negative results can occur with nonocclusive thrombi and with well collateralized thrombosis of the calf veins. False positive results occur with any condition limiting venous outflow, for example, extrinsic compression of the deep veins (tumor, pregnancy), previous DVT with inadequate collateralization or recanalization, severe congestive heart failure with elevated venous filling pressures, etc. Lack of patient cooperation, obesity, arterial occlusive disease, and low ambient temperatures may also contribute to inadequate studies.

IPG's theoretic advantage over venous scanning is its ability to detect suprainguinal thrombi in symptomatic patients. Although a high degree of accuracy has been reported by some investigators, there is significant variability in the results achieved at different institutions[33–38] (Table 2.4). Bilaterally abnormal studies are associated with acute thrombus in only 26% of patients subsequently studied with venography.[39] The hemodynamic abnormalities seen in the postoperative period[40] contribute to the possibility of false positive studies in this high risk population. Comerota et al.[37] documented that IPG is especially inaccurate as a surveillance tool in asymptomatic patients.

TABLE 2.4 Diagnostic accuracy of IPG compared with venography in selected series.

	#	SENS (%)	SPE	PPV (%)	NPV (%)
Wheeler et al[33]	78	80.0	60.4	48.8	86.5
Flanigan et al[34]	207	90.1	75.7	66.0	93.6
Moser et al[35]	42	60.9	100	100	67.9
Hume et al[36]	32	77.3	100	100	66.7
Comerota et al[37]	308	49.0	85.0	80.0	58.0
Patterson et al[38]	49	75.0	44.8	48.4	72.2

DUPLEX SCANNING

Real-time, B-mode imaging of lower extremity veins provides a rapid, noninvasive, direct means of identifying acute DVT. The scan heads used in most laboratories permit complete interrogation of the infrainguinal, deep venous system in virtually all patients. However, inadequate depth of penetration and limited image resolution preclude routine assessment of the suprainguinal veins.

Since Talbot[41] first suggested that real-time, B-mode imaging could be applied to the diagnosis of DVT, several centers have accumulated extensive experience with this technique (Table 2.5).[42–46] The patients are examined in the supine position and an effort is made to visualize all the named veins below the inguinal ligament. The vena cava and the iliac veins usually cannot be interrogated unless the patient is quite thin. The criteria for a positive duplex scan include: demonstration of echogenic material within the lumen of the vein, noncompressibility of the vein, absence of demonstrable flow within the vein when interrogated with the Doppler, and failure of the vein to dilate with the Valsalva maneuver. Difficulties in interpretation arise in obese patients and in veins that are poorly compressible due to their anatomic location (superficial femoral vein at the adduc-

TABLE 2.5 Diagnostic accuracy of duplex scan compared with ascending venography.

	#	SENS (%)	SPE (%)	PPV (%)	NPV (%)
Oliver[42]	28	80.0	95.0	95.0	87.5
Hannan et al[43]	68	95.0	89.0	93.0	92.0
Sullivan et al[44]	23	100	92.0	92.0	100
Langsfeld et al[45]	19	100	78.0	83.3	100
Comerota et al[46]	110	96.0	93.0	96.0	96.0
Patterson et al[38]	64	88.9	91.9	88.9	91.9

tor hiatus, deep peroneal vein, etc.). The use of a lower frequency scan head (4 MHz) to interrogate the deep veins sacrifices resolution for the sake of deeper tissue penetration.[47]

The combination of IPG and duplex scanning theoretically should represent an ideal diagnostic pairing. The hemodynamic evaluation of the deep venous system provided by the IPG should compliment the anatomic information obtained by B-mode imaging of the infrainguinal deep veins. The increased sensitivity of this combination should diminish the likelihood of missing potentially life-threatening thrombi.

Unfortunately, clinical experience does not support this hypothesis. The improvement in sensitivity which occurred when IPG was combined with scan versus scan alone (95% vs 88.9%) was not statistically significant.[38] Furthermore, the addition of IPG significantly diminished the specificity of duplex scan alone due to a large number of false positive tests. Although the majority of the false positives had an identifiable cause for their misinterpretation (extrinsic venous compression, chronic venous obstruction, limb paresis, etc.), if therapy had been based on the results of the combined noninvasive tests, it would have resulted in the inappropriate treatment of more than half of the patients studied.

Arguments in favor of continuing to use IPG are based on the inability of current venous scanners to detect suprainguinal thrombi. In our experience, potential proximal disease "missed" by venous scan consisted of a single focal iliac thrombus associated with extrinsic compression of the vessel.[38] Thus, the occurrence of acute thrombi limited to the iliac veins without infrainguinal extension was uncommon and the venous scan accurately detected most thrombi despite its theoretical limitations. Certainly the high rate of false positive IPGs precludes treatment based on this technique alone. By contrast, we feel that treatment may be based on venous scans with great confidence. If a negative scan occurs in a high risk patient in whom there is a strong clinical suspicion of proximal DVT, the appropriate diagnostic study is venography.

Venous Insufficiency

Chronic venous insufficiency is a common clinical problem. Unfortunately, unlike the diagnosis of DVT, there is as yet no reliable noninvasive technique for quantitatively assessing the severity of venous valvular dysfunction and localizing the site of valvular incompetence. The lack of a simple, readily available, accurate, noninvasive technique to objectively document and quantitate venous insufficiency of the lower extremities has hindered our understanding of the pathophysiology of this common problem, has restricted the selection of appropriate candidates for surgical correction and has limited our ability to access and follow the results of therapy. Direct measurements of ambulatory venous pressure have been used in clinical investigations of this problem, but these studies are cumbersome,

invasive, and impractical for routine clinical use. However, two tests, venous photoplethysmography and venous duplex, are sufficiently promising to warrant further discussion.

Continuous Wave Doppler Survey

Interrogation of venous valvular competence using a hand-held, continuous wave (CW) Doppler probe is a direct method for obtaining objective information regarding venous physiology. Unfortunately, this technique, although widely used, is very operator dependent and requires great experience, patience and precision to obtain useful information. It is also limited by its inability to directly evaluate several important but inaccessible veins such as the profunda femoris vein, the calf vein, or the soleal sinuses. In addition, many of the major, infrainguinal veins are duplicated, thus further increasing the potential for misdiagnosis. Given all of the above limitations, CW Doppler in not an adequate primary technique for the diagnosis of venous insufficiency.

Photoplethysmography

The introduction of the photoplethysmograph (PPG)[48] and the subsequent documentation that its alterations with leg exercise corresponded to similar changes in direct venous pressure measurements[49] represented a major advance in the management of venous insufficiency. This technique provided clinicians for the first time with a simple, noninvasive, objective method for studying the venous physiology in their patients and documenting the results of therapy. Using a transducer which emits infrared light into the underlying tissue and measures the backscattered infrared light, this technique indirectly gauges the cutaneous blood content in the dermal capillary network. Following a brief series of calf muscle contractions, there is a rapid fall in blood content of the skin. If venous valvular function is intact, refilling of the dermal capillaries (**Venous Recovery Time, VRT**) is gradual with VRT >23 seconds considered normal and VRT <20 seconds indicative of venous insufficiency.[49] Unfortunately, a significant number (30%) of apparently normal, young patients with no clinical or physical evidence of venous insufficiency were found to have VRT <20 seconds.[50] Furthermore, even minor amounts of exercise prior to testing or other clinical conditions such as cellulitis, which might cause reactive hyperemia of the lower extremities, resulted in further shortening of the VRT. Thus, the VRT was found to be a function not only of venous valvular competence but also of the effectiveness of antecedent venous emptying and the arterial blood flow into the extremity.

To develop a parameter that better reflected this complex interrelationship, several authors have suggested that the **rate** of venous recovery, rather than the absolute time, was a better measure of venous insufficiency,[50] that volume data must be normalized for variations in arterial

flow,[51] and that emptying the veins by elevation was preferable to reliance on calf muscle contractions.[51] Despite these improvements in the technique, PPG remains an indirect, hemodynamic test incapable of giving any of the anatomic information which necessary to select patients for surgical correction of their problem and appears to more accurately reflect superficial rather than deep venous insufficiency.[52]

Duplex Scanning

In an attempt to overcome the above objections, some investigators have recently used duplex scanners to study venous valvular function. Using an automatic cuff inflator for rapid inflation and deflation of pneumatic cuffs placed at different levels of the limb and a duplex scanner to record venous velocity time response proximal and distal to cuff inflation, van Bemmelen et al. found that the median duration of reflux in normal popliteal veins was 0.19 seconds, with 95% of the values being <0.66 seconds.[53] In distal areas, duration of reflux was short and uniform. Unfortunately, this study was limited to normal subjects and thus provides no information on what we might expect to find in patients with venous insufficiency.

Using a similar technique, Vasdekis et al. studied 46 patients with symptomatic varicose veins.[54] The best separation between patients without and with liposclerosis and/or ulceration was produced by the measured venous flow at peak reflux. In limbs with skin changes, reflux (median ±90% tolerance levels) was 30 ml/sec; whereas in limbs without skin changes, it was 10 mm/sec. Reflux >10 ml/sec was associated with a high incidence of skin changes (66%) irrespective of whether this was in the superficial or deep veins. Reflux <10 ml/sec was not associated with skin changes.

Although preliminary, these results are encouraging and suggest that duplex scanning may become an important part of the evaluation of patients with venous insufficiency. It remains as yet unclear whether or not this technique will replace the volume-based indirect tests on which we have relied up to now or whether they will complement them.

Conclusions

Our laboratory currently relies on duplex scanning alone for the documentation of venous thrombosis. We no longer perform indirect tests such as IPG or phleborrheography, since they do not increase the sensitivity of duplex scanning alone but significantly reduce its specificity when used in combination with the latter. However, in those laboratories where high quality duplex scanning is not currently available, indirect tests are far preferable to clinical diagnosis and can provide valid objective data on which to base patient management.

For the evaluation of venous insufficiency, we still largely rely on PPG to document venous reflux. If this study is consistently normal, we proceed no

further. If it is abnormal, we then perform dynamic duplex scanning to confirm the indirect study and to localize the site of valvular incompetence.

References

1. Baker WH, String ST, Hayes AC, et al. Diagnosis of peripheral occlusive disease. Comparison of clinical evaluation and noninvasive laboratory. Arch Surg 113:1308, 1978.
2. Marinelli MR, Beach KW, Glass MJ, et al. Noninvasive testing vs. clinical evaluation of arterial disease. A prospective study. JAMA 241:2031, 1979.
3. Van De Water JM, Laska ED, and Ciniero WV. Patient and operation selectivity. The peripheral vascular laboratory. Ann Surg 189:143, 1979.
4. Heintz SE, Bone GE, Slaymaker EE, et al. Value of arterial pressure measurements in the proximal and distal part of the thigh in arterial occlusive disease. Surg Gynecol Obstet 146:337, 1978.
5. Brener BJ, Tiro AC, Alpert J, et al. Three techniques for assessing iliac artery stenosis: External femoral artery compression, segmental thigh pressures, and intraarterial femoral pressures, **In** Diethrich EB (ed): *Noninvasive Cardiovascular Diagnosis*, ed. 2., Baltimore, University Park Press, 1981.
6. Flanigan DP, Gray B, Schuler JJ, et al. Utility of wide and narrow blood pressure cuffs in the hemodynamic assessment of aortoiliac occlusive disease. Surgery 92:16, 1982.
7. Lynch TG, Hobson RW, Wright CB, et al. Interpretation of Doppler segmental pressures in peripheral vascular occlusive disease. Arch Surg 119:465, 1984.
8. Franzeck UK, Bernstein EF, and Fronek A. The effect of sensing site on the limb segmental blood pressure determination. Arch Surg 116:912, 1981.
9. Lazarus HM, Albo D, Welling D, et al. Doppler ankle pressures and stiff arteries, **In** Diethrich EB (ed): *Noninvasive Cardiovascular Diagnosis: Current Concepts*, Baltimore, University Park Press, 1978.
10. Baker JD and Dix D. Variability of Doppler ankle pressures with arterial occlusive disease: An evaluation of ankle index and brachial-ankle pressure gradient. Surgery 89:134, 1981.
11. Waters KJ, Chamberlain J, and McNeil IF. The significance of aortoiliac atherosclerosis as assessed by Doppler ultrasound. Surgery 134:338, 1977.
12. Nicolaides AN, Gordon-Smith IC, Chir M, et al. The value of Doppler blood velocity tracings in the detection of aortoiliac disease in patients with intermittent claudication. Surgery 80:774, 1976.
13. Fronek A, Johansen KH, Dilley RB, et al. Noninvasive physiologic tests in the diagnosis and characterization of peripheral arterial occlusive disease. Am J Surg 126:205, 1973.
14. Johnston KW and Taraschuk I. Validation of the role of pulsatility index in quantitation of the severity of peripheral arterial occlusive disease. Am J Surg 131:295, 1976.
15. Flanigan DP, Collins JT, Schwartz JA, et al. Hemodynamic and arteriographic evaluation of femoral pulsatility index. J Surg Res 32:234, 1982.
16. Aukland A and Hurlow RA. Spectral analysis of Doppler ultrasound: its clinical application in lower limb ischaemia. Br J Surg 69:540, 1982.
17. Johnston KW, in discussion, Nicolaides AN, Gordon-Smith IC, Chir M, et al.

The value of Doppler blood velocity tracings in the detection of aortoiliac disease in patients with intermittent claudication. Surgery 80:774, 1976.
18. Kempczinski RF. Segmental volume plethysmography in the diagnosis of lower extremity arterial occlusive disease. J Cardiovas Surg 23:125, 1982.
19. Rutherford RB, Lowenstein DH, and Klein MF. Combining segmental systolic pressures and plethysmography to diagnose arterial occlusive disease of the legs. Am J Surg 138:211, 1979.
20. Laing S and Greenhalgh RM. The detection and progression of asymptomatic peripheral arterial disease. Br J Surg 70:628, 1983.
21. Kohler TR. Duplex scanning of lower limb arteries. Seminars in Vasc Surg 1:17, 1988.
22. Cossman DV, Ellison JE, Wagner WH, et al. Comparison of contrast arteriography to arterial mapping with color-flow duplex imaging in the lower extremities. J Vasc Surg 10:522, 1989.
23. Green RM, McNamara J, Ouriel K, DeWeese JA. Comparison of infrainguinal graft surveillance techniques. J Vasc Surg 11:207, 1990.
24. Sladen JG, Reid JDS, Cooperberg PL, et al. Color flow duplex screening of infrainguinal grafts combining low- and high-velocity criteria. Am J Surg 158:107, 1989.
25. Mills JL, Harris EJ, Taylor LM, et al. The importance of routine surveillance of distal bypass grafts with duplex scanning: A study of 379 reversed vein grafts. J Vasc Surg 12:379, 1990.
26. Bandyk DF. Monitoring functional patency of vascular grafts. Seminars in Vasc Surg 1:40, 1988.
27. Martin KD, Uhl S, Kempczinski RF. Transcutaneous oxygen measurement: A comparison of current monitors. J Vasc Tech 14:166, 1990.
28. Barnes RW, Wu KK, Hoak JC. Fallibility of the clinical diagnosis of venous thrombosis. JAMA 234:605, 1975.
29. Haeger, K. Problems of acute deep venous thrombosis: I. The interpretation of signs and symptoms. Angiology 20:219, 1969.
30. Albrechtsson U, Olsson C-G. Thrombotic side-effects of lower-limb phlebography. Lancet 1:723, 1976.
31. Thomas ML, MacDonald LM. Complications of ascending phlebography of the leg. Br Med J 2:317, 1978.
32. Wheeler HB. Diagnosis of deep vein thrombosis. Review of clinical evaluation and impedance plethysmography. Am J Surg 150(suppl):7, 1985.
33. Wheeler HB, Anderson FA Jr, Cardullo PA, et al. Suspected deep vein thrombosis. Management by impedance plethysmography. Arch Surg 117:1206, 1982.
34. Flanigan DP, Goodreau JJ, Burnham SJ, et al. Vascular-laboratory diagnosis of clinically suspected acute deep-vein thrombosis. Lancet 2:331, 1978.
35. Moser KM, Brach BB, Dolan GF. Clinically suspected deep venous thrombosis of the lower extremities: A comparison of venography, impedance plethysmography, and radiolabeled fibrinogen. JAMA 237:2195, 1977.
36. Hume M, Kuriakose TX, Jamieson J, Turner RH. Extent of leg vein thrombosis determined by impedance and 125-I fibrinogen. Am J Surg 129:455, 1975.
37. Comerota AJ, Katz ML, Grossi RJ, et al. The comparative value of noninvasive testing for diagnosis and surveillance of deep vein thrombosis. J Vasc Surg 7:40–49, 1988.

38. Patterson RB, Fowl RJ, Keller JD, Kempczinski RF. The limitations of impedance plethysmography in the diagnosis of acute deep venous thrombosis. J Vasc Surg 9:725, 1989.
39. Curley FJ, Pratter MR, Irwin RS, Anderson FA Jr, Phillips DA, Doherty PW, Wheeler HB, Dalen JE. The clinical implications of bilaterally abnormal impedance plethysmography. Arch Int Med 1987; 147:125–129.
40. Tripolitis AJ, Bodily KC, Blackshear WM Jr, et al. Venous capacitance and outflow in the postoperative patient. Ann Surg 190:634–637, 1979.
41. Talbot SR. Use of real-time imaging in identifying deep venous obstruction: A preliminary report. Bruit 6:41–42, 1982.
42. Oliver MA. Duplex scanning in venous disease. Bruit 9:206–209, 1985.
43. Hannan LJ, Stedje KJ, Skorcz MJ, et al. Venous imaging of the extremities: Our first twenty-five hundred cases. Bruit 10:29–32, 1986.
44. Sullivan ED, Peter DJ, Cranley JJ. Real-time B-mode venous ultrasound. J Vasc Surg 1:465–471, 1984.
45. Langsfeld M, Hershey FB, Thorpe L, et al. Duplex B-mode imaging for the diagnosis of deep venous thrombosis. Arch Surg 122:587–591, 1987.
46. Comerota AJ, Katz ML, Greenwald LL, et al. Venous duplex imaging: Should it replace hemodynamic tests for deep venous thrombosis? J Vasc Surg 11:53, 1990.
47. Karkow WS, Ruoff BA, Cranley JJ. B-mode venous imaging. **In**: Kempczinski RF, Yao JST (eds): *Practical Noninvasive Vascular Diagnosis*. Chicago, Year Book Medical Publishers, 1987, pp 464–485.
48. Barnes RW, Garrett WV, Hummel BA, et al. Photoplethysmographic assessment of altered cutaneous circulation in the post-phlebitic syndrome. Proceedings of the AAMI 13th Annual Meeting, Washington DC, March 28–April 1, 1978.
49. Abramowitz HB, Queral LA, Flinn WR, et al. The use of photoplethysmography in the assessment of venous insufficiency: A comparison to venous pressure measurements. Surgery 86:434, 1979.
50. Kempczinski RF, Berlatzky Y, Pearce WH. Semi-quantitative photoplethysmography in the diagnosis of lower extremity venous insufficiency. J Cardiovasc Surg 27:17, 1986.
51. Peirce EC, Chiang K, Schanzer H. Volume tests for chronic venous insufficiency: An appraisal. Surgery 109:567, 1991.
52. Rosfors S. Venous photoplethysmography: Relationship between transducer position and regional distribution of venous insufficiency. J Vasc Surg 11:436, 1990.
53. van Bemmelen PS, Bedford G, Beach K, Strandness DE. Quantitative segmental evaluation of venous valvular reflux with duplex ultrasound scanning. J Vasc Surg 10:425, 1989.
54. Vasdekis SN, Clarke GH, Nicolaides MS. Quantification of venous reflux by means of duplex scanning. J Vasc Surg 10:670, 1989.

3
History of Carotid Surgery: Present Status and Future

ANTHONY M. IMPARATO

The charge to present a history of carotid surgery can be daunting, raising the specter of pouring over dusty tomes in the dark recesses of libraries to compile lists of "firsts," as is often done, and thereby establish one's right to claim erudition. To quote from a recently published volume on the history of vascular surgery, Thomas Willis first recognized the significance of a totally occluded carotid artery in 1684 from his postmortem examinations, which led him to describe the configuration of the cerebral vasculature and the anastomotic circle bearing his name. The first recorded operations upon the carotid were done by Hebenstreit in 1793 and Abernathy in 1798 for control of hemorrhage, while Astley Cooper performed the first carotid artery ligation for aneurysm in 1805.[1] Such an approach admirably satisfies one of Webster's definitions of history: "an account of what has happened; narrative, story, tale." But there is another definition of history, viz., "a recording, analyzing, coordinating and explaining of past events."[2] Can such an endeavor yield sufficient insight to predict future events, to guide the course of research to accelerate acquiring the understanding necessary to gain mastery over a human affliction that has probably had as profound an effect on the recent course of human events as any other? To illustrate, three of the major participants at the Yalta Conference in 1945 suffered ischemic strokes or were suspected of having occlusive carotid arterial disease,[3] while at least three other 20th Century American presidents also experienced bouts of recognizable cerebral dysfunction characteristic of stroke syndromes: Wilson, Eisenhower and Johnson.[4,5] Admittedly, progress in stroke prevention has been slow when one realizes that King David (1050 B.C.) described the effects of a left hemispheric stroke, "Let my right hand wither, let my tongue affix to the roof of my mouth. . . .",[6] and that the ancient Greeks, namely Hippocrates (400 B.C.), understood that the carotid arteries were related to consciousness, that individuals who suffered "little strokes" would shortly suffer major ones. McHenry, in his volume *Cerebral Circulation and Stroke*, cites from the Hippocratic writings, "unaccustomed attacks of numbness and anesthesia are signs of impending apoplexy." Apoplexy, in Greek, meant struck with violence, re-

sulting in paralysis of some or all parts of the body.[7] Thereafter, there followed more than 20 centuries of intellectual struggle to understand and explain the pathogenesis of stroke syndromes until Chiari in 1905 emphasized the role of extracranial occlusive arterial disease in the production of neurologic symptoms and emphasized the frequency of carotid bifurcation atherosclerotic disease and atheromatous embolization as their direct cause.[8]

In 1914 Ramsay Hunt described the role of carotid artery occlusion in the production of symptomatic vascular lesions of the brain,[9] and of course, Miller Fisher in the early 1950s not only reemphasized the role of the carotid arteries in the causation of stroke but provided admirable descriptions of carotid pathology. Great credit goes to Dr. Fisher, a neurologist, for suggesting the possibility that surgical intervention upon the carotid bifurcation might be an effective therapeutic modality in stroke prevention.[10,11] Thereafter, it was a foregone conclusion that one or more enterprising surgeons would take up the challenge offered by Fisher and attempt to deal surgically with carotid bifurcation occlusive atherosclerotic disease. Carrea, Molins and Murphy, 1951 in Argentina, resected the stenotic segment of an internal carotid artery of a 41-year-old man who had suffered recent aphasia and right hemiparesis and performed end-to-end anastomosis between the external and distal internal carotid arterie.[12] In 1953 Strully, Hurwitt and Blankenburg at the Montefiore Hospital in New York unsuccessfully attempted to perform thromboendarterectomy of the totally occluded internal carotid artery of a 53-year-old man who suffered bouts of left-sided hemispheric cerebral transient paralysis,[13] while in that same year Dr. Michael DeBakey successfully performed thromboendarterectomy of the common and internal carotid arteries of a 53-year-old man who suffered repeated attacks of transient left-sided cerebral hemispheric strokes. He did not, however, report this experience until 1975, three years after the patient died from unrelated causes, neurologically intact, having suffered no additional small strokes.[14]

The most profound effect upon carotid surgery, however, resulted from the operation reported by Eastcott, Pickering and Rob, who on May 19, 1954, operated upon a 66-year-old woman and performed resection of the left carotid bifurcation with end-to-end anastomosis between the common and internal carotid arteries. The details of this endeavor illustrate many of the subsequent concerns associated with carotid surgery. Their patient, who had experienced 33 left-sided small strokes and transient monocular blindness and who was shown by angiography to have total occlusion of the left internal carotid artery, was operated upon under general body hypothermia, made an uneventful recovery[15] and was relieved of her attacks for over 20 years.

Thereafter, although there were sporadic attempts to reconstruct the carotid bifurcation with arterial homografts (Denman 1954)[16] and autologous saphenous vein (Lin 1955)[17]—probably greatly influenced by

Wylie, carotid endarterectomy became the most commonly performed operative procedure for carotid bifurcation atherosclerotic disease. The technique of endarterectomy had been described by Dos Santos in 1947,[18] yet it was nearly a decade before it was generally appreciated that the sharply localized and limited extent of the carotid bifurcation plaque made it ideal for removal by endarterectomy.

This severely abbreviated account of the events leading to the more recent history of carotid surgery for stroke fails to deal with the dramatic events which have occurred in the four decades since the first successful operation by Carrea and associates.

A number of excellent references listed in the bibliography provide greater detail of the early history of carotid surgery.[1,19–22] All, however, fail to deal with the events which have occurred since the acceptance of carotid surgery as a viable therapeutic endeavor. It is upon this phase of history that I shall dwell.

Over 3,000 years of astute observations of the anatomy and pathology of the cerebral circulation, of insightful clinical-pathological correlations, and of daring surgical endeavors by individuals too numerous to mention culminated in the state of the art as it was four decades ago. By then the relationship of the extracranial cerebral arteries to cerebral function, their role in the causation of transient cerebral symptoms and catastrophic strokes, and the ability of surgeons to successfully deal with occlusive lesions and eliminate symptoms had been established. Why then, in 1991, was there still doubt regarding the soundness of the concept that carotid bifurcation atherosclerotic lesions cause ischemic strokes either by embolization or by producing watershed ischemia, and that elimination of such lesions prevents or relieves ischemic strokes?

The past four decades have been fraught with controversy and confusion but in many ways blessed with brilliant advances. When, at later dates, medical historians survey the recorded events and attempt to "analyze, coordinate and understand" those events, what will they conclude?

History, as recorded, is an abstraction, an illusion conceived by historians attempting to create order out of chaos. What happened at the Battle of Gettysburg? What was it like? There were thousands of Battles of Gettysburg, each fought by the thousands of its participants, for each of whom the battle differed, and from each, were it possible to obtain their versions, would emerge a different order of understanding, each valuable in its own way and perhaps more valuable than the composite version necessarily created by historians. Having lived through the four decades of the modern era of carotid surgery and participated in its evolution or its battles if you will, what were they like? What can be learned from a recounting by one of the combatants of some of the events of the battle to conquer this terrible affliction that threatens and destroys the most valuable and irreplaceable of our many organs? The events are too numerous to recount in toto, the

participants legion, the weapons marshalled numerous, complex and effective; and so, only the highlights will be documented, the dates, recalled from memory, approximated.

For nearly a decade after the first reported carotid operation, many surgeons successfully explored the possibility that these operations could be performed with an acceptable degree of safety. Cerebral angiography became a nearly routine procedure as a cadre of vascular and neuroradiologists appeared upon the scene to relieve the surgeons of the onerous and unfamiliar task of performing the angiographic studies. The toxicity of the iodinated radiographic contrast media became markedly lessened by the introduction of the methylglucamine salt of renografin to replace the much more toxic sodium salts—Urokon and Diodrost, probably names long forgotten.[23] To Fields, DeBakey, Lyons, Thompson, Wylie[24–28] and numerous others, already prominent in the field as successful carotid surgeons, it became apparent that there was need to demonstrate the efficacy of the operation in preventing and relieving stroke and perhaps in prolonging life. I well recall a conference at the New York Academy of Medicine in the late '50s when Dr. Charles Rob offered a challenge by publicly asking whether the patient who became asymptomatic following carotid endarterectomy did so because of, or in spite of, operation, since it was known by then that transient symptoms associated with stenotic carotid arteries could stop occurring upon thrombosis of the artery, and asymptomatic post carotid endarterectomy thrombosis had already been recognized!

In any event, spurred by Dr. DeBakey and the astute and amiable neurologist, Dr. William Field, a prospective study of carotid endarterectomy was sponsored by the NIH. By 1962, 21 university services were participants, 19 of which had agreed to randomize patients to surgical treatment. Participants in The (JSEAO) Joint Study of Extracranial Arterial Occlusions[29–33] agreed to perform four-vessel angiographic studies to include visualization of the intracranial vessels on all patients who presented with any complex of neurologic symptoms, including devastating acute strokes that might possibly be due to cerebral ischemia, and to surgically correct all accessible extracranial arterial lesions producing 30% or more stenosis, whether affecting the anterior or posterior circulation, whether deemed "appropriate" to focal symptoms or not. Nonfocal cerebral symptoms were to be studied to learn the full spectrum of clinical syndromes that might result from extracranial arterial disease. Thirty percent diameter reduction was considered significant, since the radiologists felt that that degree of stenosis was the least that all radiologists would recognize on the films. The surgeons, already experienced in vascular operations, recognized that even minimal atherosclerotic lesions could rapidly progress to total occlusion by thrombosis. Although the concept of embolization had previously been described by Chiari, Hunt and Fisher, it

was not recognized in the original protocol. Follow-up was to be for five years at least, or until statistical significance was achieved, the end points being death, stroke or transient cerebral symptoms.

The enthusiasm of the participants was almost electric. Meetings were held regularly to exchange ideas and review problems. A major concern of all the participants was the possibility of precipitating cerebral infarction during the period of flow interruption to permit endarterectomy. There was no unanimity of opinion, however, regarding how best to monitor the brain or how to protect it from suffering ischemic damage. The discussions were lengthy and would sound absolutely familiar today, since they continue to occur in the same vein: local versus general anesthesia; electroencephalographic monitoring; carotid stump pressures; jugular venous oxygen saturation determinations; cerebral protection by routine shunting, by artificial elevation of blood pressure, by using specific general anesthetic agents, or by induction of general body hypothermia. I admit to having been alarmed by the inability of the most experienced surgeons to agree upon the best anesthetic management. I have been influenced by the thoughtful and wise observations of Champ Lyons, who suggested that the predictive key to toleration of carotid clamping might ultimately be determined by analyzing the patterns of intracerebral blood flow observed on the angiographic studies, and by Jr. Jack Wylie who spoke of the apparent unreliability of electroencephalography in predicting cerebral ischemia under general anesthesia. As a junior participant I decided to adopt the technique of local anesthesia to permit monitoring conscious patients, employing selective shunting for clamping intolerance aided by artificial elevation of blood pressure. This approach would afford the opportunity to compare the reliability of various monitoring techniques and protective measures in conscious patients.

Equally disturbing was the fact that the descriptions of carotid plaques in situ, as observed by the surgeons before distortion was caused by surgical manipulation, varied so markedly that drawing meaningful conclusions regarding pathogenesis of the arterial lesions and of cerebral symptoms was not possible. Indeed, the descriptions of plaques given to us by our pathologist frequently were at marked variance from our original operative observation, until a fine camera was secured with which to photograph plaques in situ. When supplied with this record, our pathologist could properly reconstruct the plaque and give more accurate descriptions.

Although the validity of the study has been repeatedly questioned for many reasons, the findings were dramatic, enlightening, exciting, and set the stage for all subsequent investigations, which in essence have confirmed its essential findings:

1. Acute stroke with altered consciousness is not surgically correctible without doubling stroke mortality from 20% for medically treated patients to 40% for surgical treatment.

2. Carotid endarterectomy must be done prophylactically before cerebral infarction has occurred.
3. The period of operation is the most crucial in determining outcome. Serious operative complications varied from approximately 2% for the most successful groups to over 20% for the least successful.
4. The first cohort to demonstrate statistically significant improvement following carotid endarterectomy consisted of patients with mild neurologic deficit and corrected carotid stenosis who enjoyed improved survival compared to medically treated patients.
5. While the high average surgical complication rates obscured the possibly beneficial effects of surgical intervention on late follow-up, it was clearly demonstrated that unscathed survivors of carotid endarterectomy had decided protection against suffering future stroke when compared to the medically treated groups, and those with the most advanced arterial involvement, namely bilateral carotid stenoses or stenoses opposite occlusions, fared best following successful operations.
6. Vertebrobasilar symptoms were frequently relieved by carotid endarterectomy, and so, many groups ceased to operate upon the vertebral arteries. Additionally, there was no agreement as to which of a number of operations might be best to relieve the most commonly encountered lesions at the vertebral origin from the subclavian.
7. Subclavian steal rarely required operation.
8. The major deficiency of the study was in not stratifying patients according to degrees of arterial stenosis, the major emphasis being on clinical status.

Many other facts emerged from the study based upon 4,776 protocols, among which was the realization that cerebral hemorrhage could result from carotid endarterectomy and that complete internal carotid occlusions could rarely be surgically relieved. Yet there emerged no unanimity of opinion regarding how best to perform the operation either as to anesthesia, monitoring techniques, protective measures, or even how to surgically deal with the artery: short versus long arteriotomy versus eversion internal carotid endarterectomy; primary closure versus patch closure. The pathogenesis of the arterial lesions and indeed the components of carotid plaques were not well recorded, so that the mechanisms leading to cerebral ischemia and stroke, unless internal carotid thrombosis occurred, were usually not analyzed.

What has occurred since? In spite of the more recent recognition of the prominence of intracarotid plaque hemorrhage in symptomatic patients,[34,35] the findings in 1972 of small emboli in retinal arteries, although most often seemingly composed of minute yellow particles thought to be cholesterol particles,[36] led to a prospective randomized study of the effects of antiplatelet agents in patients with transient cerebral episodes in stopping attacks and preventing strokes. We participated in this

study using aspirin and sulfinpyrazone in both nonoperated patients and in patients post-carotid endarterectomy. The results were equivocal for stroke prevention.[37] Nevertheless, 10 additional studies have been done with equally equivocal results.[38] Was this not predictable, given the facts that had already been learned from the pathologic findings at the carotid bifurcations? Curiously, however, there is a rather clearcut effect of antiplatelets on death from coronary artery disease, only equivocal benefit on stroke incidence, and some beneficial effect on TIAs.

Probably as significant as any development in carotid surgery was the recognition in the '70s and '80s that intraoperative strokes, which occurred with such a widely disparate incidence regardless of what monitoring or protective measures were employed, were due to a variety of causes and that, indeed, different surgical groups did not share the same incidences of causes.[39,40] I well remember during the decade of the '70s interviewing prospective applicants for our vascular trainee position and asking the innocent question, "What does your professor tell you is the cause of stroke in the patient who unfortunately awakens from general anesthesia post-carotid endarterectomy?" Invariably the answer was: "embolization." Unable to resist the urge to foster skepticism, the counter was, "How do we know that there had not been rethrombosis of the artery, or that clamping ischemia had not occurred, or that intracerebral hemorrhage had not been precipitated as a reperfusion injury?"

If for no other reason than the fact that it was possible to differentiate intraoperative cerebral embolization from clamping ischemia, the not infrequently criticized practice of doing our operations under local anesthesia was continued. By employing various monitoring techniques and protective measures in conscious patients, it was possible to learn very early the pitfalls of relying upon jugular venous oxygen saturations; electroencephalography; carotid stump pressures (originally said to be safe at 25 Torr but subsequently increased to higher levels); routine shunting; the intracerebral patterns of cross-fill on the angiogram; and preoperative carotid compression testing in the upright or semirecumbent patient. False positives and false negatives occurred with all those techniques.[41–52]

There was a curious resistance on the part of the medical community to accept the fact that intraplaque hemorrhage was a prominent event in the evolution of the carotid plaque, and that it appeared to be a primary event following the myointimal proliferation that resulted from the flow peculiarities demonstrated to occur at the carotid bifurcation,[53,54] rather than a secondary phenomenon, secondary to degeneration and breakdown of a fat-laden plaque. The rebuttals to the claims of primary intraplaque hemorrhage came from those pathologists who claimed prior knowledge and insisted that the clotted blood found within fibrous plaques was dissection of the flowing column of blood into the wall of the plaque, either through grossly visible fissures or, if they were not seen, through micro-

scopic cracks. Indeed, there were some who said they found no blood within plaques while others said that it was created by the surgical manipulations. Yet Paterson had described the phenomenon of primary intraplaque hemorrhage in coronary arteries in 1936.[55]

How exciting to be given our wonderful noninvasive tools for examining both the arteries and the brain in our patients: ultrasound to measure luminal diameters and to provide images; computerized axial tomography and MRI to look at the brain and the arteries; PET scanning to study details of metabolism; and pathologic anatomy of the brain. Each has helped to confirm the essential facts and concepts deduced from previous clinical and pathological studies. It has been possible to establish the threatening nature of highly stenotic plaques by various sequential noninvasive tests of the carotid bifurcations (Gee, Kartchner, Strandness);[56–58] to establish the importance of plaque softening by hemorrhage and by its probable degeneration to atheromatous debris, the "cholesterol abscess;" and to confirm the concept of massive embolization of encysted blood or atheromatous material by noninvasive techniques,[59–62] such as those used by Norris, who demonstrated what was initially termed "spontaneous regression of plaques," better interpreted as embolization of large segments of plaque.

The insidious onset of ischemic brain syndromes was originally recognized by the organizers of the Joint Study, who insisted that even patients with nonlocalizing symptoms should be evaluated and included in the study if the appropriate arterial lesions were encountered. CT and PET scanning have shown that areas of the brain can be damaged by ischemia without occurrence of focal symptoms either by repeated embolization or through "watershed" ischemia. Is there now not recognition of vascular dementia?[63–67] And yet, there occurred disturbing rumbles in the country during the decade of the '80s. Undoubtedly spurred by poor surgical operative results, discovered in various parts of the U.S., and perhaps encouraged by the tantalizingly almost favorable results of antiplatelet agent studies, there arose "expressions of concern",[68] usually on the part of neurologists, who were skeptical that localized surgical intervention on the arterial system diffusely involved by atherosclerosis could be beneficial, who felt that the available data regarding the beneficial effects of carotid endarterectomy obtainable from nonrandomized trials was unconvincing,[69] and who were alarmed by some of the reports pertaining to surgical complication rates. The surgical results reported by the most successful groups were said not to be representative of what prevailed throughout the country, and anyway, surgeons tend to select the most favorable cases to operate, thereby skewing results in favor of surgery.

The RAND Organization study of complications of carotid endarterectomy in "representative" geographic areas,[70] reports of "community hospital experience",[71] and the skeptism of nonsurgical medical groups in

believing that self-interested surgeons could accurately report surgical results led to a marked decline in carotid endarterectomy operations in the latter part of the '80s.

The failure of the Extracranial/Intracranial By-Pass Randomized Clinical Trial in 1988 to show a definite beneficial effect in patients with internal carotid occlusion further spurred the negativism toward surgical intervention.[72] Yet that trial was flawed at its onset when, during its organizational phases, there was a reluctance to accept the proviso that EC-IC bypass be done only on patients with internal carotid occlusions in whom all other extracranial lesions had been corrected yet had persistence of ischemic symptoms.

In this climate of negativism and skepticism regarding favorable outcome for surgical intervention, five prospective randomized clinical trials of carotid endarterectomy versus medical treatment were organized, three in the U.S., two in Europe. Criteria for selection of patients for randomization differed markedly and ranged from asymptomatic marked carotid stenosis to any degree of carotid stenosis greater than 30% diameter limitation appropriate to clearly recognizable transient cerebral ischemic symptoms. Only one, however, specifies a clearcut irrefutable end point, stroke, the others permitting crossover to surgical treatment of patients randomized to medical therapy who suffer TIAs. Additionally, TIA is considered to represent a therapeutic failure.

Two of the studies have clearly confirmed what has been apparent through previous clinical pathological correlations and studies of cohorts of patients found to have carotid lesions, namely, that stroke risk is related to the stage of carotid pathology.

The North American Symptomatic Carotid Endarterectomy Trial, for which I have been on the Data Monitoring Committee, has conclusively shown after only 18 months of observation that patients who have had focal cerebral hemispheric symptoms and appropriate 70% to 90% carotid bifurcation stenosis and who underwent operation with approximately a 5% operative complication rate fared better than randomized nonoperated patients, so that that phase of the trial dealing with tight stenoses has been discontinued. Groups with lesser degrees of stenosis remain on trial. The European Carotid Endarterectomy Trial has reached identical conclusions.

The Veterans Administration Asymptomatic Carotid Endarterectomy Trial, for which I have also served as a data monitor, is nearly completed. Admission to the trial was dependent upon the existence of 50% or greater diameter reduction at the carotid bifurcation in asymptomatic patients. Unfortunately, results will be diluted by the fact that TIA, stroke or death are end points, TIA being considered a "soft" end point. Nevertheless, the results of that trial, which I cannot reveal, should be announced before the end of this year.

What can be concluded from the available information, voluminous as it is, as it pertains to the management of patients with extracranial cerebro-

vascular occlusive disease? In general, the concept that advanced pathologic changes at the carotid bifurcation produce cerebral ischemia by embolization of microparticles or of large segments of the arterial wall and by flow impairment is well established. Marked stenosis manifested by 70% or greater diameter constriction is the best current marker of the patient at risk of stroke. This is evident from a number of sources, including pathologic study of surgically excised plaques, from sequential noninvasive arterial studies of patients with carotid stenoses, from the recently completed portions of two randomized prospective clinical trials of carotid endarterectomy, as well as from a number of nonrandomized clinical surgical studies. The concept of symptomatic versus asymptomatic in the presence of advanced arterial stenosis is probably not valid and is evident from the reported incidence of stroke without premonitory TIA in patients with advanced carotid stenosis and from the incidence of "silent" brain infarctions found on brain CT scans in patients with carotid lesions. This last observation is not surprising since there are areas of the brain clinically relatively silent, while the entire brain is silent during the eight hours or so when one is asleep.[73] This further confirms the validity of the JSEAO, wherein admission to the study was for even vague and ill-defined symptoms when found in association with significant arterial lesions.

Indeed, the advanced stage of the arterial lesion has been the stimulus to perform endarterectomy even in patients with acute strokes, provided the infarct size involved only a portion of a hemisphere and the patient had not lost consciousness, while observing certain precautions at operation, including employing general anesthesia and routing intraluminal shunting.[74,75]

What, one might ask, should we do about the contralateral asymptomatic lesion; about combined carotid and coronary lesions,[76–79] aneurysms and carotids? The answers at the moment would appear to be that the carotid arterial lesion and its degree of stenosis represent the overriding factor in deciding whether carotid endarterectomy should be done. If sufficiently stenotic it represents a risk and should be operated upon. The controversial aspect of management is with regard to the proper sequence of operations. Our own bias is to perform carotid endarterectomy under cervical block anesthesia, an anesthetic technique for which claim is made that cardiac risk is minimal compared to carotid endarterectomy performed under general anesthesia, and proceed with either coronary artery surgery or abdominal aortic aneurysm surgery, preferably the next day, rarely on the same day.

Many challenges exist for the future and range from how to make the operation of carotid endarterectomy safe in the hands of most surgeons trained to do vascular operations,[80–82] to refining the selection of patients for operation,[83–86] to acquiring a better understanding of the evolution of carotid plaques and of the precise mechanisms by which cerebral ischemia is produced, including the role of blood rheology.[87–93]

The immediate challenge relates to surgical management. Although many claim superior results employing varieties of techniques, the anatomy, pathology and physiology involved in performing carotid endarterectomy are the determinants of what will succeed and what will not. Many variables are involved, any of which may determine outcome: extent of surgical exposure of the carotid triangle required for a safe operation; length of the arteriotomy; specific plaque resection technique used; management of end points, redundancies and kinks; arteriotomy closure technique, whether primary or with roof patch of either autologous vein or prosthetic material; method of inlying shunt insertion and the particular shunt to be used. Indeed, there are those who insist that only completion angiography is a satisfactory determinant of whether a proper operation has been done. The marked variability of surgical results reported even from highly experienced groups indicates that more than frequent repetition of the procedure is required to achieve excellence.

An additional immediate challenge deals with the relatively unsatisfactory methods of selecting which arteries merit surgical intervention based upon degrees of stenosis. The same films, examined by different individuals using different or even the same criteria for estimating stenosis, result in sometimes widely varying estimates. Unfortunately, although repeatedly recommended in surgical publications, no one of the known prospective randomized clinical trials included the recording, photographically, of carotid plaques in situ for subsequent comparison to angiograms and to histologic sections. We are left now with the relatively vague concept that plaques echogenically "soft" are dangerous, while those echogenically hard are less so. A more precise estimate of the contents of any plaque, correlated with clinical course, is needed to eliminate a large element of subjectiveness and error involved in relying upon the relatively crude measurement of stenosis as the major criterion for whether the brain needs to be protected from undergoing infarction.

"Watershed" infarcts are being recognized with increasing frequency. Although most internal carotid occlusions cannot be reopened because of the propagation of thrombus to the inaccessible portions of the internal carotid artery, they do pose the risk of future stroke even while asymptomatic. There are, however, a significant number of total occlusions that start at the origin of the internal carotid, but which, as late as one month after onset, remain easily operable since thrombus has not reached the intracranial carotid. Duplex scanning, CT scans with contrast, and NMR all merit investigation to determine reliability in revealing such operable situations. If not operable, does extracranial-intracranial bypass merit additional evaluation in those in whom all other lesions have been corrected?

Beyond the immediate challenges expressed are those which delve into the pathogenesis of cerebral ischemia. Will NMR and PET scanning of the brain help to identify carotid arterial lesions that have already produced

subtle brain abnormalities even before advanced stenoses have occurred? Such a protocol was written nearly eight years ago as a proposal for the Javits Center of Neurologic Excellence but it was rejected as frivolous. Yet such techniques must be developed to identify truly dangerous plaques before severe and irreversible brain damage occurs.

Few surgical groups deal with vertebral arterial lesions, although they are the second most commonly found arterial lesions, second only to carotid lesions. Hutchinson and Yates defined the role of vertebral lesions in contributing to death of patients with strokes when associated with carotid lesions. Our own concern with uncorrected vertebral lesions has been, aside from the production of incapacitating symptoms, with their possible role in the syndrome of sudden unexplained death, since 23 of our patients suffered bouts of syncope as their predominant symptom associated with bilateral vertebral origin stenoses. Syncopal attacks were relieved by restoring flow to one vertebral.[94–96]

There are many other issues for the future. It is not possible to visualize how any percutaneously introduced intraarterial device could remove carotid plaques with the same degree of finesse and safety as skillfully performed open carotid endarterectomy, which now carries operative risks of 1%–2%. It is difficult to justify limiting the field of study of stroke-threatened patients to the carotid bifurcation with ultrasonic devices when it can be shown by comparative studies that although immediate catastrophes because of limited studies occur infrequently, nevertheless, errors of omission and commission can occur in as many as 8% of patients operated on the basis of duplex scanning of the neck alone.

In terms of pathogenesis of the carotid plaque, a burning question that at present suggests only a number of divergent answers is, "What causes the intrafibrous plaque hemorrhage?" Myointimal thickening in response to flow anomalies at the carotid bifurcation occurs universally. Were it to progress as such, as it does in some, to sufficient stenosis resulting in thrombosis there would be few strokes. When hemorrhage occurs within such fibrous plaques, the threatening nature of the plaque becomes manifest. Continued hemodynamic effect? Inflammatory intraplaque vascularity? "Cement substance" deficiency secondary to vitamin C deficiency??

In parting, it is vital to remember that the truly significant advances in the effort to control the ischemic stroke problem have come from clinicians. Clinical pathological correlations, observations of the pathology in situ in living patients, diagnostic devices, clinical trials of medical and surgical treatments, and development of safe surgical techniques have all been by clinicians. The brilliant ancillary devices that have facilitated study of living patients, ranging from ultrasound equipment to CT, NMR and PET scanning equipment, have been instrumental in confirming concepts derived from clinical observations. The challenge to continue to contribute as clinicians persists.

References

1. Friedman SG. *A History of Vascular Surgery*. Futura Publishing Co. Inc. Mount Kisco, NY 1989.
2. Webster's Dictionary—20th Century—Unabridged—2nd Ed. The World Publishing Co. Cleveland & New York, 1970.
3. Toole JF. *Cerebrovascular Disorders*—Third Edition. Raven Press. NY 1984.
4. Friedlander WJ. About 3 old men: An inquiry into how cerebral arteriosclerosis has altered world politics. A neurologist's view. (Woodrow Wilson, Paul von Hindenburg and Nikolai Lenin) Stroke 3:467, 1972.
5. Robertson CW. Some observations on presidential illnesses. Boston, Med Q 8:33,76, 1957.
6. King David. The Old Testament. Psalms 137:5 (1050 B.C.)
7. McHenry LC Jr. A history of stroke. In *Cerebral Circulation and Stroke*. Ed. McHenry, LC Jr. Warren H Green, Inc. St. Louis, MO 1978, p. 1.
8. Chiari H. Uebger das verhalten des teilungswinkels de carotid communis bei der endarreritis chronica deformans. Verh Tdach Pathol Ges 9:736, 1905.
9. Hunt JR. The role of the carotid arteries in the causation of vascular lesions of the brain, with remarks on certain special features of the symptomatology. Am J Med Sci 147:704, 1914.
10. Fisher CM. Occlusion of the internal carotid artery. Arch Neurol Psych 65: 346, 1951.
11. Fisher CM. Occlusion of the carotid arteries. Arch Neurol Psych 72:187, 1954.
12. Carrea R, Molins M, Murphy G. Surgical treatment of spontaneous thrombosis of the internal carotid artery in the neck. Carotid-carotideal anastamosis. Acta Neurol Latinoamer 1:71, 1955.
13. Strully KH, Hurwitt ES, Blankenberg HW. Thromboendarterectomy for thrombosis of the internal carotid artery in the neck. J Neurosurg 10:474, 1953.
14. DeBakey ME. Successful carotid endarterectomy for cerebrovascular insufficiency: Nineteen year follow-up. J.A.M.A. 233:1083, 1975.
15. Eastcott HHG, Pickering GW, Rob CG. Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. Lancet, ii, 994. 1954.
16. Denman FR, Ehni G, Duty WS. Insidious thrombotic occlusion of cervical carotid arteries, treated by arterial graft: Case report. Surgery 38:569, 1955.
17. Lin PM, Javid H, Doyle EJ. Partial internal carotid artery occlusion treated by primary resection and vein graft. J Neurosurg 13:650, 1956.
18. Dos Santos JC. Sur la desobstruction des thromboses arterielles anciennes. Mem Acad Chir 73:409, 1947.
19. Thompson JE. History of carotid artery surgery. Surg Clin NA 66:225, 1986.
20. Fein JM. A history of cerebrovascular disease and its surgical management. In *Stroke and the Extracranial Vessels*. Ed. Smith RR: Raven Press. NY.
21. *The Classics of Vascular Surgery*. Ed. Rob CG, *The Classics of Surgery Library*—Series Eds. Reentsma L, Schwartz, SI. Apollo Press, Medford, N.J.
22. Eastcott HHG. The beginning of carotid surgery. In *Cerebrovascular Insufficiency*. Ed. Bergan JJ, Yao JST: Grune & Stratton, NY 1983 p. 3–16.
23. Mark MF, Imparato AM, Hutner SH, Baker H. Estimate of toxicity of radiopaque agents by means of a ciliate. Angiography 14:383, 1961.

24. Lyons C, Galbraith G. Surgical treatment of atherosclerotic occlusion of the internal carotid artery. Ann Surg 146:487, 1957.
25. Thompson JE, Austin DJ. Surgical treatment of arteriosclerotic occlusion of carotid arteries in the neck. Surg 51:74–83, 1962.
26. Wylie EJ. Cerebrovascular insufficiency caused by extracranial arteriosclerotic disease. Trans pas Coast Otoophthal Soc 41:1–11, 196.
27. DeBakey ME, Crawford ES, Morris JC, Cooley DA. Arterial reconstructive operations for cerebrovascular insufficiency due to extracranial occlusive arterial disease. J Cardiovasc Surg 3:12–25, 1962.
28. Fields WS, Bruetman ME, Weibel J. Collateral circulation of the brain. Monogr Surg Sci 2:183–259, 1965.
29. I. Fields WS, North RR. Organization of study and survey of patient population. J.A.M.A. 203:955, 1968.
30. II. Hass WK, Fields WS. Arteriography, techniques, sites and complications. J.A.M.A. 203:961, 1968.
31. III. Bauer RB, Meyer JS. Progress report of controlled long-term survival in patients with and without operation. J.A.M.A. 208:509, 1969.
32. IV. Blaisdell WF, Clauss RH, Galbraith JG, Imparato AM, Wylie EJ. A review of surgical considerations. J.A.M.A. 209:1889, 1969.
33. V. Fields WS, Maslenikof V. Progress report of prognosis following surgery or non-surgical treatment for transient cerebral ischemic attacks and cervical carotid lesions. J.A.M.A. 211:1993, 197.
34. Imparato AM, Riles TS, Gorstein F. The carotid bifurcation plaque: Pathologic findings associated with cerebral ischemia. Stroke 10:238, 1974.
35. Imparato AM, Baumann G. The significance of gross hemorrhage in carotid plaque. Ann Surg 197:195, 1983.
36. Hollenhorst RW. Significance of bright plaques in the retinal arterioles. J.A.M.A. 178:2, 1961.
37. Fields WS, Lemak NA. Controlled trial of aspirin in cerebral ischemia. Stroke 8:53, 1977.
38. Sze PC, Pincus M, Chalmers TC et al. Antiplatelet agents in secondary prevention of stroke: Meta-analysis of the randomized control trials. Stroke 19:436, 1988.
39. Imparato AM, Ramirez A. Cerebral protection in carotid surgery. Arch Surg 117:1073, 1982.
40. Hart R, Hindeman B. Mechanisms of perioperative cerebral infarction. Stroke 13: 776, 1982.
41. Green RM, Messick WJ. Benefits, shortcomings, and costs of EEG monitoring. Ann Surg 201:785, 1985.
42. Baker DJ, Gluecklich BB. An evaluation of electroencephalographic monitoring for carotid study. Surgery 78:787, 19.
43. Hobson RW, Wright CB, Sublett JW, et al. Carotid artery back pressure and endarterectomy under regional anesthesia. Arch Surg 109:682, 1974.
44. Rich NM, Hobson RW. Carotid endarterectomy under regional anesthesia. Am J Surg 41:253, 1975.
45. Horsch S. Intraoperative assessment of cerebral ischemia during carotid surgery. J Cardiovasc Surg 41:599, 1990.
46. Hays RJ, Levinson SA, Wylie EJ. Intraoperative measurement of carotid back

pressure as a guide to operative management for carotid endarterectomy. Surgery 72:953, 1972.
47. Strawn DJ, Hunter JC. The relationship of intraluminal shunting to technical results of carotid endarterectomy. J Cardiovasc Surg 31:429, 1990.
48. Baker WH, Littooy FN. Carotid endarterectomy without a shunt: The control series. J Vasc Surg 1:50, 1984.
49. Bloodwell RD, Hallman GL, Keats AS et al. Carotid endarterectomy without a shunt: Results using hypercarbic general anesthesia to prevent cerebral ischemia. Arch Surg 96:644, 1968.
50. Imparato AM. Local anaesthesia for carotid endarterectomy. In *Current Therapy in Vascular Surgery*. BC Decker. 1987 p. 33.
51. Haffner CD, Evans WE. Carotid endarterectomy with local anaesthesia: Results and advantages. J Vasc Surg 7:232, 1988.
52. Thompson JE. Complications of carotid endarterectomy and their prevention. World J Surg 3:155, 1979.
53. Imparato AM. The "major" and "minor" carotid artery in arterial reconstruction. Stroke IX:15, 197.
54. Zarins CK, Giddens DP. Carotid bifurcation atherosclerosis: Quantitative correlation of plaque localization with flow velocity profiles and wall shear stress. Circ Res 53:502, 1983.
55. Paterson JC. Capillary rupture with intimal hemorrhage as a causative factor in coronary thrombosis. A Pathol 25:474, 1938.
56. Gee W, Mehigan JI, Wylie EJ. Measurement of collateral hemispheric blood flow by ocular pneumoplethysmography. Am J Surg 130:121, 1975.
57. Kartchner MM, McCrae LO. Non-invasive evaluation and management of the asymptomatic carotid bruit. Surgery 82:840, 1977.
58. Roederer GO, Langlois YE. The natural history of carotid arterial disease in asymptomatic patients with carotid bruits. Stroke 15:605, 1984.
59. Glover JL, Bendick PJ. Duplex ultrasonography, digital subtraction angiography in assessing carotid atherosclerosis. Arch Surg 119:664, 1984.
60. O'Donnel TF, Erdoes L. Correlation of B-mode ultrasound imaging and arteriography with pathologic findings at carotid endarterectomy. Arch Surg 120:443, 1985.
61. Gray-Weale AC, Graham JC, Burnett JR, Byrne K, Lusby RJ. Carotid artery atheroma: Comparison of B-mode ultrasound appearance with carotid endarterectomy specimen pathology. J Cardiovasc Surg 29:676, 1988.
62. Gomez CR. Carotid plaque morphology and risk for stroke. Stroke 21:148, 1990.
63. Sette G, Baron JC, Mazoyer B, Levasseur M, Pappata S, Crouzel C. Local brain hemodynamics and oxygen metabolism in cerebrovascular disease: Positron emission tomography. Brain 112:931, 1989.
64. Yanagihara T, March WR, Piepgras DG, Ivnik RJ. Dementia in bilateral carotid occlusive disease. Stroke 21 Suppl. to 8, 199, 1990.
65. Hershey LA. Dementia associated with stroke. Stroke 21(Suppl. II):II-9-II-11, 1990.
66. Nicolaides AM, Papadakis K, Gregg M, et al. The significance of silent cerebral infarction and atrophy. In Amaurosis Fugax Ed. Bernstein F Springer Verlag. New York, 1987.

67. Millikan C, Futrell N. The fallacy of the Lacune hypothesis. Stroke 21:1251, 1990.
68. Barnett JHM, Plus F, Walton JN. Carotid endarterectomy: An expression of concern. Stroke 15:941, 1984.
69. Dyken ML. Carotid endarterectomy studies: A glimmering of science. Editorial. Stroke 17:355, 1986.
70. Winslow CM, Solomon DH, Chassin MR, Kosecoff J, Brook RH. The appropriateness of carotid endarterectomy. N Engl J Med 31:721, 1988.
71. Modi JR, Finch WT, Sumner DS. Update of carotid endarterectomy in two community hospitals: Springfield revisited. Stroke 14:128, 1983.
72. EC-IC Bypass Study Group. Failure of extracranial-intracranial arterial bypass to reduce the risk of ischemic stroke: Results of an international randomized trial by the EG-IC bypass study group. N Engl J Med 313:1191, 1985.
73. Bruno A, Biller J, Adams HP, Corbett JJ. Retinal infarction during sleep and wakefulness. Stroke 21:1494, 1990.
74. Goldstone J, Effeney DJ. The role of carotid endarterectomy in the treatment of acute neurologic deficits. Prog Cardiovasc Dis 22:415, 1980.
75. Whittemore AD, Mannick JA. Surgical treatment of carotid disease in patients with neurologic deficits. J Vasc Surg 5:910, 1987.
76. Bernhard VM, Johnson WD, Peterson JJ. Carotid artery stenosis: Association with surgery for coronary artery disease. Arch Surg 105:837, 1972.
77. Berkoff HA. Turnipseed WD. Patient selection and results of simultaneous coronary and carotid artery procedures. Ann Thor Surg 38:172, 1984.
78. Emery RW, Cohn LH, Whittemore AD, Mannick JA, Couch NP, Collins JJ. Coexistent carotid and coronary artery disease. Arch Surg 118:1035, 1983.
79. Riles TS, Kopelman I, Imparato AM. Myocardial infarction following carotid endarterectomy. Surg 85:249, 1979.
80. Whisnant JP. Does carotid endarterectomy decrease stroke and death in patients with transient ischemic attacks? Ann Neurol 22:72, 1987.
81. Imparato AM. Surgery for cerebrovascular insufficiency. In Ransohoff J Ed. 63. Sette G, Baron JC, Mazoyer B, Levasseur M, Pappata S, Crouzel C. Local lishing Co. 1980 p. 90.
82. Imparato AM. The role of patch angioplasty after carotid endarterectomy. J Vasc Surg 7:715, 1988.
83. Arnolds BJ, von Keutern GM. Transcranial dopplersonography examination technique and normal reference values. Ultrasound in Med and Biol 12:115, 1986.
84. Halsey JH, McDowell HA, Gelman S. Transcranial doppler and rCBF compared in carotid endarterectomy. Stroke 17:1206, 1986.
85. Moore WS, Ziomek S, Quinones-Baldrich WJ, Machleder HI, et al. Can clinical evaluation and non-invasive testing substitute for arteriography in the evaluation of carotid disease? Ann Surg 208:91, 1988.
86. Barnes RW, Nix ML. Late outcome of untreated asymptomatic carotid disease following cardiovascular operations. J Vasc Surg 2:843, 1985.
87. Imparato AM. The carotid bifurcation plaque: A model for the study of atherosclerosis J Vasc Surg 3:249, 1986.
88. Fisher M, Freman S. Geometric factors of the bifurcation in carotid atherogenesis. Stroke 21:267, 1990.

89. Imparato AM, Baumann FG. Electron microscopic studies of fibromuscular intimal arterial lesions experimentally produced. Surg Gynec Obst 139:494, 1974.
90. Hart RG, Kanter MD. Hematologic disorders and ischemic stroke: A selective review. Stroke 21:1111, 1990.
91. Rajiv J, Riddle JM, Welch MD, D'Andrea G. Platelet ultrastructure and secretion in acute ischemic stroke. Stroke 20:1316, 1989.
92. Israelson B, Brattstrom LE, Hultberg BL. Homocysteine and myocardial infarction. Atherosclerosis 71:227, 1988.
93. Tell GS, Howard G, McKinney WM, Toole JF. Cigarette smoking cessation and intracranial carotid atherosclerosis. J.A.M.A. 261:1178, 1989.
94. Humphries AW, Young JR. Relief of vertebrobasilar symptoms by carotid endarterectomy. Surg 57:48, 1965.
95. Hutchinson EC, Yates PO. Carotico vertebral stenosis. Lancet i,2, 1957.
96. Imparato AM. Vertebral arterial reconstruction: A nineteen year experience. J Vasc Surg 2:625, 1985.

4
Carotid Endarterectomy: Indications and Techniques

ALLAN D. CALLOW

"The eventual definition of the precise role of surgery in the management of cerebral vascular insufficiency must await the outcome of further studies, but it may be safely said at this time that surgical measures have already demonstrated their effectiveness in many patients with stroke syndromes."[1] Jesse Thompson, a most distinguished carotid surgeon, wrote this in 1968. Nearly a quarter of a century had to pass before official accreditation was bestowed.[2] During this time, rejection of surgical therapy among many physicians ranged from simple withholding of acceptance to stifling opposition. Largely because of the conviction among most vascular and neurosurgeons, based on their personal experience, that carotid endarterectomy was a beneficial and durable procedure for the prevention of carotid territory stroke, the number of operations increased each year. Approximately 17,000 were performed in 1977 and over 100,000 in 1984 in the United States alone. From 1954 to 1990 the worldwide number of carotid endarterectomies was estimated as approximately one million. Although excellent results in terms of operative morbidity and mortality as well as duration of stroke prevention were published,[3–7] many reports were less salutary. This was particularly true in some series dealing with the asymptomatic carotid lesion with clearly unacceptably high rates of perioperative stroke and death.[8–15] Confusion as to the proper indications for and the true utility of carotid endarterectomy increased in the medical and lay communities, and the need for controlled randomized studies became apparent.[16–18]

A Clinical Alert was issued in March of 1991 as the consequence of an interim review of data from the North American Symptomatic Carotid Endarterectomy Study.[2] Carotid endarterectomy was found to be clearly superior to nonsurgical therapy, consisting of antiplatelet agents, among symptomatic male and female patients with 70–99% stenosis of the internal carotid artery. Acquisition of patients to this study group was halted. The earlier claims of vascular and neurological surgeons, albeit based on nonrandomized retrospective experience, were at long last now confirmed.

From a review of the history of carotid endarterectomy and from the

TABLE 4.1. Recommended limits: perioperative mortality/stroke morbidity.*

Mortality	Limit
All operations/causes	2%
Stroke	
Asymptomatic	3%
Transient ischemic attack	5%
Ischemic stroke	7%
Recurrent disease	10%

*From the Committee on Carotid Surgery Standards, American Heart Association.

NASCES trial, two additional items of enormous importance deserve emphasis: (1) excellent surgical results require a meticulous surgical technique supported by a committed team of specialists in neurology, internal medicine, cardiology, anesthesiology, and angiography; and (2) rigorous adherence to a detailed protocol for patient selection. Carotid endarterectomy can be justified only if it results in better long-term stroke prevention than does medical management. Case reports with high perioperative stroke rates and limited long-term benefit represent more an indictment of those individual practice patterns than of the usefulness of the operation itself.

The Ad Hoc Committee on Carotid Surgery Standards of the Stroke Council of the American Heart Association has published maximal acceptable perioperative mortality and morbidity rates according to specific clinical categories[19] (Table 4.1):

In our literature review of surgical series reporting good to excellent results, certain common practice patterns became apparent[19] (Table 4.2).

Chief among important determinants of outcome were: (1) patient selection; (2) anesthesia evaluation and management; and (3) operative technique.

Common to all series reporting minimal perioperative stroke morbidity, often below those rates suggested by the Committee on Carotid Surgery

TABLE 4.2. Optimal perioperative mortality and stroke morbidity.

	Perioperative	
Report	Mortality	Stroke
Callow and Mackey (3)	0.6%	1.6%
Healy (4)	0.5%	2.0%
Hertzer (5)		
Symptomatic group	0%	1.2%
Asymptomatic group	1.1%	3 4%
Hafner (26)	0.7%	0.9%

Standards of the American Heart Association, are the procedures described below.

A Generous Arteriotomy

Direct visualization of the distal end point of the disease must be obtained because a perceptible ridge of tissue extending into the internal carotid artery after a major portion of the plaque has been removed serves as a nidus for platelet aggregation, thrombus formation and embolization.

Placement of so-called tacking sutures, although allegedly lessening the likelihood of dissection or elevation of the plaque by the pulsing column of blood, does not eliminate the chance of thrombosis and embolization. In addition, it is conceivable that atherosclerotic progression within the residual disease remnant is accelerated by the surgical trauma of the endarterectomy. Enhancement of residual atherosclerosis may also occur at the proximal end of the endarterectomy site in the common carotid artery. Thus, what is often interpreted as recurrent stenosis may in truth be acceleration of residual disease.

"Feathering" of the distal end of the plaque is essential. Although it can be accomplished with a shunt in place, it is more difficult and time consuming, particularly with a high lying, distally extending plaque, than when visualization is not shunt obscured.

A meticulous and complete surgical procedure includes bright light illumination, magnification, exposure of the normal internal carotid artery above the distal extent of disease, an endarterectomy dissection plane beneath the plaque and deep to the media, and removal of all "floaters" aided by copious irrigation of the endarterectomy field. The arteriotomy closure must be meticulous, avoiding eversion of the edges, and butting them together with sutures close to the edge and close to each other. We limit the use of a patch to those operations where, for one reason or another, immediate reopening of the arteriotomy site is required, reoperation for recurrent carotid stenosis is required and, in a very small number of patients, when the internal carotid artery is unusually small. We are not convinced that the incidence of recurrent stenosis is reduced by routine use of a patch. When one is needed we prefer the proximal greater saphenous vein.

Cerebral Protection

Excellent results can be obtained with a variety of techniques of cerebral protection, illustrating again that it is probably more a matter of the meticulousness with which a given technique is applied than any special merits of one particular technique over another. Our preference is for general

anesthesia under normocarbic and normotensive conditions with selective shunting based on EEG monitoring. We have done many patients awake with regional block anesthesia and have also utilized carotid back pressure to assess the need for a shunt. In our experience the most reliable method is continuous EEG monitoring utilizing 20 electrodes cemented to the scalp and with 10 leads to the recording console. There is approximately a 10–15 percent error in the correlation between back pressure and cerebral perfusion when checked against the electroencephalogram. This inconsistency has been displayed on both sides of the equation, i.e., a better perfusion with a low back pressure than would have been anticipated as well as EEG evidence of poor cerebral perfusion despite a so-called safe level back pressure. For high risk patients with both carotid and coronary disease, operating with the patient awake may add an element of safety.

Systemic heparin is administered before cross clamping. It is not reversed. Low molecular weight Dextran is administered prior to cross clamping and is continued for approximately 24 hours or until the patient can take aspirin by mouth. Neither the arteriogram nor the operation are deferred because the patient is on a maintenance dose. Prior to closure of the arteriotomy the operated segment is copiously irrigated with heparinized saline followed by Dextran diluted half and half with saline to avoid both air and thrombus embolization. The arteriotomy site is back bled by opening the distal or internal carotid artery clamp first. The external is also back bled into the arteriotomy site and then the common carotid is opened followed by the external branch. Should there be any remaining air or debris, it should enter the external carotid system. The internal carotid is opened last.

Completion arteriography, imperfect as it may be, is recommended after closure of the arteriotomy site. Its shortcomings are failure to give a reliable representation of the lumen in more than one plane and to identify minimal amounts of platelet, fibrin or red clot. Ultrasonography is also useful, but again, may fail to detect the minimal imperfection created by a small amount of clot or a strand of tissue floating in the lumen. Nevertheless, in view of the experiences of Blaisdell and Courbier,[20,21] whereby unsuspected imperfections of the endarterectomy site were much higher than anticipated, some form of assessment of the reconstruction is strongly recommended.

Peripheral Nerves

Although the normal position of the vagus nerve, with its accompanying recurrent laryngeal branch, is between the carotid artery and the internal jugular vein and posterolateral to the internal and common carotid arteries, it may occupy an anteromedial location in approximately 5% of individuals. The plane of dissection of the carotid must be close to the wall of the artery to avoid nerve injury. Vocal cord paresis follows upon trauma to

the vagus and the recurrent laryngeal nerves. Possibly as many as 10% of individuals display the recurrent laryngeal nerve in an anomalous nonrecurrent course. It fails to descend to the area of the clavicle but instead traverses behind the common carotid artery directly to the larynx. Dissection must also be kept close to the carotid bifurcation to avoid trauma of the superior laryngeal nerve, which, if damaged, could result in unilateral numbness of the orifice of the larynx, mild difficulty in swallowing and some change in voice quality due to cricothyroid muscle dysfunction. The hypoglossal nerve descends along the course of the internal carotid artery and passes medially over the external carotid in a more superficial position. This nerve, usually encountered during carotid dissection, is easily mobilized medially to expose the most distal reaches of the internal carotid artery. Trauma to the hypoglossal nerve results in paralysis of the ipsilateral side of the tongue, which if severe, may progress to atrophy. The ansa hypoglossus, branches of the hypoglossal nerve, takes various positions in the neck, frequently lies in the field, and can usually be easily mobilized medially. The ansa may fix the hypoglossal nerve in the operative field, and if this is the case, it may be sacrificed.[22]

Patient Selection

The Ideal Candidate

Second only in importance to meticulous operative technique for avoidance of perioperative complications is selection of the patient. The ideal candidate has an acceptable cardiac risk, has had one or more carotid territory TIAs, and demonstrates carotid disease appropriate to the involved cerebral territory. Using patient selection criteria similar to these, the North American Symptomatic Carotid Endarterectomy Trial (NASCET) demonstrated in a randomized prospective study that carotid endarterectomy carried a perioperative mortality/morbidity rate of only 3.4% at 30 days. This rate was 5.0% in patients treated with antiplatelet agents. At 12 months these figures were 6.5% and 17.8%, respectively. The study group consisted of patients of less than 80 years of age with a hemispheric TIA or a nondisabling stroke and angiographic evidence of 70–99% stenosis.[2] Further patient entry was stopped.

Patients at increased operative risk are those with: (1) a history of a previous stroke; (2) a positive CT scan with or without a history of a previous stroke; (3) coexisting vertebral basilar insufficiency signs and symptoms or angiographic evidence of disease; and (4) occlusion or severe stenosis of the contralateral carotid. These conditions are all indicative of reduced collateral circulatory reserve. During carotid artery cross clamping, approximately 25% of these patients will require an in-dwelling shunt as determined by EEG monitoring. Presumably patients with impaired cerebral vascular reserve are more vulnerable to intraoperative emboli or a

fall in cerebral perfusion pressure than are normal subjects. Positron emission tomography studies indicate that the best index of cerebral vascular reserve is the ratio of cerebral blood flow to cerebral blood volume.[23] In that study the significant risk factors for intraoperative neurologic deficit were: (1) age over 65 years; (2) a residual neurologic deficit prior to operation; (3) complex plaque morphology; and (4) the combination of impaired cerebral vascular reserve and a positive CT scan for infarct in the symptomatic hemisphere. Patients with these findings, however, can be brought through carotid endarterectomy quite successfully and with no higher incidence of perioperative neurologic deficit or death than patients in the ideal candidate group if great care is taken to identify the coexisting conditions and to utilize measures to avoid undue physiologic stress.

The High Risk Patient

These patients are those with an evolving stroke, a so-called crescendo stroke, or a waxing and waning of neurologic signs and symptoms. It is often difficult to determine whether the patient is undergoing a true stroke or merely suffering a prolonged episode of a transient ischemic attack. The patient with a completed stroke with substantial recovery but who has retained significant functioning carotid territory is also at high risk. Coexisting coronary artery disease is a frequent finding if careful search is made[24] (Fig. 4.1). In our experience some 53% of 614 patients either had overt coronary artery disease as determined by history or electrocardiographic studies, or covert disease discoverable upon careful search. The long-term survival of these patients is considerably reduced compared to those in whom no coronary artery disease was present or identifiable[25,26] (Fig. 4.2).

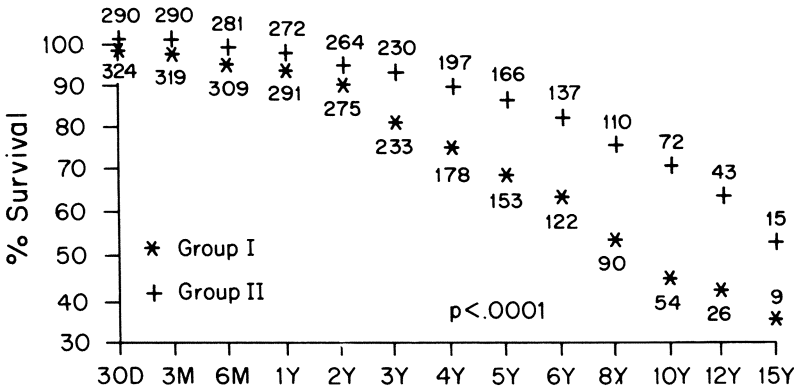


FIGURE 4.1. Life-table survival for patients with (group I) and without (group II) overt coronary disease.

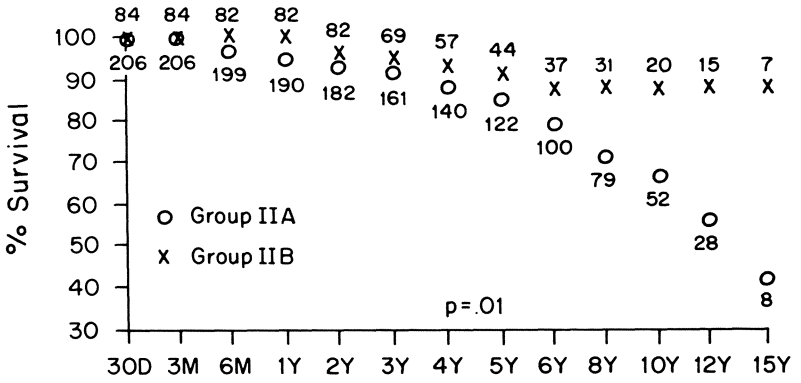


FIGURE 4.2. Life-table survival for patients without overt coronary disease but with (group IIA) and without (group IIB) risk factors (diabetes, cigarette use, hyperlipidemia).

Summary

Common to all series reporting excellent results in terms of perioperative morbidity and mortality and long-term duration of the benefit of carotid endarterectomy are certain items we have labeled shared practice patterns:

1. careful evaluation of the patient to identify, document and classify coexisting disease;
2. meticulous anesthesia and surgical technique with appropriate measures carefully applied to preserve cerebral perfusion or to detect its impairment during carotid cross clamping;
3. particular attention to the possibility of coexisting coronary artery disease and myocardial vulnerability;
4. avoidance of excess manipulation of the carotid bulb and the internal carotid artery because of the probable existence of fragile plaque and thrombus within the vessel;
5. avoidance of complete dissection of the common carotid, the bulb and the internal carotid from its bed on the basis that it is not only unnecessary but is additionally hazardous;
6. complete removal of the plaque and avoidance of tacking sutures in most endarterectomies;
7. for the surgeon with limited experience the use of a shunt and systemic heparinization are considerably safer than a hasty operation in an effort to reduce cross-clamp time;
8. proper first-time closure of the arteriotomy to avoid the need for even partial reclamping; and
9. checking of the patient's postoperative neurologic status by the re-

sponsible surgeon because a minimal neurologic deficit, a possible harbinger of stroke, may go undetected by someone else.

References

1. Thompson JE. Surgery for Cerebrovascular Insufficiency (Stroke). Charles C. Thomas, Springfield, IL, 1968.
2. North American Symptomatic Carotid Endarterectomy Study, Annual Conference on Stroke, American Heart Association, February, 1991.
3. Callow AD, Mackey WC. Long term follow-up of surgically managed carotid bifurcation atherosclerosis: Justification for an aggressive approach. Ann Surg 1989;210:308–316.
4. Healy DA, Clowes AW, Zierler RE, Nicholls SC, et al. Immediate and long-term results of carotid endarterectomy. Stroke 1989;20:1138–1142.
5. Hertzer NR, Arison R. Cumulative stroke and survival ten years after carotid endarterectomy. J Vasc Surg 1985;2:661–668.
6. Bernstein EF, Humber PB, Collins GM, Dilley RB, et al. Life expectancy and late stroke following carotid endarterectomy. Ann Surg 1982;19:80–86.
7. Stewart G, Ross-Russell RW, Browse NL. The long term results of carotid endarterectomy for transient ischemic attacks. J Vasc Surg 1986;4:600–605.
8. Dyken ML, Pokras R. The performance of endarterectomy for disease of the extracranial arteries of the head. Stroke 1984;15:948–50.
9. Chambers BR, Norris JW. The case against surgery for asymptomatic carotid stenosis. Stroke 1984;15:964–967.
10. Merrick NJ, Park RE, Kosecoff J, et al. A review of the literature and ratings for the appropriateness of indications for selected medical and surgical procedures: carotid endarterectomy. Santa Monica, CA: Rand Corporation. 1986 (Publication No. R-3280).
11. Committee on Health Care Issues. American Neurological Association. Does carotid endarterectomy decrease stroke and death in patients with transient ischemic attacks? Ann Neurol 1987;22:72–76.
12. Warlow C. Carotid endarterectomy: Does it work? Stroke 1984;15:1068–1076.
13. Easton JD, Sherman DG. Stroke and mortality rate in carotid endarterectomy: 228 consecutive operations. Stroke 1977;8:565–568.
14. Toole JF, Yuson CP, Janeway R, et al. Transient ischemic attacks: a prospective study of 225 patients. Neurology 1978;28:746–753.
15. Brott T, Thalinger K. The practice of carotid endarterectomy in a large metropolitan area. Stroke 1984;15:950–955.
16. Dyken ML. Carotid endarterectomy studies: a glimmering of science. Stroke 1986;17:355–358.
17. Callow AD, Caplan LR, Correll JW, et al. Carotid endarterectomy: What is its current status? Am J Med 1988;85:835–837.
18. Barnett HJ, Toole JF, Hobson RW. Design of Clinical Studies in Carotid Surgery. 40th Annual Meeting, The Society for Vascular Surgery, New Orleans, LA, 1986.
19. Callow AD, Mackey WC. Optimum results of the surgical treatment of carotid territory ischemia. Circulation (Suppl) 1991;83:I-190–I-195.

20. Blaisdell FW, Lim R Jr, Hall AD. Technical Results of carotid endarterectomy. Am J Surg 1967;114:239.
21. Courbier R, Jausseran JM, et al. Routine intraoperative angiography. Its impact on operative morbidity and carotid restenosis. J Vasc Surg 1986;3:343.
22. Matsumoto GH, Cossman D, Callow AD. Hazards and safeguards during carotid endarterectomy: technical considerations. Am J Surg. 1977;133:458–462.
23. Naylor AR, Merrick MV, Ruckley CV. Risk Factors for intra-operative neurological deficit during carotid endarterectomy. Eur J Vasc Surg 1991;5:33–39.
24. Mackey WC, O'Donnell TF Jr, Callow AD. Carotid endarterectomy contralateral to an occluded carotid artery: Perioperative risk and late results. J Vasc Surg 1990;11:778–785.
25. Mackey WC, O'Donnell TF Jr, Callow AD. Cardiac risk in patients undergoing carotid endarterectomy: impact on perioperative and long-term mortality. J Vasc Surg 1990;11:226–234.
26. Hafner CD. Minimizing the risks of carotid endarterectomy. J Vasc Surg 1984;1:392–397.

5
Carotid Endarterectomy (How I Do It, A Safe Approach)

JOHN B. CHANG

Introduction

Carotid endarterectomy was first performed in 1954 as a logical procedure for the prevention of ischemic stroke distal to carotid artery stenosis.[1]

Callow et al. gave their review and made the following observations: First, their aggressive surgical approach to carotid bifurcation atherosclerosis is superior to "optimal" medical therapy in symptomatic patients. Second, their aggressive surgical approach is also superior in selected asymptomatic patients, most notably those with hemodynamically significant stenoses. Third, to achieve superiority over medical therapy, surgical series must maintain low perioperative mortality and stroke morbidity rates. And fourth, late death in the carotid endarterectomy patient is primarily related to coexisting coronary artery disease, and improvements in long-term survival await improvements in the management of such coexisting disease.[2]

The numbers of patients undergoing endarterectomy in the United States (excluding VA hospitals) rose from 15,000 in 1971 to 107,000 in 1985.[3] There are many reports showing reasonable and lower rates of preoperative complications with the carotid endarterectomy.[4–7]

The beneficial effects of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis have been reported recently in randomized trial.[8] In this report, life-table estimates of the cumulative risk of any ipsilateral stroke at two years were 26% in the 331 medical patients and 9% in the 328 surgical patients—an absolute risk reduction (±SE) of $17 \pm 3.5\%$ ($P < 0.001$). For a major or fatal ipsilateral stroke, the corresponding estimates were 13.1% and 2.5%—an absolute risk reduction of $10.6 \pm 2.6\%$ ($P < 0.001$). Carotid endarterectomy was still found to be beneficial when all strokes and deaths were included in the analysis ($P < 0.001$). Therefore, it was concluded that, in a randomized trial study, carotid endarterectomy is highly beneficial to patients with recent hemis-

pheric and retinal transient ischemic attacks or nondisabling strokes and ipsilateral high-grade stenosis (70–99%) of the internal carotid artery. There is no definite randomized trial study available at the present time for carotid endarterectomy of asymptomatic carotid stenosis, however, there are many reports on this subject.

In a study of the natural history of the growth of carotid atheromata for a period of one to nine years, there was no change in 41% and a significant increase in 59% of patients studied.[9] The progress of the atherosclerosis is not predictable in each patient. A moderate rate of progression is common for a carotid lesion as it becomes symptomatic within three to four years. Progress can be very slow, but in other cases, the lesion can progress rapidly to produce symptoms within one or two years.[10]

The author suggests that after noninvasive screening and arteriography, specific indications for carotid endarterectomy of asymptomatic carotid artery stenosis are: (1) severe unilateral stenosis greater than 70%; (2) bilateral stenoses greater than 50%; (3) unilateral stenosis and contralateral occlusion; (4) progressing stenosis to greater than 50%; (5) positive noninvasive tests, such as duplex scan, spectral analysis, OPG, and perhaps CT and MRI studies; (6) before other major surgery, if the noninvasive tests and arteriography are strongly positive; and (7) a markedly ulcerated plaque (6).

The author strongly believes that, with well-chosen patients who have had careful preoperative evaluation, carotid endarterectomy can be a safe procedure, minimizing perioperative morbidty and mortality.

Preoperative Evaluation

1. Careful medical history and assessment.
2. Carotid duplex scan.
3. CAT scan and/or MRI study of head to make a proper assessment of silent infarction, staging of the infarcted brain, and to exclude other possibilities such as nonvascular pathology of the brain.
4. Cardiac evaluation, including noninvasive and, in selected cases, invasive study such as cardiac catheterization.
5. Aortic arch study including extracranial as well as intracranial arteriography.
6. Preoperative Swan Ganz catheter after cardiac evaluation whenever indicated.
7. Routine insertion of radial artery catheter for intra- and postoperative monitoring.
8. Attachment of 12-lead EEG electrode to the head and recording of baseline EEG tracing.

Operation

1. General endotracheal tube anesthesia, normotensive and normocarbia.
2. Routine intraoperative carotid shunting.
3. Intraoperative EEG monitoring.
4. Routine use of vein patch utilizing proximal greater saphenous vein graft except for specific criteria.
5. All patients undergo aortic arch arteriography using intraarterial DSA technique. Recently, magnetic resonance imaging was initiated as a preoperative evaluation and, in selected cases, in lieu of formal angiography.

Operative Technique

1. Patient is admitted to the intensive care unit the night before surgery for cardiac evaluation and optimization for the operation. During this time, preoperative Swan Ganz catheter is inserted for preoperative optimization and intraoperative monitoring as well as postoperative monitoring.
2. Patient is placed in a semi-Fowler position with the neck extended and rotated to the contralateral side.
3. Routine preoperative prophylactic antibiotics are given.
4. The neck, chest and selected lower extremity are prepped with Betadine solution.
5. The operative fields are exposed—operative side of the neck, selected side of the neck and selected side of the thigh to harvest vein segment for vein patch.
6. A semivertical incision is made on the operative side of the neck along the anterior border of the sternocleidomastoid muscle. In younger female patients, sometimes a transverse incision is made under the mandible at the level of the bifurcation (Fig. 5.1). The dissection is carried out using sharp and blunt technique, controlling hemostasis with electrocoagulation and ligatures. As dissection is deepened, the sternocleidomastoid muscle is freed and retracted laterally, and the carotid sheath is gradually opened.

   The first anatomic landmark to be identified is a facial branch of the internal jugular vein. Most of time, the level of bifurcation is under this facial vein. The vein is freed, ligated and divided (Fig. 5.2). Then the common carotid artery is freed proximal to the plaque. The dissection is carried out distally to expose the bifurcation. A 0.5% marcaine solution, about 0.5 to 1.0 cc, is infiltrated into the carotid body. The first branch of the external carotid artery is then easily identified by careful dissection. The proximal external carotid artery is then freed and circled with vessel loop. Distal dissection is then carried out to the internal carotid artery. Many times ansa hypocrossi can be identified, running

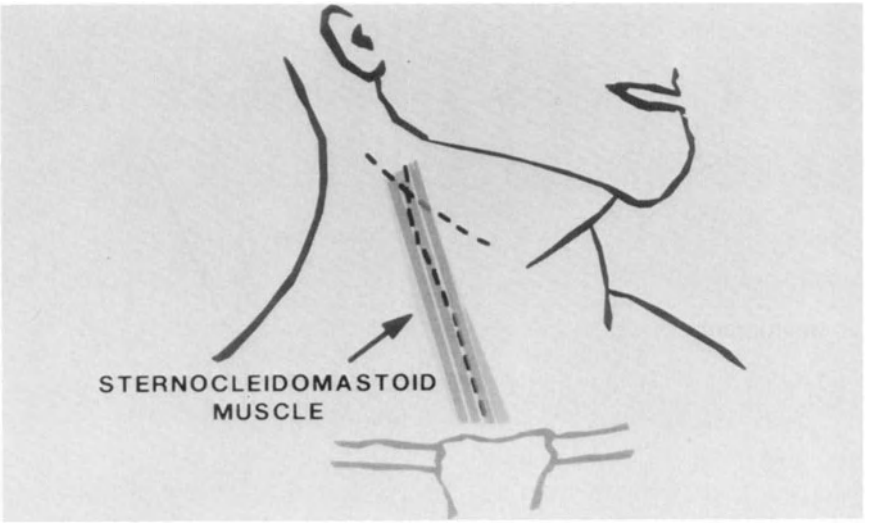


FIGURE 5.1. Schematic drawing showing two types of incisions for carotid endarterectomy. A transverse incision and a semivertical incision along the anterior border of the sternocleidomastoid muscle.

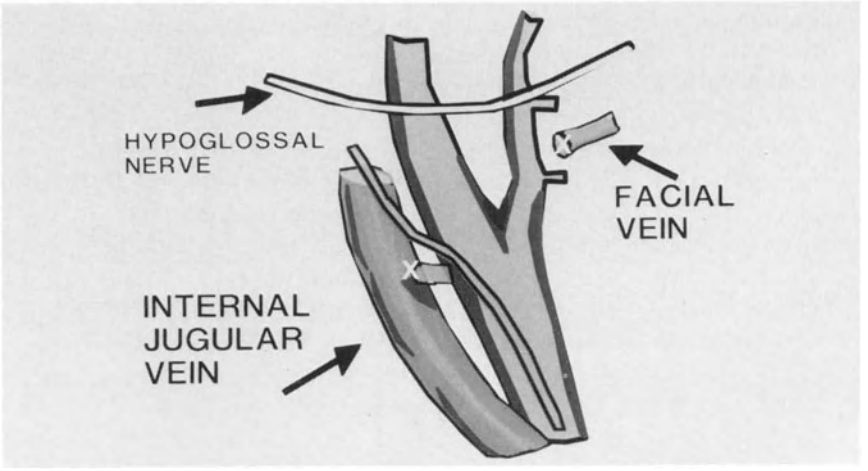


FIGURE 5.2. Schematic drawing indicating facial branch of the internal jugular vein (facial vein) as a landmark of an anatomic position of the carotid bifurcation. The hypoglossal nerve is shown in relation to the carotid bifurcation.

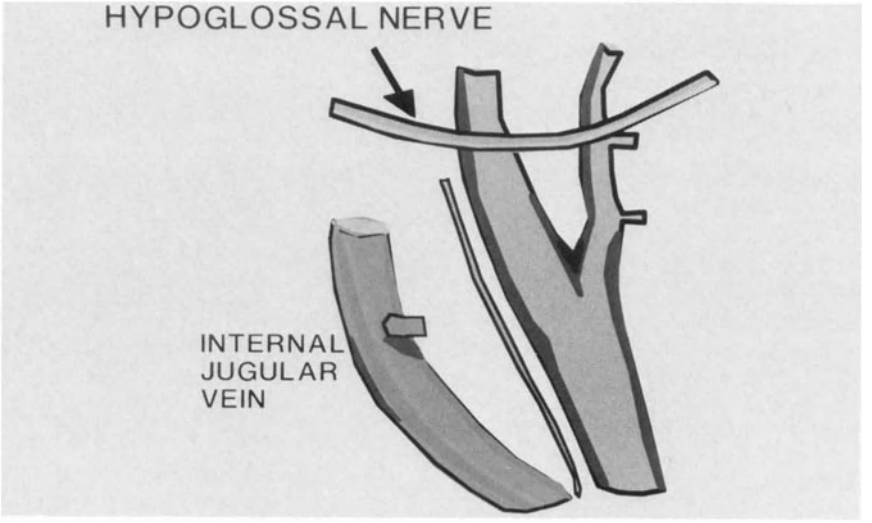


FIGURE 5.3. Schematic drawing after ligation and division of facial vein.

anteriorly, medially and sometimes laterally, joining up to the hypoglossal nerve. The hypoglossal nerve is identified and preserved intact (Fig. 5.3).

When the lesion is high, with high bifurcation, the "sling vessels" are carefully freed, ligated and divided. By this maneuver, the hypoglossal nerve is further freed distally. In selected cases, the ansa hypoglossal can be divided to facilitate the mobilization of the hypoglossal nerve. Sometimes, the digastric muscle is carefully divided to gain further distal exposure. During this maneuver, care should be taken to avoid injury to the glossopharyngeal nerve.

With these maneuvers, the mandible joint subluxation or dislocation is seldom necessary. At this point, a 2-1/2 inch length of proximal greater saphenous vein is harvested and divided at the line of branch(es), if any. Patient is given systemic heparinization, usually 6,000 to 7,000 units, depending upon the size of the patient. The common, internal and external carotid arteries are cross clamped using atraumatic vascular clamps. A longitudinal arteriotomy is made, extending from the proximal common carotid artery to the distal internal carotid artery distal to the plaque (Fig. 5.4). It is also my practice to make two separate initial arteriotomies, one on the proximal common carotid and the other on the distal internal carotid artery if the lesion is critically stenotic, making a single arteriotomy difficult. Then a T-shape, indwelling shunt catheter is introduced into the common and internal carotid artery (Fig. 5.5). In cases where two separate arteriotomies are made, these two arteriotomies are then connected after the shunt is inserted and opened.

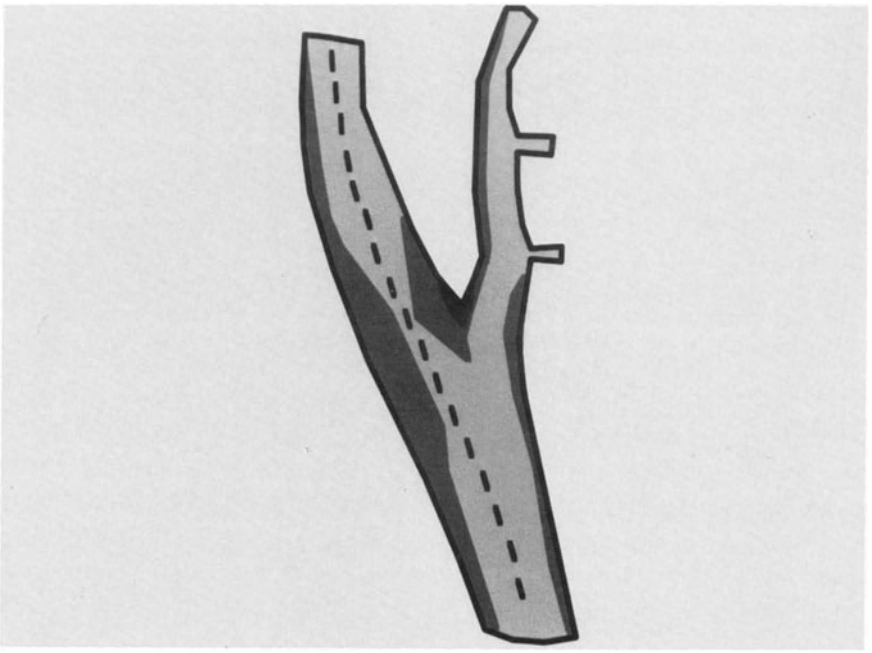


FIGURE 5.4. Schematic drawing with arteriotomy extending from the common carotid artery proximal to the plaque to the distal internal carotid artery distal to the plaque (dotted line).

Care should be taken to evacuate all possible air or clot from the shunt unit and arterial system before the shunt is opened. During this maneuver, intraoperative EEG monitoring is helpful in monitoring the technique of cross clamping and shunting.

Under direct vision, with the shunt in, the endarterectomy is carried out, starting from the opposite side of the external carotid artery origin, coming down to the proximal common carotid artery (Fig. 5.6, circle 1). Then the endarterectomy is extended proximally using a special spatula instrument. At the point of normal intima, a sharp dissection is carried out, cutting at the normal intima to the other side of the common carotid artery (Fig. 5.6, circle 2). Then proceed distally, on the side of the external carotid artery, up to the origin of the external carotid artery (Fig. 5.6, circle 3). Attention should then be paid to the distal portion of the endarterectomy technique, proceeding to the end point of the plaque at the internal carotid artery (Fig. 5.6, circle 4). Continue down the internal carotid artery to the origin of the external carotid artery. At the origin of the external carotid artery, the careful blunt, sometimes direct endarterectomy, is carried out into the external carotid artery beyond the origin of the first branch if necessary (Fig. 5.6, circle 5). At this

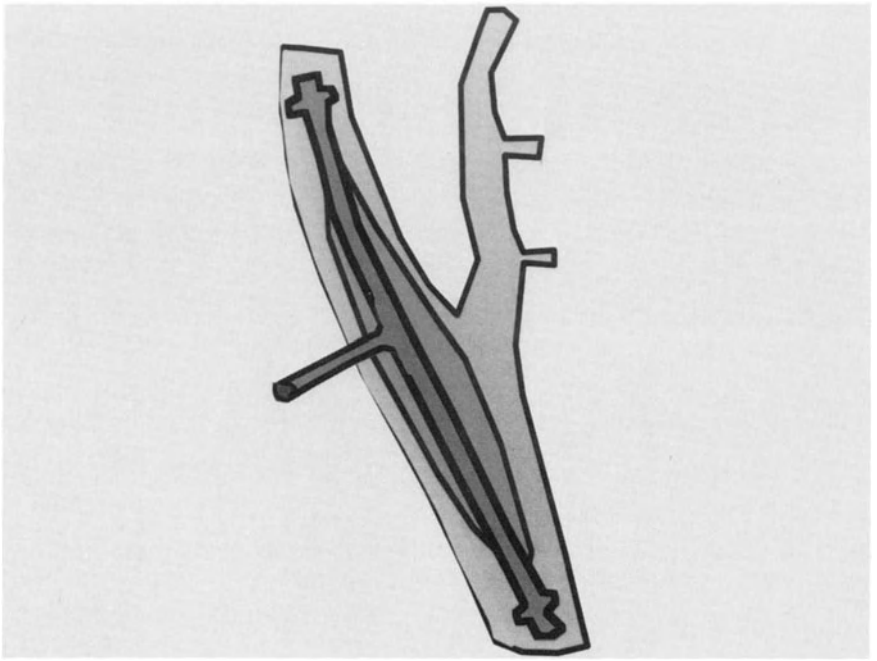


FIGURE 5.5 Schematic drawing showing carotid artery shunt (T type). By using this type of catheter, it is easy to evacuate any possible air bubbles or debris within the shunt before the shunt is opened. Also using this T type of shunt, a hemodynamic study can be performed with ease, such as stump pressure.

point, the entire specimen is hanging on the origin of the internal carotid artery (Fig 5.6, circle 5). As the plaque from the external carotid artery is pulled out, the entire procedure of endarterectomy is completed, removing the entire specimen as one piece.

If the end point at the internal carotid artery is not clean, leaving normal intima behind, this normal intima is tacked down with 6-0 Prolene sutures, in interrupted fashion, to prevent possible distal dissection. If the end point is clean, there is no need for a tack-down suture. The arterial lumen is irrigated with a copious amount of heparinized solution. Painstaking effort should be given to remove all possible debris from the arterial lumen. Then the arteriotomy is repaired using the proximal greater saphenous vein patch graft. The patch graft should be extended far proximally to the margin of the intima at the common carotid artery and far distally beyond the end point, as indicated in (A′) and (B′) of Fig. 5.7. At the time of carotid endarterectomy, if there is kinking at the internal carotid artery (Fig. 5.8), this is corrected at the time of vein patch by using plication technique, bringing B to B′ point (Fig. 5.8) by making transverse sutures using 6-0 Prolene sutures evagi-

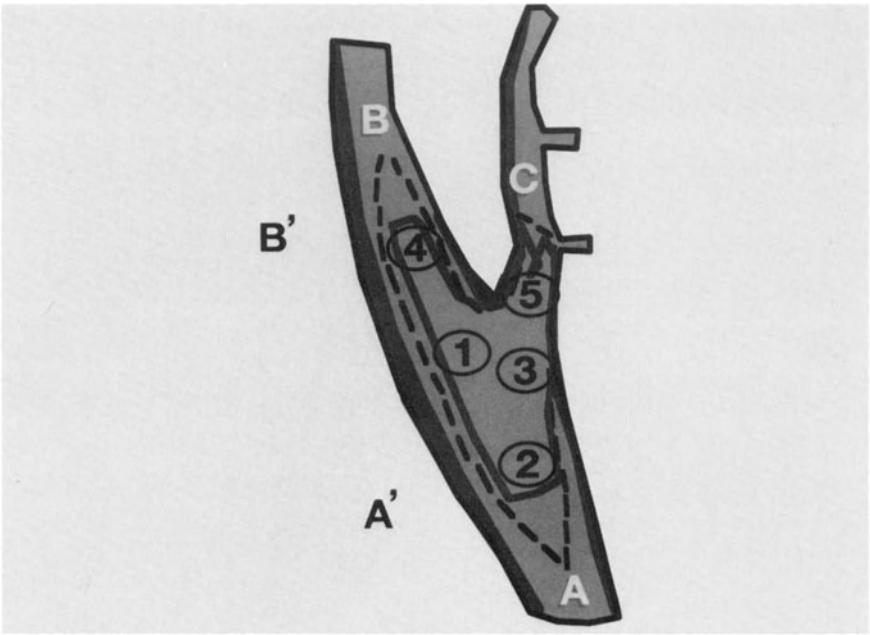


FIGURE 5.6. Schematic drawing showing carotid endarterectomy in sequential steps. **A** indicates proximal end of arteriotomy. **B** indicates distal end of arteriotomy. **C** indicates proximal portion of the external carotid artery. **A′** indicates line of endarterectomy, proximally. **B′** indicates line of endarterectomy and/or distal endpoint at the internal carotid artery. The carotid endarterectomy normally starts at the midportion of the arteriotomy, opposite to the orifice of the external carotid artery (one). Endarterectomy is extended proximally and transects the specimen at the proximal common carotid artery (two). The dissection proceeds to the side of the external carotid artery extending distally (three). Then attention is directed to the distal portion of the endarterectomy plane opposite to the external carotid artery origin. The endarterectomy is then carried distally into the internal carotid artery to the endpoint extending proximally on the opposite side of the internal carotid artery (four). The final stage of the endarterectomy is centered to the origin of the external carotid artery. Depending upon the nature of stenosis of the external carotid artery, either blind or open endarterectomy is performed to complete the procedure delivering the specimen in total (five).

nating the redundant portion of the backwall as shown on (B) and (B′) in Fig. 5.9. After this is done, the vein patch is anastomosed at the arteriotomy, which will result in a straighter arterial system as shown in Fig. 5.9.

Before the vein patch is completed, the indwelling shunt catheter is removed. Again, care should be taken to remove all possible air or clot from the arterial system. Circulation is restored, first to the external

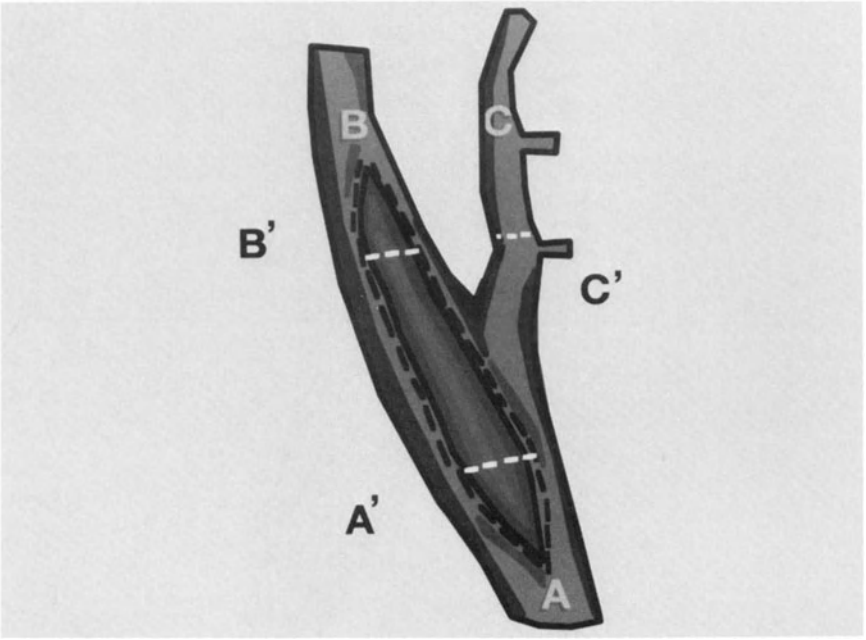


FIGURE 5.7. Schematic drawing showing vein patch after carotid endarterectomy. **A** indicates proximal end of the arteriotomy. **B** indicates distal end of the arteriotomy. **C** indicates distal external carotid artery. **A′** indicates line of endarterectomy proximally. **B′** indicates line of endarterectomy or endpoint at the internal carotid artery. **C′** indicates distal end of the endarterectomy at the external carotid artery. Effort is given to extend the arteriotomy and vein patch proximal to the proximal end of the endarterectomy line (**A**) and distal to the distal endpoint (**B**).

carotid artery, then to the internal carotid artery in a sequential manner. EEG monitoring is especially useful during this process. If there is no major problem or bleeding from anastomosis, the heparin is neutralized with the Protamine in about a three to four ratio. The wound spaces are irrigated with antibiotic solution. The incisions are closed using 2-0 Vicryl or Dexon sutures and 4-0 absorbable sutures to the skin in subcuticular fashion. Sterile dressing is applied to the wound. On the neck, gentle pressure dressing is applied using 4×8 and combine pads. Using a sterile towel, a gentle collar is made around the neck incision

FIGURE 5.9. Schematic drawing indicating surgical correction of internal carotid artery kink after endarterectomy. **A** approximating to point **C** after plicating and evaginating sutures between **B** and **B′** in Fig. 5.8—the distal internal carotid artery (**A**) is now approximated to the proximal internal carotid artery (**C**). Then the vein patch is sewn in as indicated with the white dotted line.

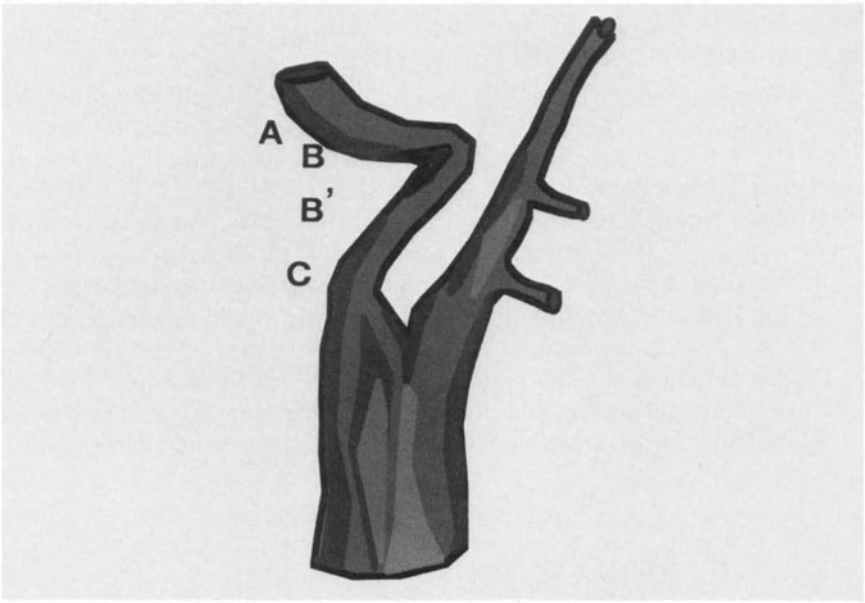
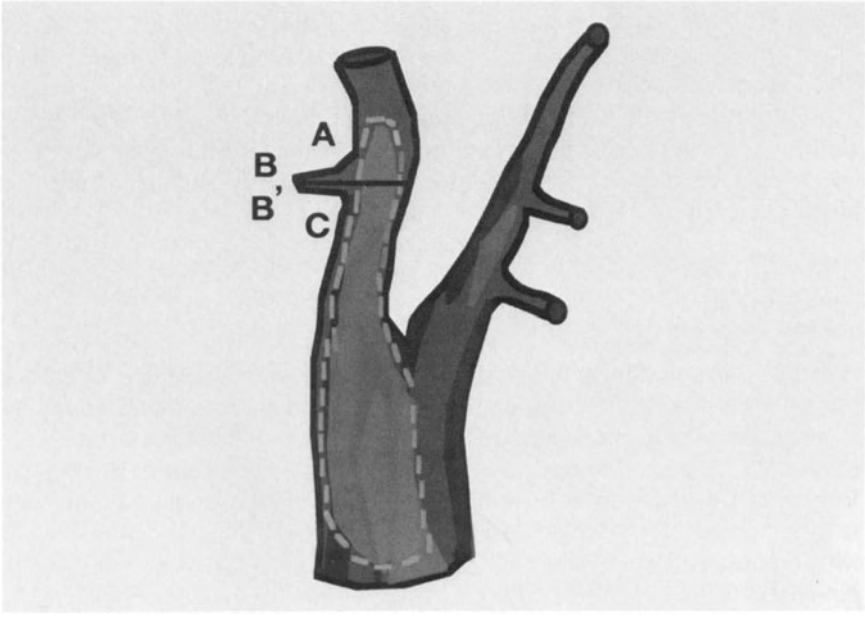


FIGURE 5.8. Schematic drawing indicating kink of internal carotid artery. **A** indicates distal internal carotid artery endarterectomized. **B** indicates imaginary line of plication with **B′** to correct the kink. **C** indicates proximal internal carotid artery endarterectomized.



dressing. Patient is allowed to recover from general anesthesia, on the table, with EEG monitoring. Patient is extubated on the table.

Once the patient is neurologically intact, moving all extremities on command, transfer can be made to the recovery room. Until the patient's neurological assessment is completed, the surgical instruments remain in the operating room without contamination.

In the recovery room, cardiac and neurological evaluations are made periodically. The first night the patient remains either in the recovery room or the intensive care unit for observation. The dressing is removed on the first postoperative day, and the patient is allowed to get out of bed and ambulate, resuming a full regular diet.

The patient is normally discharged on the third postoperative day unless there are specific reasons to keep the patient longer. The patient is discharged with an antiplatelet agent, buffered aspirin and/or Persantine.

Long Term Management

Patient is followed closely on an outpatient basis by the vascular surgeon, using noninvasive vascular studies, including duplex scan.

Results

With this protocol, the author has performed 450 carotid endarterectomies since 1976. The earlier results indicated 0.5% mortality with 1.4% stroke rate.[11] In a recent review of 450 carotid endarterectomies, there was a 0.4% stroke rate with 0.2% stroke related mortality, and 0.4% of myocardiac infarction with 0.2% MI related mortality. Of the last 300 carotid endarterectomies performed using the rigid protocol outlined above, there was no stroke rate or mortality rate, perioperatively and up to 30 days postoperatively.

Discussion

Among symptomatic patients with high grade stenosis (70% to 99%), those who underwent carotid endarterectomy had an absolute reduction of 17% in the risk of ipsilateral stroke at two years ($P < .001$) in the recent randomized study.[1] Similar results have recently been reported from the European Carotid Surgical Trial.[12] Among 778 symptomatic patients with severe stenosis (70% to 99%) who were randomly assigned to treatment with carotid endarterectomy or medical care alone, 7.5% of the surgical patients had an ipsilateral stroke or died within 30 days of surgery. Life-

table estimates show that the risk of ipsilateral stroke during the next three years yield an additional risk of 2.8% for surgical patients as compared with 16.3% for medical patients ($P < 0.0001$). Until a further guideline is given for proper cerebral protection during carotid endarterectomy, the author believes in a combination of intraoperative shunting and EEG monitoring to minimize morbidity and mortality from the surgical procedure.

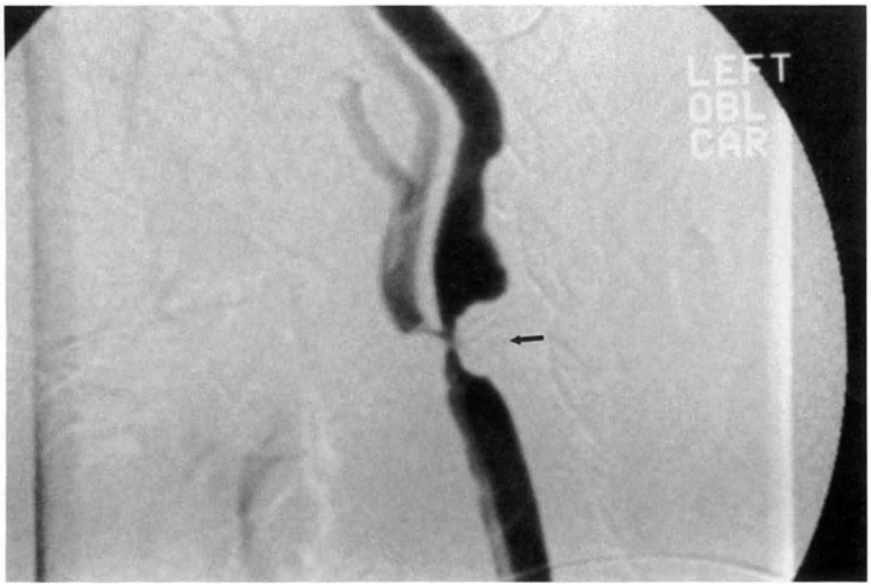
There are many papers about saphenous vein patch and other patch materials vs. primary closure after carotid endarterectomy.[13–19] Biochemical adaptation of venous patches have been studied, indicating that the vein patch angioplasty favorably influences surface thrombogenicity of healing on the endarterectomized arteries.[20–23] Some suggest that saphenous vein angioplasty appears appropriate in habitual smokers and likely in patients with small internal carotid arteries.[24–25]

In high bifurcation or extremely high lesion, there are other techniques available. In our experience, careful dissection along the anatomical boundary of the distal carotid artery, ligation and division of "sling vessels" (sternoidcleidmastoid artery and vein), and division of posterior belly of digastric muscle have all been successful. However, there are other techniques, including anterior subluxation of the mandible, styloidectomy in combination with other techniques, and lateral mandiblectomy or posterior approach with mastoidectomy.[26] Temporary mandibular subluxation by diagonal interdental/Steinmann pin wiring has been utilized.[27] Various techniques of exposure and control of the internal carotid artery near the base of skull, Zone III (from the lower margin of the first cervical vertebra to the base of the skull) have been proposed by different investigators. An exposure of the internal carotid artery was described that involved preauricular amd postauricular incisions, making anterior and posterior flaps. Dissection of the parotid gland anteriorly and resection of digastric muscle from the mastoid process was burred down and resected. The glossopharyngeal nerve was sacrificed, permitting exposure of the internal carotid artery at the base of the skull.[28] Subperiosteal osteotomy at the angle of the mandible has been described by Welsh et al.[29] The posterior segment was rotated upward over the temporomandibular joint, and the anterior segment was retracted forward. The styloid process and digastric muscle were divided. Some surgeons perform radical mastoidectomy with obliteration of the middle ear and resection of the mandibular condyle and occasionally the zygoma.[30] Arch bar wires were used to maintain subluxation of the mandibular joint.[31] These techniques are of value in carotid trauma at the base of skull, high internal carotid artery aneurysms, fibromuscular dysplasia, and recurrent stenosis requiring endarterectomy near the base of the skull. However, these techniques are associated with significant morbidity in the form of bone infection, loss of ear function, nonunion of mandible, cranial nerve paralysis, and compression of opposite carotid artery.[32] Atherosclerotic high plaque ended at Zone II in patients

(from the lower margin of the second to the lower margin of the first cervical vertebra), indicating that other forms of techniques for exposure are rarely necessary for primary endarterectomy for atherosclerotic carotid bifurcation stenosis. The greater auricular nerve may need to be sacrificed, causing temporary cutaneous paresthesia. The glossopharyngeal nerve may be disturbed in dissection necessary for these high bifurcated lesions. The possibility of high plaque should be considered in male patients with bilateral severe carotid occlusive disease or patients with contralateral occlusion. With careful dissection along the anatomic planes as well as knowledge of anatomy superior to hypoglossal nerve and cephalad exposure, endarterectomy can be accomplished. All atherosclerotic carotid bifurcation plaque in internal carotid artery, however high, will ultimately taper off in Zone II and should never have to be sharply divided, which would compromise the adequacy of the endarterectomy.[33]

Conclusions

Carotid endarterctomy can be performed with reasonable safety using the following rigid protocol that the author has applied to his 450 cases:



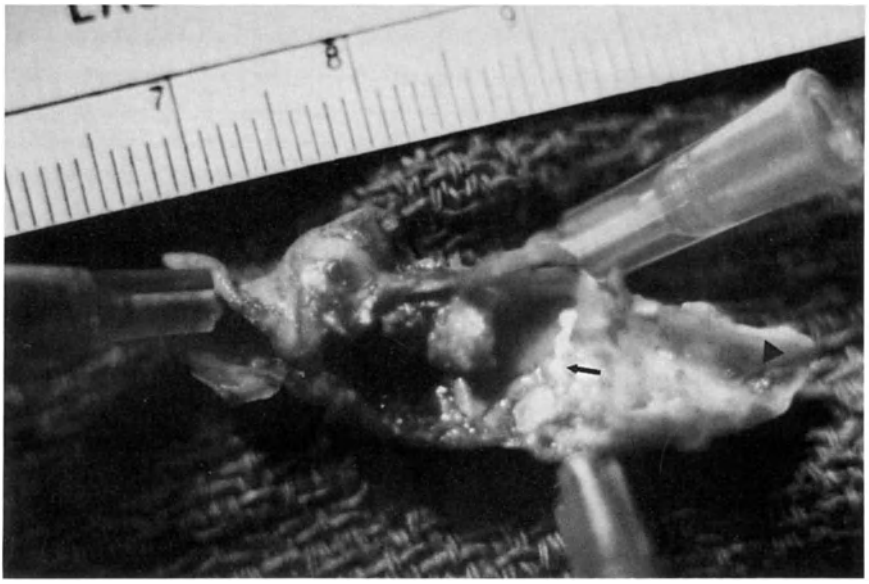
A

FIGURE 5.10A. Angiogram with high grade stenosis at left distal common and proximal internal carotid artery as well as external carotid artery (arrow).

1. Careful clinical assessment.
2. Preoperative evaluation including duplex scan, CT/MRI, and more recently, with MRI angiogram.
3. Preoperative aortic arch angiogram.
4. Independent assessment of patient prior to surgical decision by medical/cardiological/neurological consultants.
5. Preoperative Swan-Ganz catheter insertion.
6. Liberal use of autogenous vein patch (preferably proximal greater saphenous vein segment).
7. Routine shunting.
8. Intraoperative EEG monitoring.

Most patients are discharged home on the third postoperative day. Long-term follow-up is important for all patients, with periodic clinical evaluation, duplex scan and, if necessary, angiogram. Recently, MRI angiogram has been useful. This may be replaced, in some cases, for aortic arch angiogram.

The following cases have been selected for further illustration of the author's clinical cases (see Figs. 5.10–5.21):

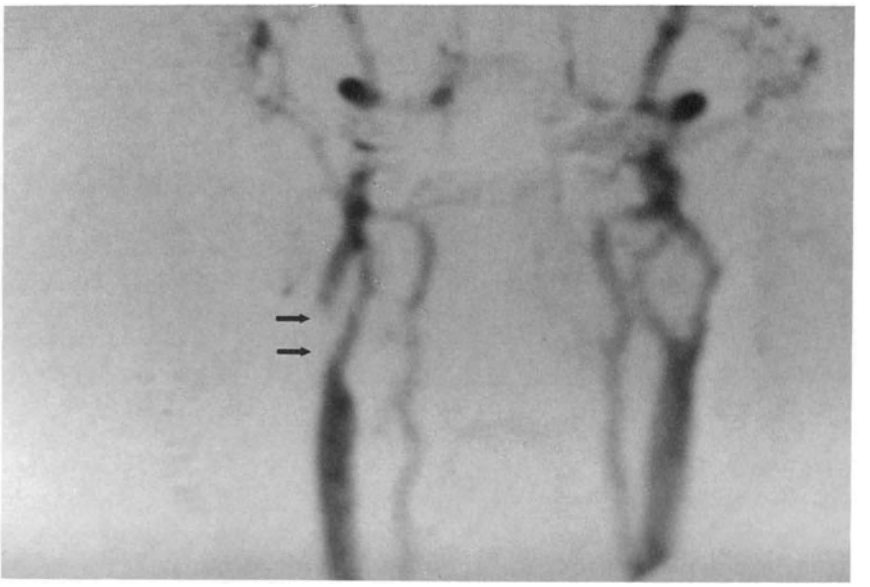


B

FIGURE 5.10B. Endarterectomy specimen with severe stenosis and ulcerating plaque (arrow). It is extremely important that the distal internal carotid artery should be freed far away from the endpoint of the plaque (triangular arrow).

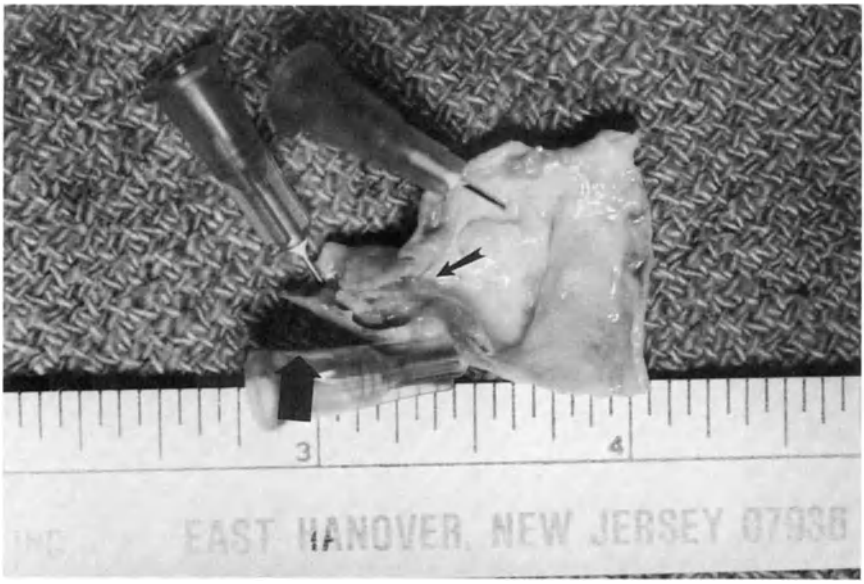
C

FIGURE 5.10C. Vein patch. The distal end of the vein patch should be distal to the endpoint of the plaque. Care is taken to extend distal arteriotomy further distal to the plaque (arrow). The proximal end of the vein patch is further proximal to the proximal endpoint or cut margin of the intima (triangular arrow).



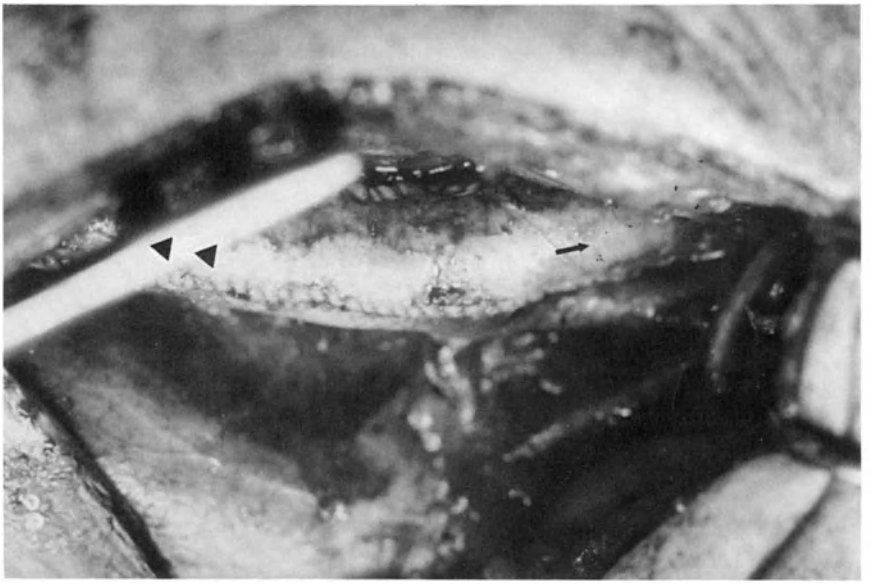
A

FIGURE 5.11A. Angiogram shows high grade stenosis with pseudoocclusion of the right internal carotid artery (two arrows).



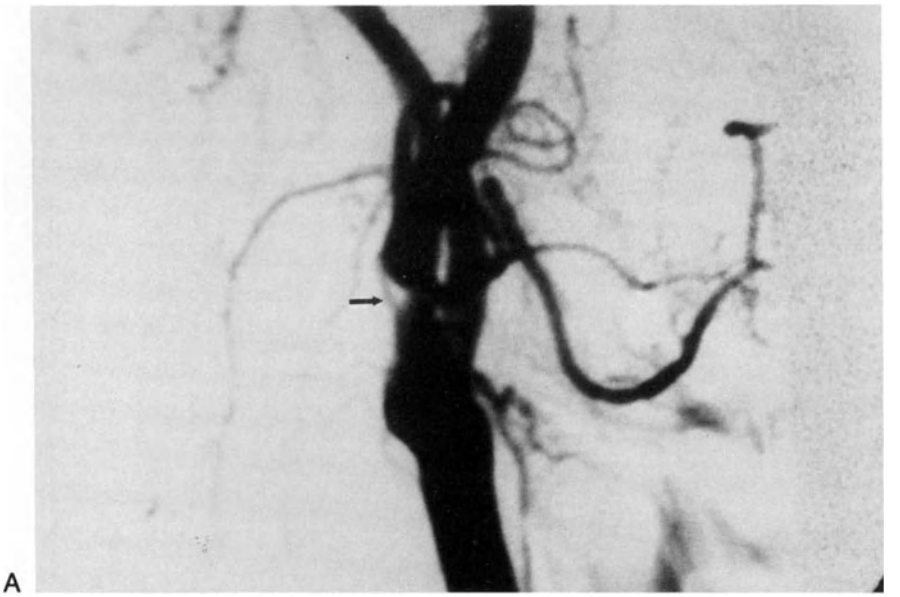
B

FIGURE 5.11B. Specimen. High grade stenosis at right internal carotid artery (big arrow) with ulcerating plaque, proximally (small arrow).



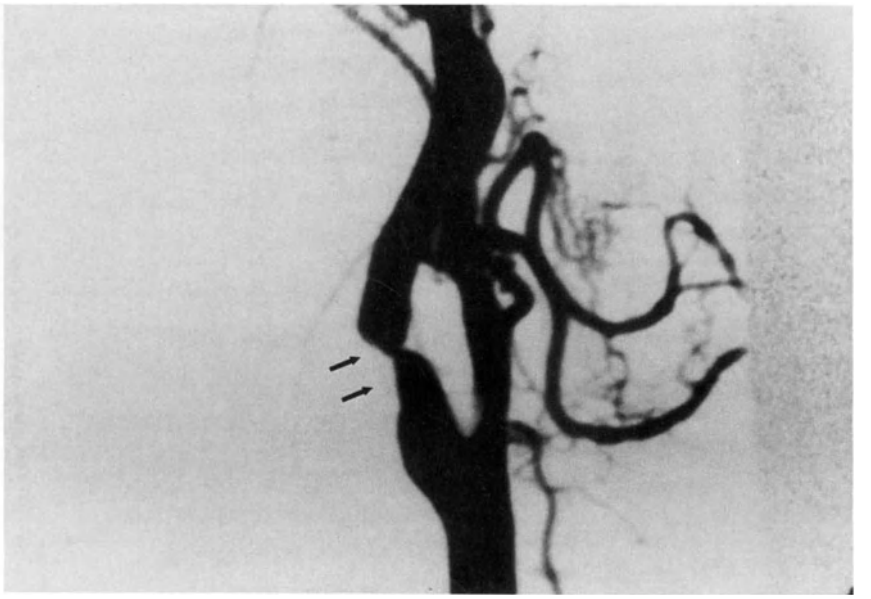
C

FIGURE 5.11C. Vein patch with proximal end (arrow) and distal end (two triangular arrows) beyond the end points.



A

FIGURE 5.12A. Angiogram showing stenotic lesion in the right internal carotid artery (arrow).



B

FIGURE 5.12B. On the same patient, an oblique view showing the lesion better (two arrows).

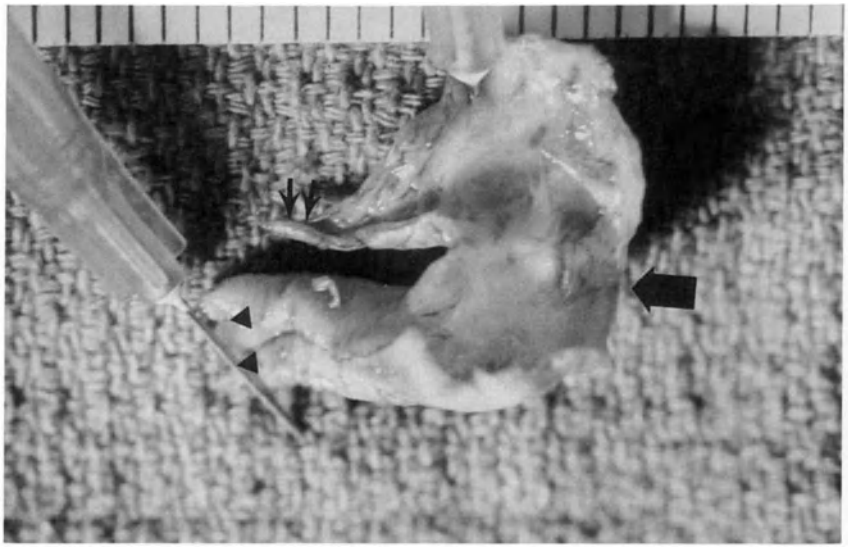


FIGURE 5.12C. Specimen. High grade stenosis at the internal carotid artery (two triangular arrows). Plaque at the external carotid artery is removed up to the endpoint (two arrows) by blind or sometimes open endarterectomy technique. The proximal end of the endarterectomy is extended to the endpoint or most normal level of the intima (big arrow).

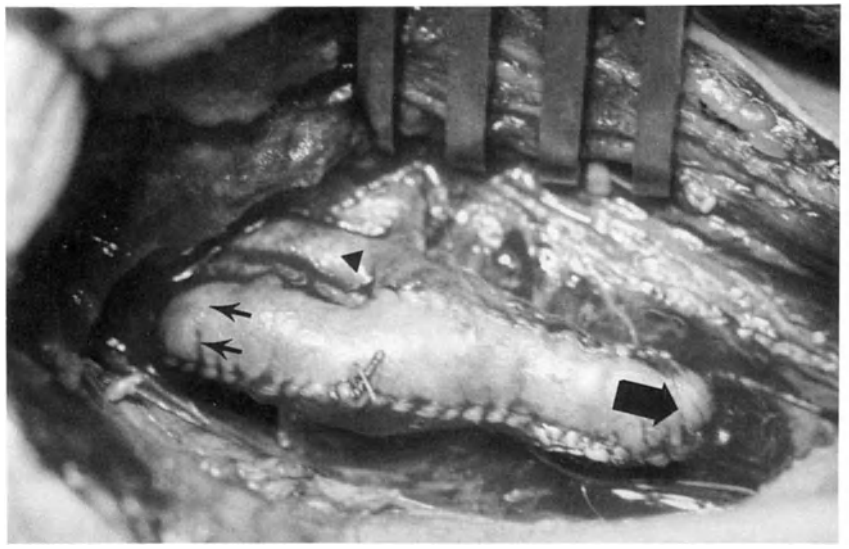
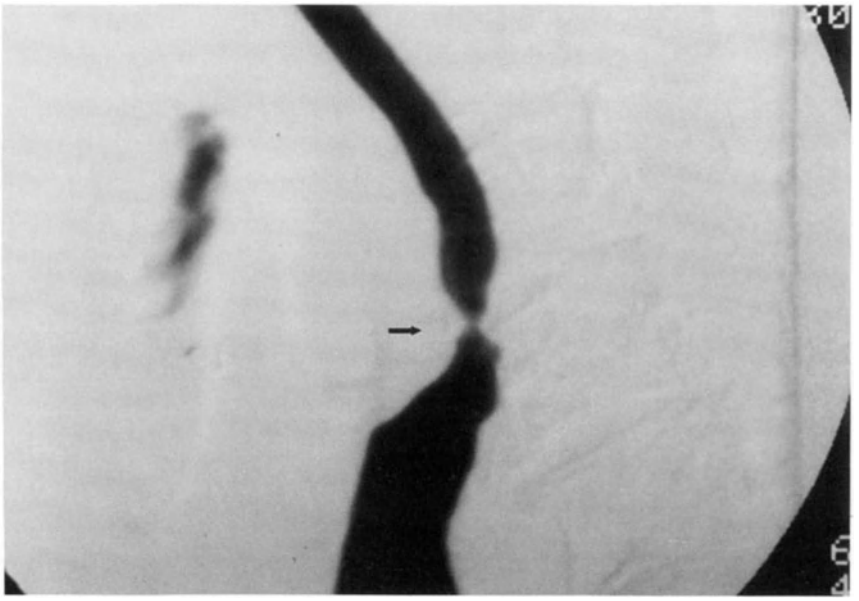


FIGURE 5.12D. Vein patch. Proximal (large arrow) and distal (two small arrows) ends. The external carotid artery is shown in relation to the vein patch (triangular arrow).



A

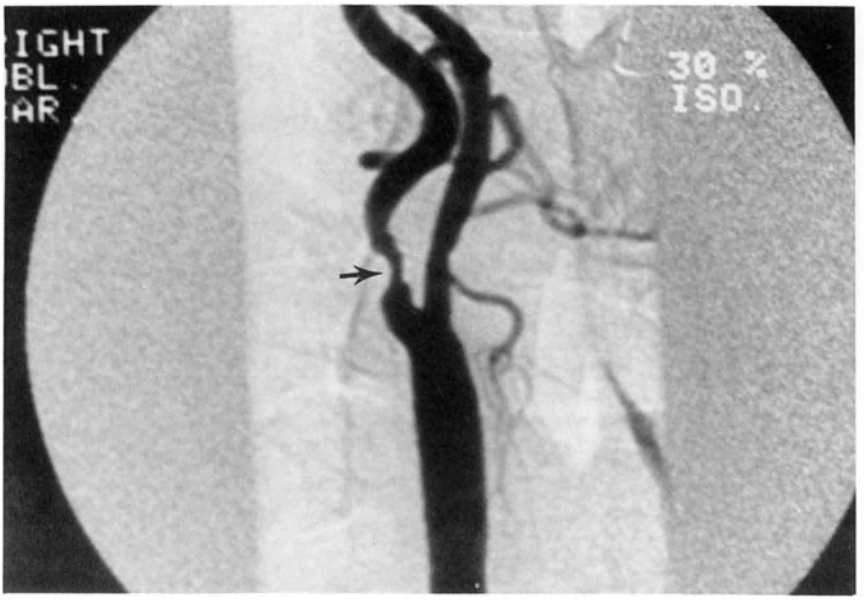
FIGURE 5.13A. Angiogram shows high grade stenosis at the right internal carotid artery (arrow) with complete occlusion of the external carotid artery (nonvisualization).

B

FIGURE 5.13B. Specimen. High grade stenosis at right internal carotid artery. (arrow). Completely occluded orifice of the external carotid artery (two triangular arrows) with endpoint of external carotid endarterectomy (large arrow). A large complex ulcer filled with friable, cheesy material (two arrows) at common carotid artery.

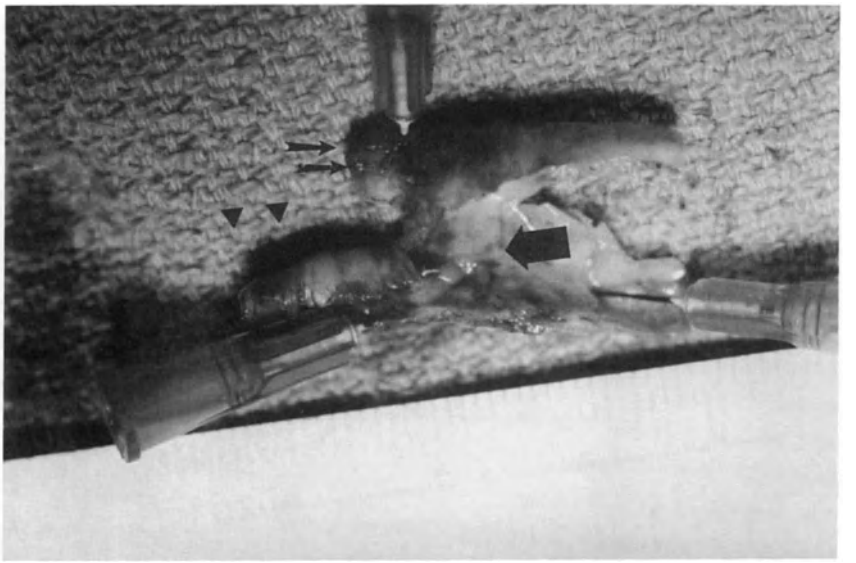
C

FIGURE 5.13C. Vein patch with distal (triangular arrow) and proximal (two triangular arrows) ends. After endarterectomy of the external carotid artery in conjunction with the common-internal carotid endarterectomy, blood flow is established to the external carotid artery (arrow).



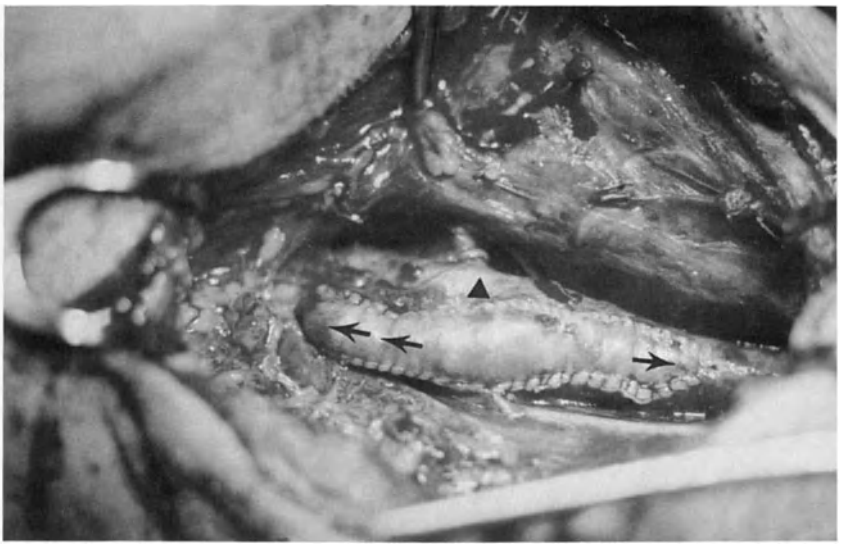
A

FIGURE 5.14A. Angiogram shows segment of stricture. Because of sudden progression of stable, mild stenotic lesion to a hemodynamically significant stenosis and occurrence of symptoms, patient was treated surgically.



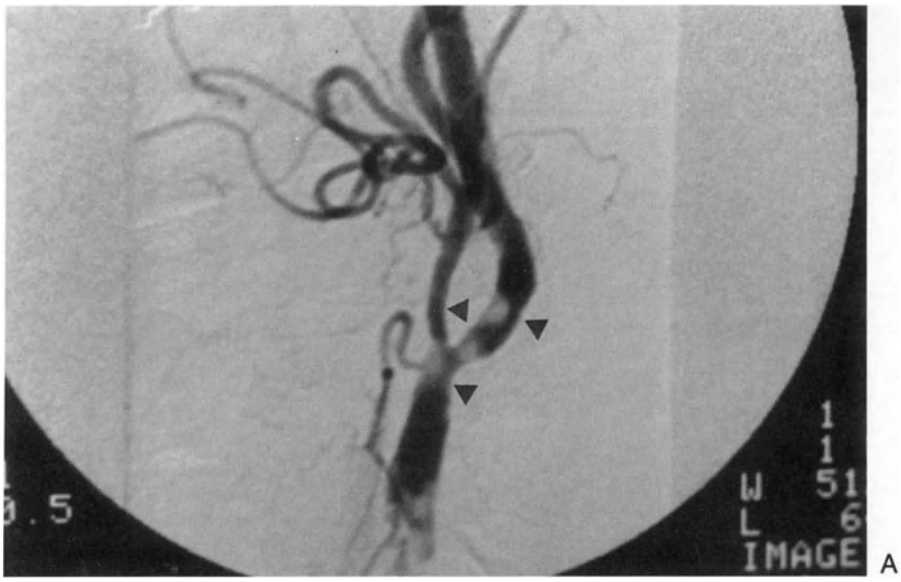
B

FIGURE 5.14B. Specimen shows stenosis (two triangular arrows) with subintimal hemorrhage. At the proximal internal carotid artery (large arrow), stenosing plaque was removed in conjunction with endarterectomy specimen (two arrows) even though angiogram did not show significant stenosis at the proximal external carotid artery.



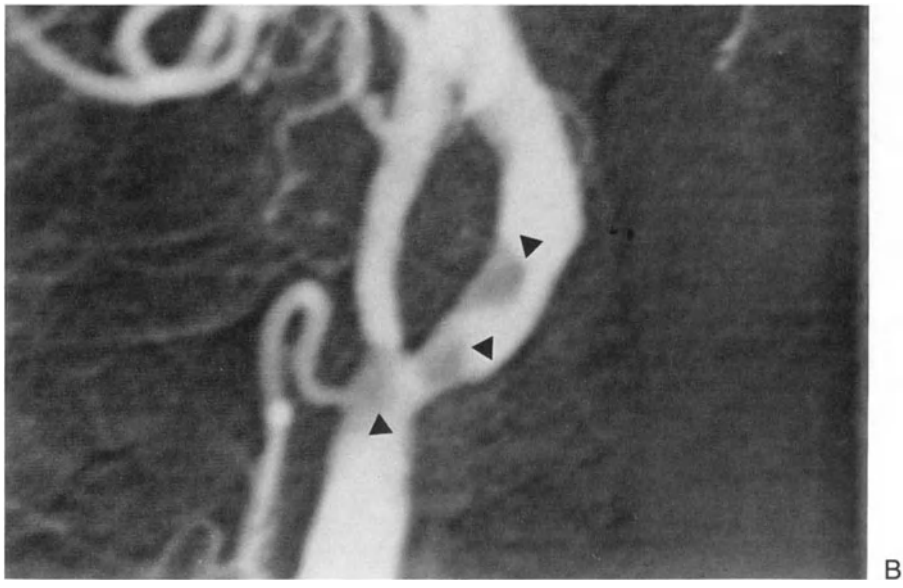
C

FIGURE 5.14C. Vein patch with proximal (one arrow) and distal (two arrows) ends. The external carotid artery is shown with the first branch being dissected free (triangular arrow).



A

FIGURE 5.15A. Angiogram shows multiple filling defects suggesting multiple thrombi (triangular arrows).

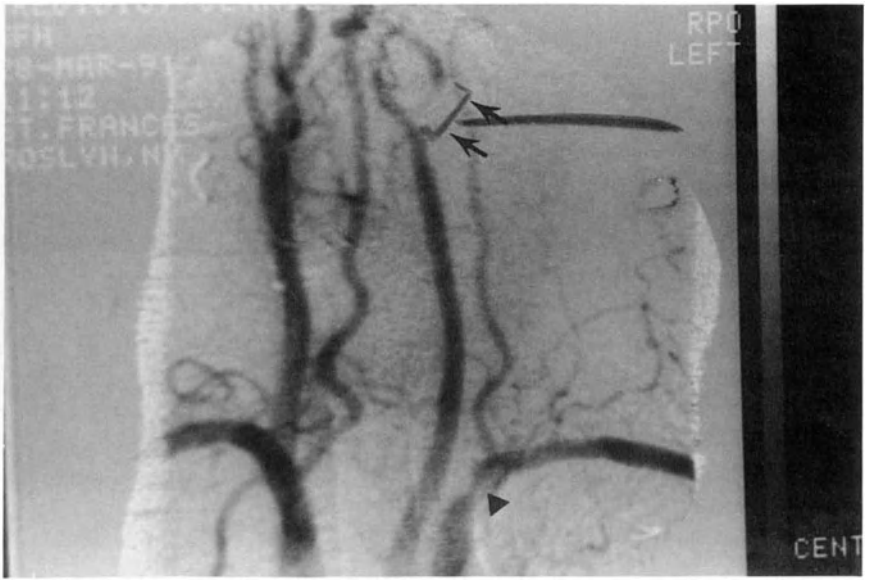


B

FIGURE 5.15B. Close-up film shows the same defects (triangular arrows).

C

FIGURE 5.15C. Specimen. Subintimal hemorrhage at the distal common carotid artery (arrow) with a large ulcer (two arrows) and multiple thrombi (triangular arrows).

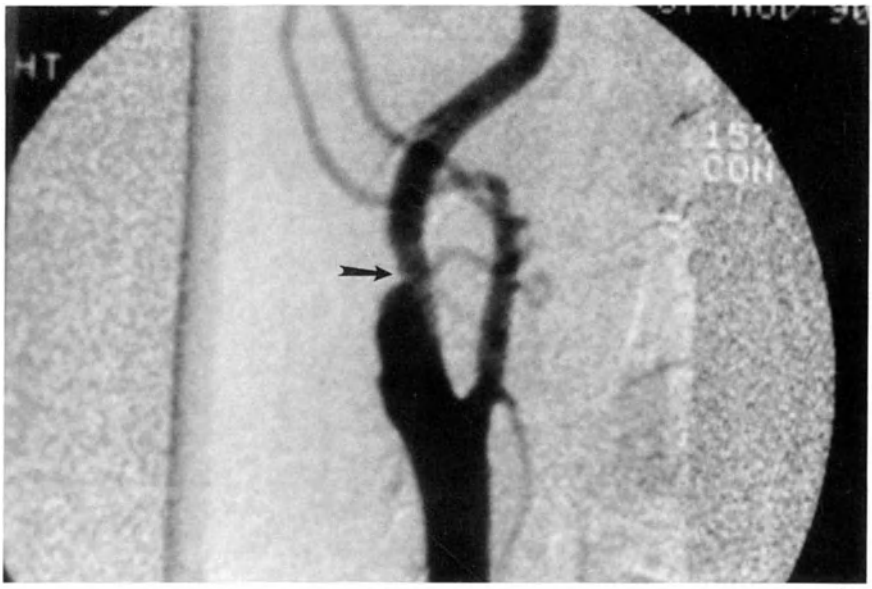


A

FIGURE 5.16A. Aortic arch angiogram shows high grade stenotic and pseudo-occlusive lesion at the left internal carotid artery (two arrows) with incidental mild stenotic lesion at the left subclavian artery (triangular arrow), which is not hemodynamically significant.

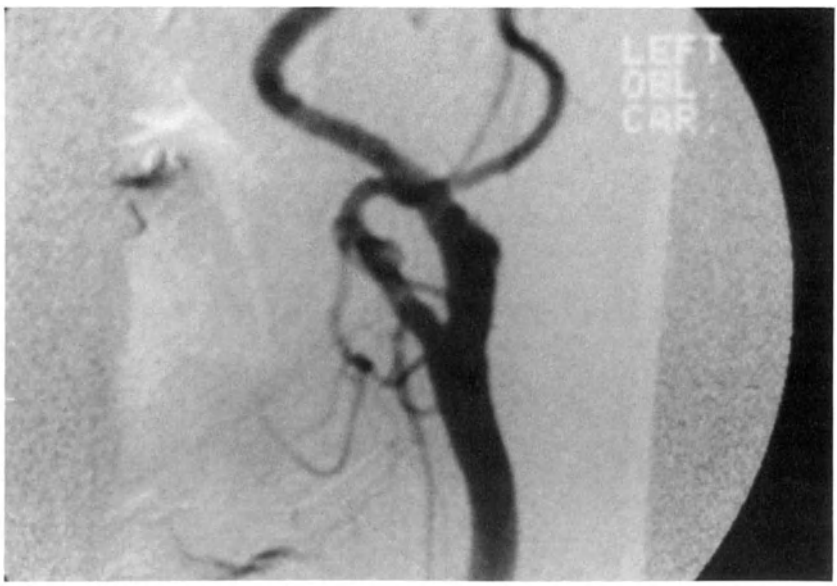
B

FIGURE 5.16B. Specimen. Extensive subintimal hemorrhage extending from the common carotid artery into the internal carotid artery (big arrow), and complex ulcer filled with atheromatous material and thrombi (triangular arrow). Distal endpoint at the internal carotid artery is seen on the right hand side of the specimen (two big arrows).



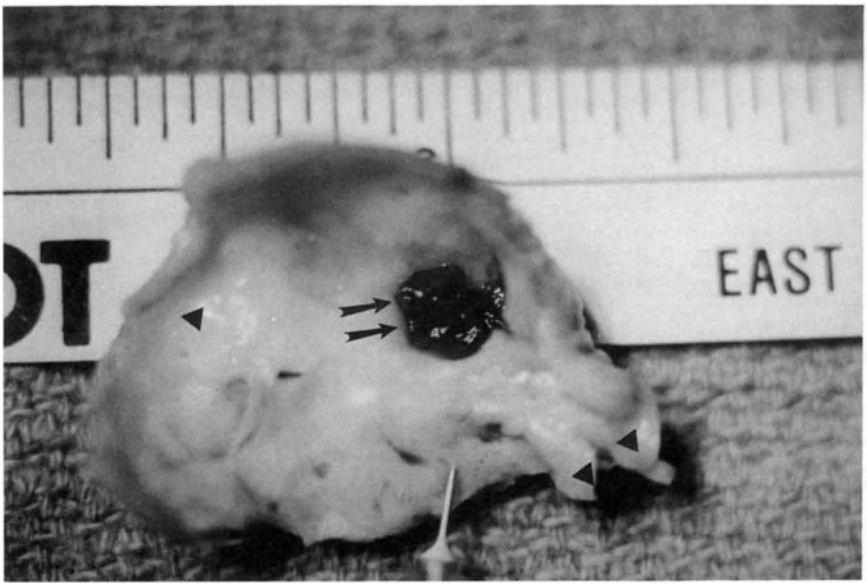
A

FIGURE 5.17A,B. Angiographic films show a moderate degree of stenosis but with large ulcerating plaque (arrow) at the right internal carotid artery.



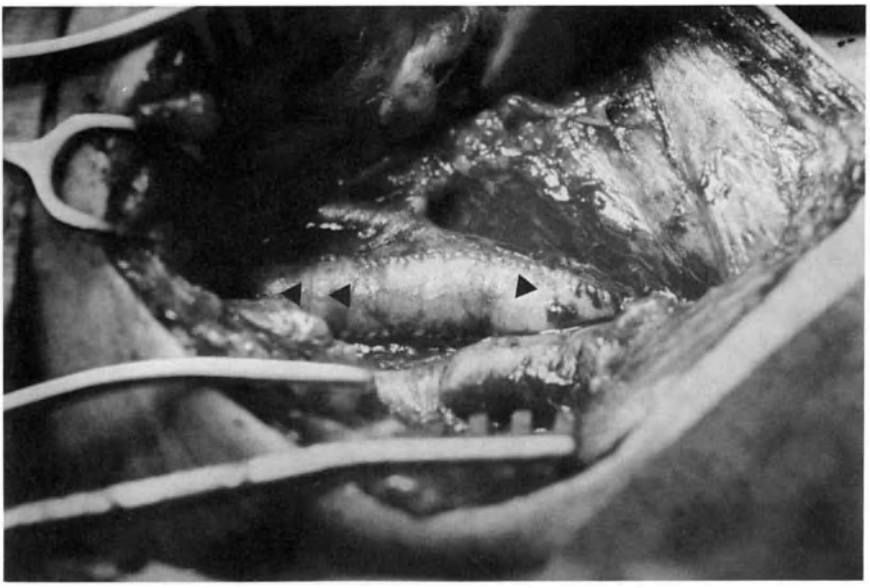
C

FIGURE 5.17C. No major stenotic lesion is seen on the left carotid artery system of the same patient.



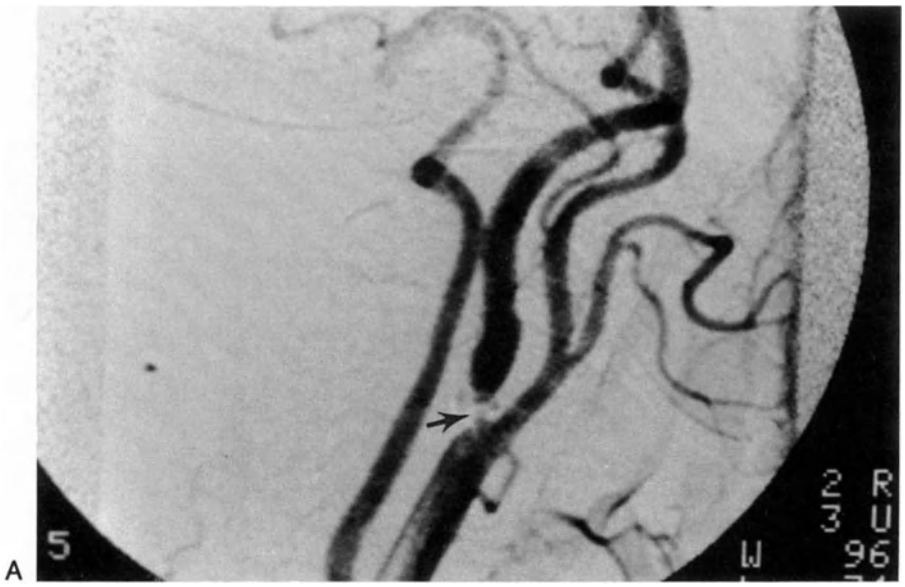
D

FIGURE 5.17D. Specimen shows an organized clot at the ulcer (two arrows) with proximal (triangular arrow) and distal (two triangular arrows) endpoints.



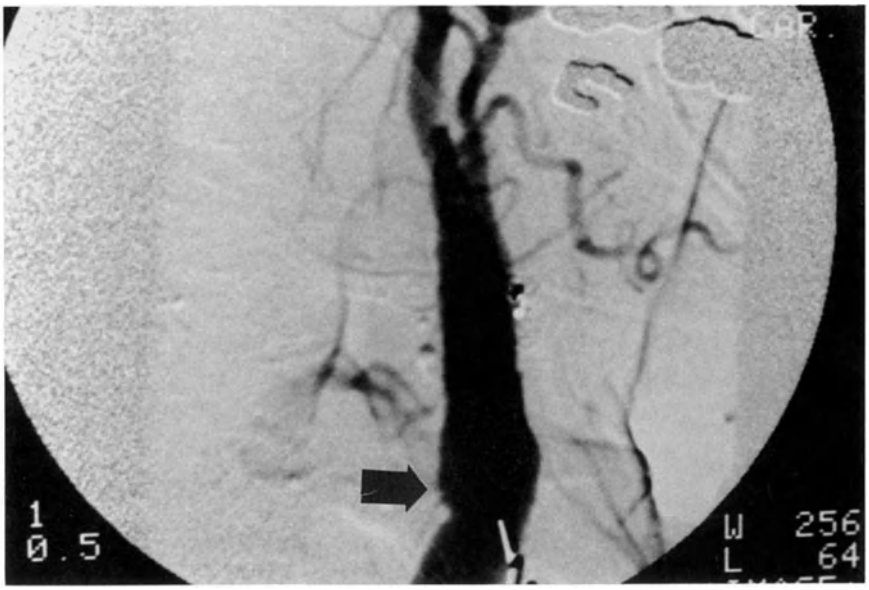
E

FIGURE 5.17E. Vein patch with proximal (triangular arrow) and distal (two triangular arrows) ends.



A

FIGURE 5.18A. Angiogram shows high grade stenosis at the right internal carotid artery (arrow).



B

FIGURE 5.18B. Angiogram of the same patient shows one-year-old carotid endarterectomy and vein patch with proximal end (arrow).

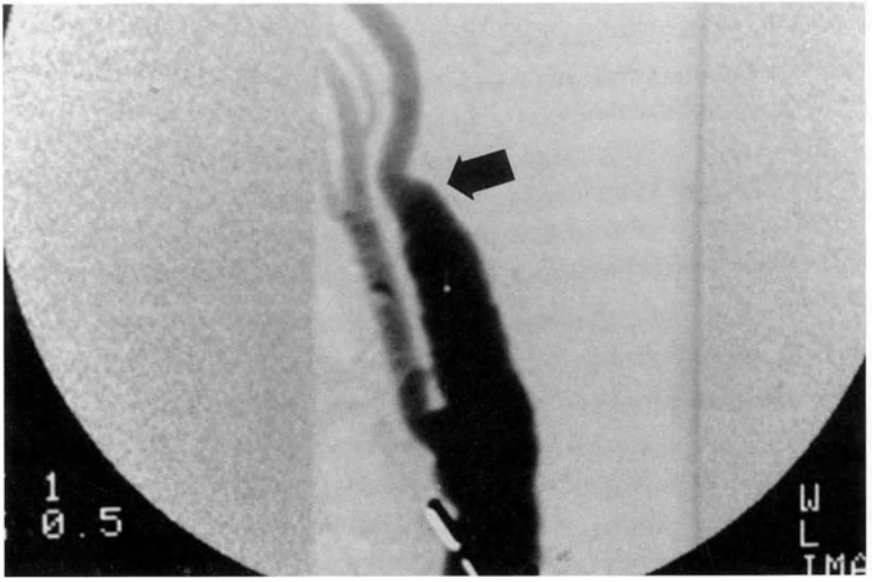


FIGURE 5.18C. The same angiogram shows vein patch with the distal end (arrow).

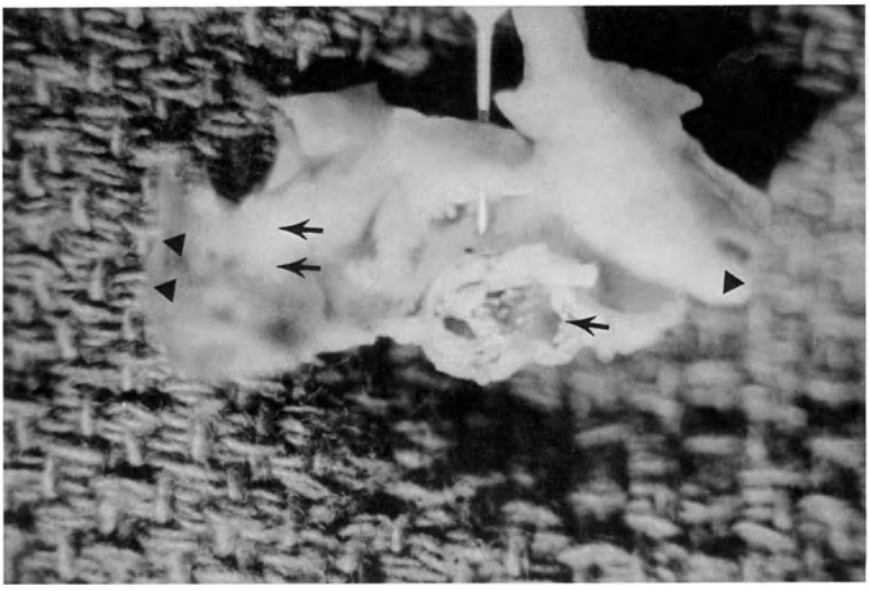
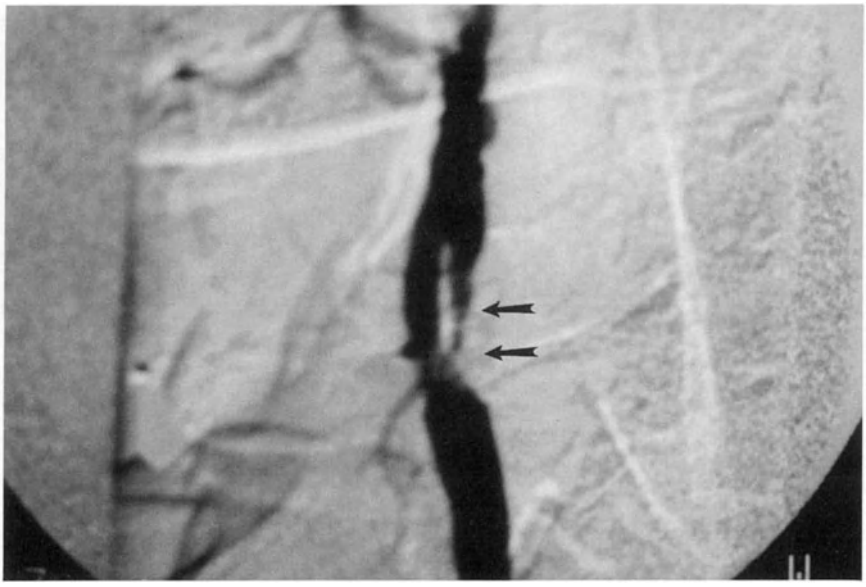
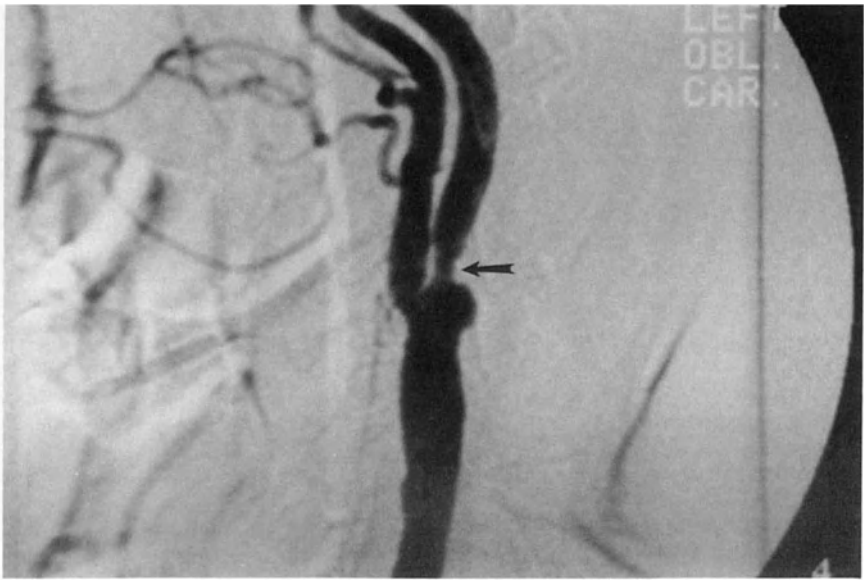


FIGURE 5.18D. Specimen shows high grade stenosis (two arrows) and subintimal dissection and hemorrhage (arrow) with proximal (triangular arrow) and distal (two triangular arrows) endpoints.



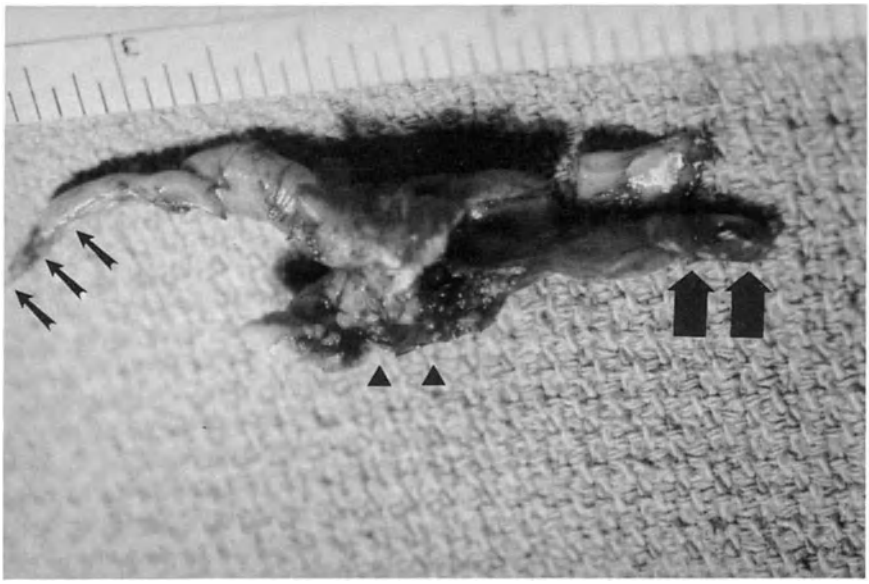
A

FIGURE 5.19A. Angiogram shows a long segment of stenotic lesion at the right internal carotid artery (two arrows).



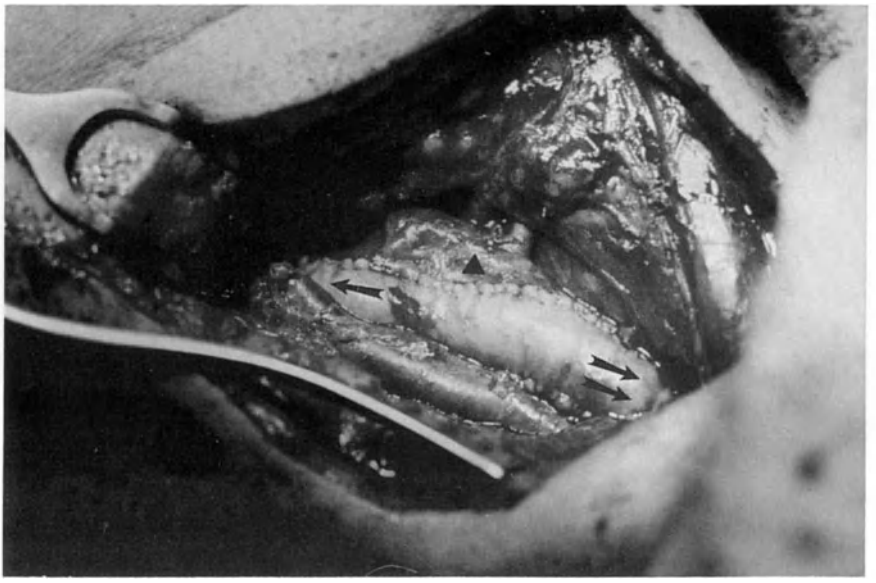
B

FIGURE 5.19B. Angiogram of the same patient shows moderate stenosis at the left internal carotid artery (arrow).



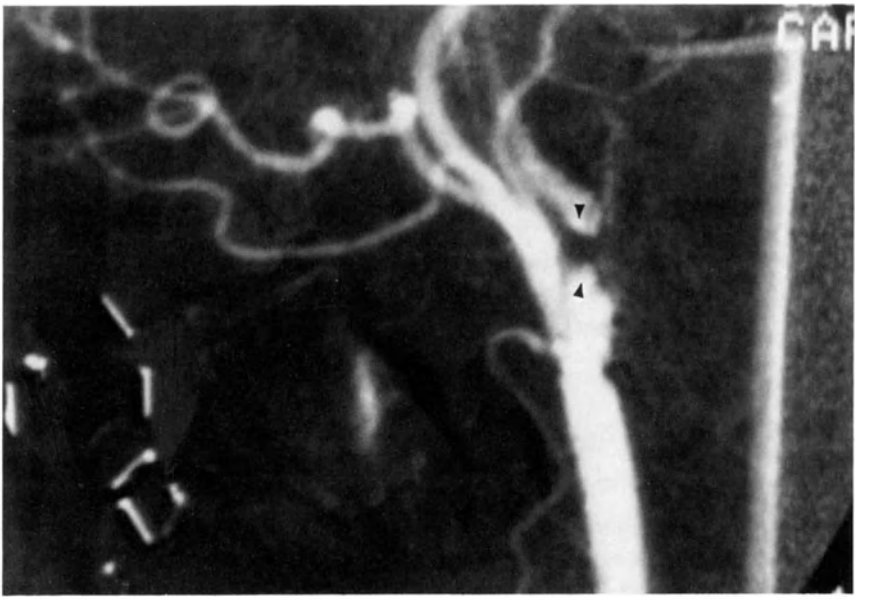
C

FIGURE 5.19C. Specimen shows severe stenosis with ulcers (two triangular arrows), extended plaque into the distal internal carotid artery (three arrows). The proximal end of the specimen is on the right hand side (two big arrows).



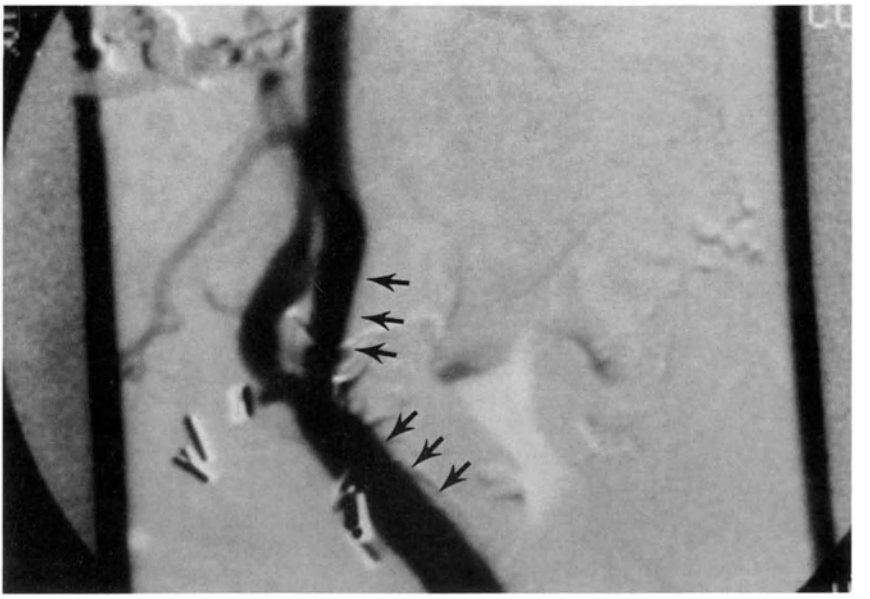
D

FIGURE 5.19D. Vein patch after endarterectomy with proximal (two arrows) and distal (one arrow) ends. External carotid artery (triangular arrow) is seen in relationship with the vein patch.



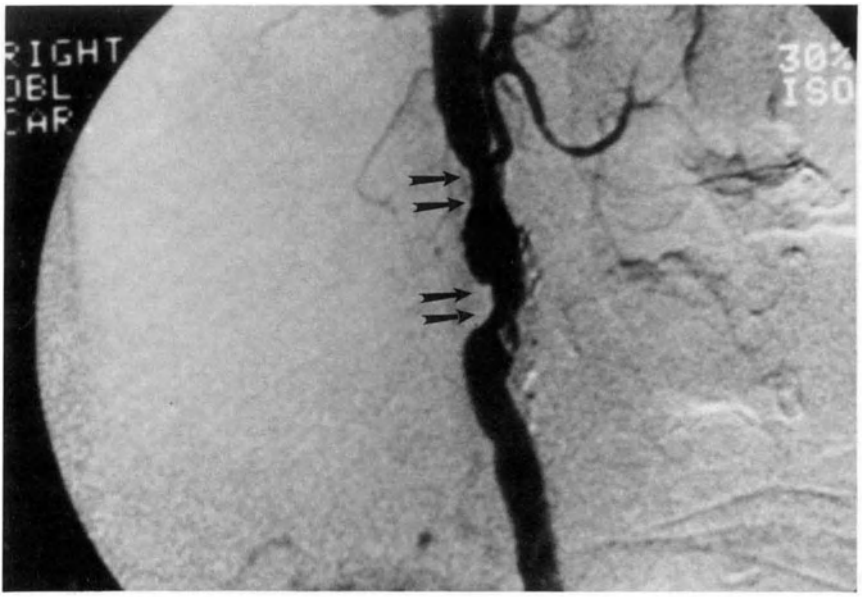
A

FIGURE 5.20A. Angiogram shows high grade stenosis with near occlusion at the left internal carotid artery (triangular arrows).

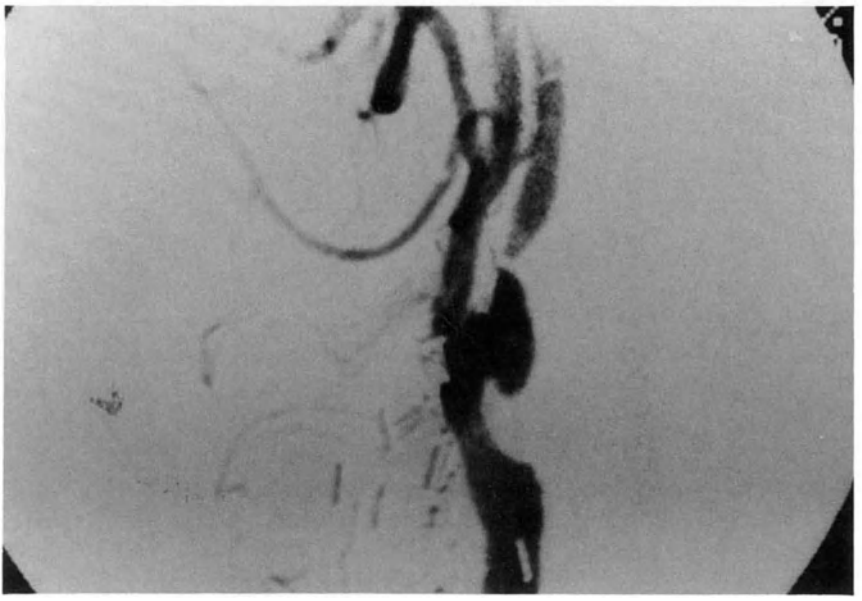


B

FIGURE 5.20B. Angiogram of the same patient with status post-right carotid endarterectomy with widely patent common-internal carotid artery system (arrows) for the past 12 years after surgery with primary closure.



A



B

FIGURE 5.21A,B. Angiograms show restenosis nine years after carotid endarterectomy and vein patch (arrows).

References

1. Eastcott HHG, Pickering GW, Rob CG. Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia. Lancet 1954, 2:994–996.
2. Callow AD, Mackey WC. Long-term follow-up of surgically managed carotid bifurcation atherosclerosis. Ann Surg 1989, 210:308–316.
3. Pokras R, Dyken ML. Dramatic changes in the performance of endarterectomy for diseases of the extracranial arteries of the head. Stroke 1988, 19:1289–1290.
4. Baker WH, Littooy FN, Greisler HP, et al. Carotid endarterectomy in private practice by fellowship-trained surgeons. Stroke 1987, 5:957–958.
5. Sundt TM Jr, Whisnant JP, Houser OW, Fode NC. Prospective study of the effectiveness and durability of carotid endarterectomy. Mayo Clin Proc 1990, 65:625–635.
6. Thompson JE. Carotid endarterectomy for asymptomatic carotid stenosis: An update. J Vasc Surg 1991, 13:669–676.
7. Maini BS, Mullins TF III, Catlin J, O'Mara P. Carotid endarterectomy: A ten-year analysis of outcome and cost of treatment. J Vasc Surg 1990, 12:732–740.
8. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. N Engl J Med 1991, 325:445–453.
9. Javid H, Ostermiller WE, Hengesh JW, et al. Natural history of carotid bifurcation atheroma. Surgery 1970, 67:80–86.
10. De Bakey ME. Patterns of atherosclerosis and rates of progression. Atherosclerosis Rev 1978, 3:1–56.
11. Chang, JB. Extracranial revascularization. In: Vascular Surgery (Chang JB, Ed), ch 5, Spectrum Publications Inc 1985.
12. European Carotid Surgery Trialists' Collaborative Group. MRC European Carotid Surgery Trial: Interim results for symptomatic patients with severe (70–99%) or with mild (0–29%) carotid stenosis. Lancet 1991, 337:1235–1243.
13. Lord RSA, Raj TB, Stary DL, et al. Comparison of saphenous vein patch, polytetrafluoroethylene patch, and direct arteriotomy closure after carotid endarterectomy. Part I. Perioperative results. J Vasc Surg, 9:521–529.
14. Katz MM, Jones GT, Degenhardt J, et al. The use of patch angioplasty to alter the incidence of carotid restenosis following thromboendarterectomy. J Cardiovasc Surg 1987, 28:2–8.
15. Deriu GP, Ballotta E, Bonavina L, et al. The rationale for patch-graft angioplasty after carotid endarterectomy. Early and long-term follow-up. Stroke 1984, 15:972–979.
16. Ouriel K, Greene RM. Clinical and technical factors influencing recurrent carotid stenosis and occlusion after endarterectomy. J Vasc Surg 1987, 5:702–706.
17. Little JR, Bryerton BS, Furlan AJ. Saphenous vein patch grafts in carotid endarterectomy. J Neurosurg 1984, 61:743–774.
18. Eikelboom BC, Ackerstaff RGA, Hoeneveld H, et al. Benefits of carotid patching: A randomised study. J Vasc Surg 1988, 7:240–247.
19. Sundt TM Jr, Houser IW, Fode NC, Whisnant JP. Correlation of postopera-

tive and two-year follow-up angiography with neurological function in 99 carotid endarterectomies in 86 consecutive patients. Ann Surg 1986, 203:90–100.
20. Govostis DM, Bandyk DF, Bergamini TM, Towne JB. Biochemical adaptation of venous patches placed in the carotid circulation. Arch Surg 1989, 124:490–493.
21. Bush HL, Hong SI, Deykin D, et al. The effect of surgical trauma on prostacyclin production by vein grafts. Surg Forum 1982, 33:463–465.
22. Eldor A. Hoover EL, Pett SB, et al. Prostacyclin production by arterial autogenous vein grafts in dogs. Prostaglandins 1981, 22: 485–498.
23. Bush HL Jr, Jakubowski JA, Curl GR, et al. The natural history of endothelial structure and function in arterialized vein grafts. J Vasc Surg 1984, 3:204–215.
24. Rosenthal D, Archie JP Jr, Garcia-Rinaldi R, et al. Carotid patch angioplasty: Immediate and long-term results. J Vasc Surg 1990, 12:326–333.
25. Clagett GP, Patterson CB, Fisher DF Jr, et al. Vein patch versus primary closure for carotid endarterectomy. J Vasc Surg 1989, 9:213–223.
26. Mock CN, Lilly MP, McRae RG, Carney WI Jr. Selection of the approach to the distal internal carotid artery from the second cervical vertebra to the base of the skull. J Vasc Surg 1991, 13:846–853.
27. Dossa C, Shepard AD, Wolford DG, Reddy DJ, Ernst CB. Distal internal carotid exposure: A simplified technique for temporary mandibular subluxation. J Vasc Surg 1990, 12:319–325.
28. Purdue GF, Pelligrini RV, Arena S. Aneurysms of the high internal carotid artery: A new approach. Surgery 1981, 89:268–270.
29. Welsh P, Pradier R, Repetto R. Fibromuscular dysplasia of the distal cervical internal carotid artery. J Cardiovasc Surg 1981, 22:321–326.
30. Fisch UP, Oldring DJ, Senning A. Surgical therapy of internal carotid artery lesions at the skull base and temporal bone. Otolaryngol Head Neck Surg 1980, 88:548–554.
31. Fry RE, Fry WJ. Extracranial carotid artery injuries. Surgery 1980, 88:581–586.
32. Fisher DF Jr, Clagett GP, Fry RE, et al. Mandibular subluxation for high carotid exposure. J Vasc Surg 1984, 1:727–733.
33. Hans SS, Shah S, Hans B. Carotid endarterectomy for high plaques. Am J Surg 1989, 157:431–434.

6
Techniques for Transthoracic Reconstruction of the Supraaortic Trunks

RAMON BERGUER

The supraaortic trunks (SAT) are the branches of the aorta extending up to the take-off of the vertebral arteries and to the carotid bifurcations.

Reconstruction of these vessels is usually done using the cervical approach, generally with horizontal bypasses that often cross the midline. In the seventies the popularity of the cervical approach was derived in part from data indicating that direct anatomic reconstruction through the sternum was riskier and also from lack of familiarity with the transthoracic approach. Today, improvements in anesthetic and surgical technique have brought the mortality of transthoracic reconstructions down to 3–5%, similar to that noted following cervical reconstructions.

Reconstructions of the SAT have become more frequent. Patients with multiple SAT lesions are seen more often since four-vessel arteriography, including two views of the arch, is part of the standard work-up for patients with cerebrovascular symptoms. In addition, it is accepted that about half, if not more, of cerebrovascular ischemic pathology is caused by thromboembolization as opposed to restriction of blood flow. Ulcerative disease of the innominate artery, without severe stenosis, may cause the same symptoms as a similar carotid bifurcation plaque. Since techniques for extrathoracic reconstruction of the supraaortic trunks do not always permit an *easy exclusion* bypass of thromboembolic lesions, particularly of the innominate artery, indications for transthoracic reconstruction have increased.

Today we limit the extrathoracic approach to patients who present with symptomatic and *single* lesions of the subclavian or common carotid arteries. These patients usually undergo a transposition of the involved vessel into the neighboring subclavian or common carotid artery, whatever the case may be. We have practically abandoned the technique of carotid subclavian bypass for proximal subclavian artery disease except in patients who have had a left internal mammary anastomosis to a coronary artery and now have either coronary or arm symptoms because of stenosing disease of the proximal subclavian artery. These patients must be managed by carotid subclavian bypass. The cervical approach is also considered in patients who had a previous midsternotomy, particularly if done for an aorto-

coronary bypass operation, and in those patients who have poor pulmonary reserve. In the first case reentering the mediastinum is fraught with complication, and injury to a previously placed aortocoronary bypass could be fatal.

The transthoracic approach is recommended for severely stenosing or embolizing lesions of the innominate artery and for the synchronous repair of multiple lesions involving the innominate, subclavian and common carotid arteries. It is also the technique of choice in patients who are to undergo an aortocoronary revascularization and have severe and multiple disease of the SAT. Both operations can be done in the same setting. If present, the existence of severe left proximal subclavian disease should be repaired at the same time as the other symptomatic trunks if the patient is a potential candidate for coronary artery reconstruction in the future, to permit a later left internal mammary transposition.

By far the most common lesion involving the SAT is an atherosclerotic plaque. The second, but much less common indication, is Takayasu's disease, which typically involves all the SAT but spares the vertebral artery. The latter is often the remaining avenue for brain supply in these patients. Rarely, a posttraumatic aneurysm of the innominate artery or a chronic embolus, lodging in one or more of the SAT, may constitute a surgical indication.

Transsternal Reconstruction of the SAT

The incision is the standard midsternotomy usually prolonged for an inch or two into the right side of the neck to obtain easy access to the bifurcation of the innominate artery below the strap muscles. If the bypass is to be tented to a carotid bifurcation, the incision is prolonged anterior to the sternocleidomastoid on the appropriate side. If both carotid bifurcations need to be exposed, the incision is usually prolonged into the right side, up to the carotid bifurcation. A separate carotid incision on the left side will be used to access the left carotid bifurcation. If the reconstruction is prolonged to the carotid bifurcation on one side, usually the right, the head is slightly turned to the left. If both carotid bifurcations need to be approached, the nasotracheal tube is brought over the midline and taped to the forehead to permit rotation of the head to both sides for the carotid anastomoses. The Swan-Ganz catheter is placed through the left side for reconstructions involving the innominate artery or left common carotid artery. If the left subclavian artery is one of the SAT to be revascularized, the Swan-Ganz catheter is inserted through the femoral approach, since exposure of the left subclavian through the midline requires division of the brachiocephalic vein.

After division of the sternum and hemostasis of its periosteal edges, the thymus is dissected through its midline and preserved for ulterior coverage of the prosthesis at the time of closure. The brachiocephalic vein is dis-

sected and any thymic or thyroid venous branches entering it are divided to free the vein and permit its mobilization. On the right side the strap muscles are divided to expose the bifurcation of the innominate artery and the proximal portion of its two branches. The pericardium is opened from its reflection, usually about the take-off of the innominate artery, down to the right ventricular surface. The pericardial edges are cauterized and gently tented up with stay sutures. The pericardial stays on the right side should not be pulled tight to avoid interference with the drainage of the superior vena cava.

The appropriate distal vessels are dissected. The dissection of the innominate artery bifurcation must be done with care to avoid injuring the recurrent nerve. The latter can be injured both when dissecting the proximal right subclavian artery and when freeing the posterior wall of the most proximal right common carotid artery. The proximal common carotid and subclavian arteries are slung with tapes. If one of the distal anastomosis of the graft is going to be made to the right carotid bifurcation, the latter is dissected at this point. On the left side, if the disease of the common carotid artery is located below the strap muscle, the artery is exposed through the chest. If the disease of the common carotid artery extends distal to the strap muscles, the left carotid bifurcation is approached through a separate neck incision for the distal anastomosis of the left limb of the graft.

Contrary to common belief, the proximal segment of the left subclavian artery can be approached safely through an anterior sternotomy. This exposure requires division of the brachiocephalic venous trunk to permit further opening of the sternal retractor. The proximal left subclavian artery can then be exposed up to the take-off of the left vertebral artery.

Endarterectomy of the Innominate Artery

We use this technique exclusively in individuals who have lesions involving the distal half of the innominate artery. In those who have lesions of the ostium of the innominate artery, which is the most frequent situation, the proximal break-off of the plaque during endarterectomy often presents technical problems. More importantly, the exclusion clamping of the innominate artery may result in involuntary occlusion of the ostium of the neighboring left common carotid artery or in the fracture of the plaque at its origin.

The classic description by Wylie involves clamping the origin of the right common carotid and right subclavian arteries as well as the origin of the innominate artery, the latter with a partial exclusion clamp (the "J" clamp) especially designed for this purpose. The endarterectomy should not be carried deep into the external elastic lamina because this could lead to a frail wall, which may be difficult to close and may cause bleeding. In most

cases the endarterectomy is terminated proximally by cutting the plaque and tacking the intima about the origin of the innominate artery with a continuous suture. Failure to tack the intima may result in a flap or subintimal dissection of the aortic arch. The appeal of this operation is that it reestablishes anatomic continuity and does not require the use of a prosthetic. Its indications, however, are limited.

Ascending Aorta to SAT Bypass

This is the technique most frequently used. The dissection does not need to include the origin of the innominate artery or of the left common carotid artery—it is limited proximally to the proximal ascending aorta where the quality of this artery is best, and distally to the recipient vessels, whether they be in the chest or in the neck.

Once an appropriate segment of the ascending aorta has been isolated, the systemic pressure is pharmacologically lowered with nitroprusside or isoflurance to 100–110 mm Hg systolic. Using a Lemole-Strong clamp (Fig 6.1) an adequate portion of the wall is excluded. The arterial line monitor is observed, noting if there is any change in systolic pressure. If this is the case it is likely that too much aorta has been excluded, causing outflow obstruction and requiring replacement of the clamp. Once the clamp is applied the excluded aorta is aspirated with a needle, seeing that it does not fill again. An arteriotomy is made and a small sliver of the wall is excised to facilitate anastomosis.

Choice of Graft

The graft we use is PTFE although a collagen-impregnated Dacron graft is also suitable. When there is more than one trunk to be inserted, some have advocated the use of an inverted bifurcation graft. This is not the best solution because the geometry of the bifurcation graft is such that it will usually result in crowding of the anterior mediastinum and compression of the airway or venous structures. If one were to use an inverted bifurcation, the limbs that will match an innominate artery or carotid are generally a size 10 or 8. This will call for a prosthesis with a trunk of size 20 or 16, which, after beveling for anastomosis, will require an unnecessarily large aortotomy, will occupy too much space in the anterior mediastinum, and is more likely to kink against the sternum or compress the anterior mediastinal structures.

Our choice is a size 10 graft anastomosed to the ascending aorta. If the artery to be bypassed is the innominate, a size 10 prosthesis will match well the size of the distal anastomosis. To these, main graft branches of size 8 are added that will extend to the other SAT.

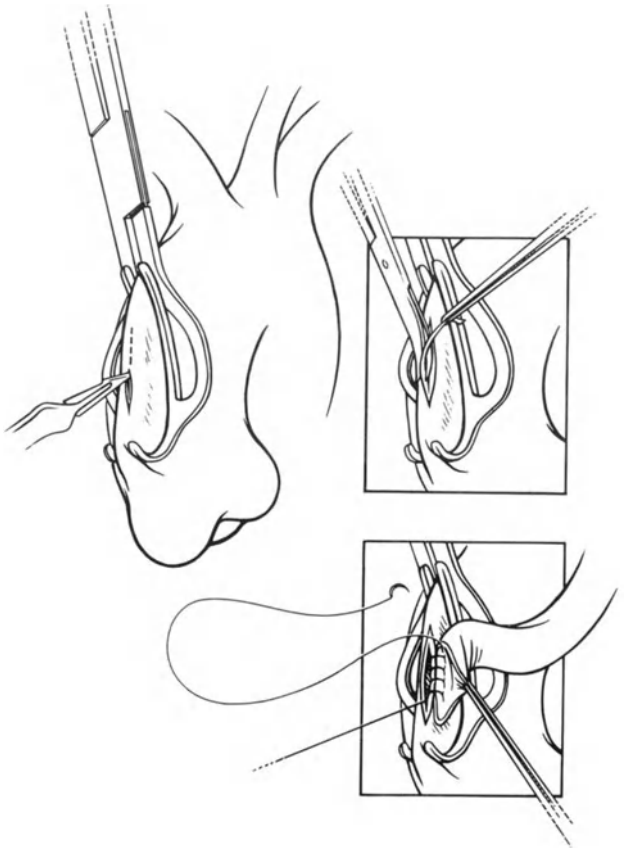


FIGURE 6.1 Partial exclusion clamping of the ascending aorta and aortotomy. A small sliver of the aortic wall is excised and the prosthesis is anastomosed to it with open technique.

The proximal anastomosis is done to the ascending aorta using a continuous 3-0 polypropylene (Fig. 6.1). If the wall of the aorta is friable, it is sandwiched between a Teflon strip outside and the graft inside. When testing the proximal anastomosis, the patient is placed in Trendelenburg to avoid causing embolization from any pocket of air in the proximal graft that may escape into the ascending aorta rather than into the graft being purged. The graft is then clamped immediately above the anastomosis, and the patient is returned to the normal position. If any branches (size 8) are to be tented to the left common carotid or left subclavian artery, they are sewn into the graft at this point. The anastomosis of a branch to the main graft is done as low as possible to avoid kinking and crowding of the high portion of the thoracic inlet. Once the side branch (or branches) has been

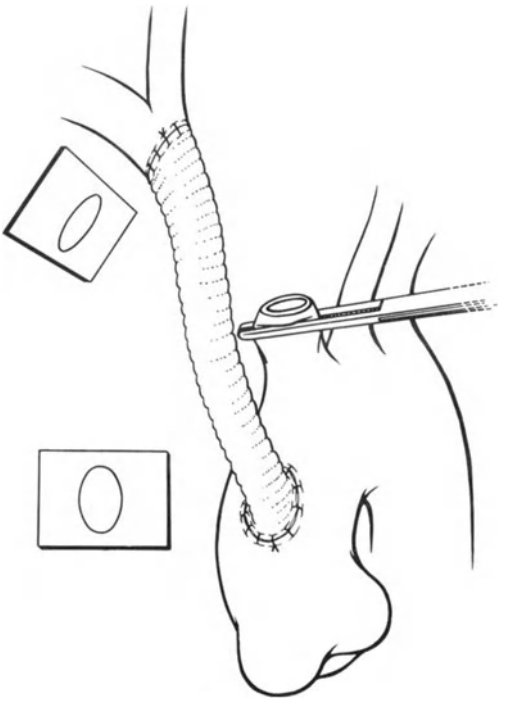


FIGURE 6.2. In an aorto-innominate bypass the planes of the anastomoses at both ends of the graft are at 90° from each other.

anastomosed to the main prosthesis, it is excluded with a clamp before proceeding with the distal anastomosis of the main graft.

At this point 7000 units of of intravenous heparin are given, and the distal vessels are prepared for anastomosis. If the distal anastomosis is to be done to the innominate artery bifurcation (Fig. 6.2), the right common carotid and subclavian arteries are clamped. The innominate artery is clamped in its midportion and divided above this clamp. If the bifurcation of the innominate artery is free of disease, the distal anastomosis is done in an end-to-end manner using 5-0 polypropylene. It is best to use a thin needle to suture this vessel which has a rather friable wall. The size 10 prosthetic tube lies better when it rests on top of the brachiocephalic vein. Following the distal anastomosis and after appropriate backward and forward bleeding, flow is resumed (Fig. 6.3), first into the arm and then into the common carotid artery.

A number of configurations (Fig. 6.4) may be used for the distal anastomosis of an innominate artery bypass depending on whether the disease extends into the common carotid, subclavian artery or both.

There are two techniques that may be used if the distal anastomosis on

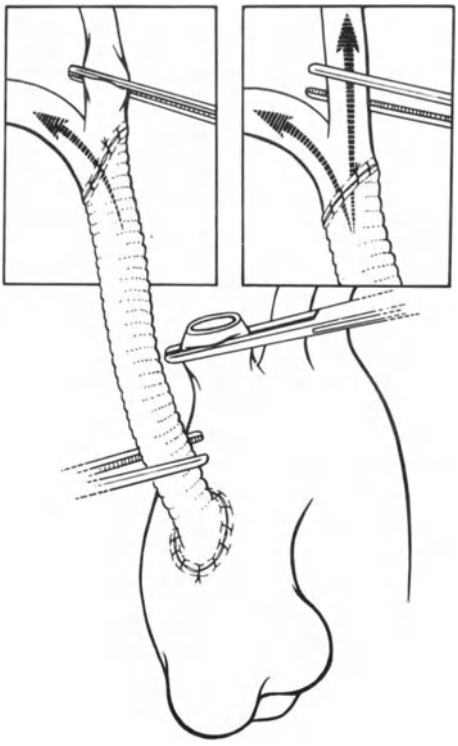
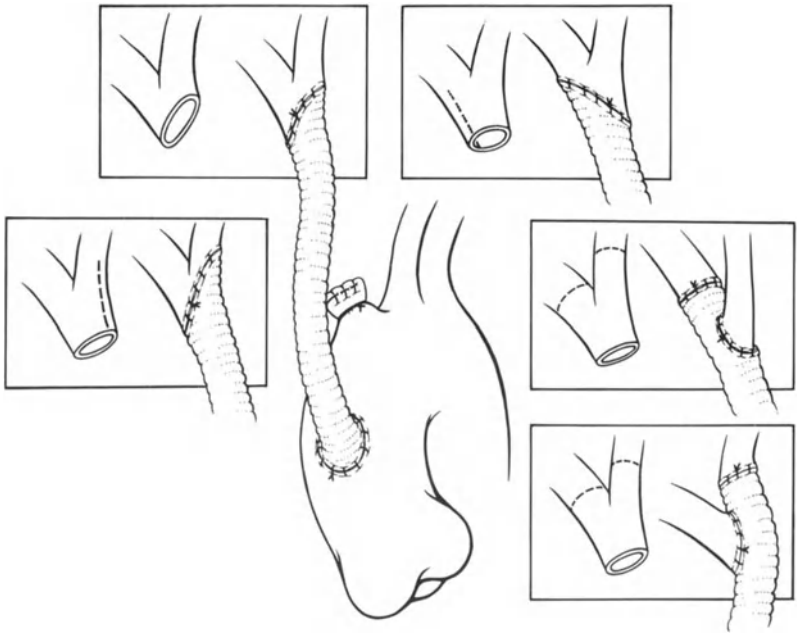


FIGURE 6.3. Sequence of declamping after completing the anastomoses of an aorto-innominate bypass.



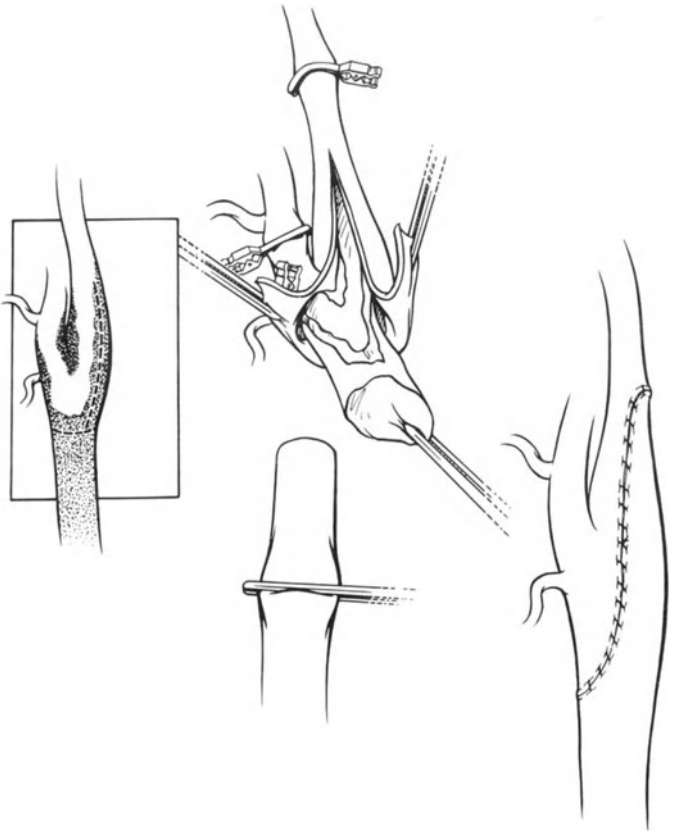


FIGURE 6.5. Distal anastomosis of the limb of a prosthesis to the carotid bifurcation after transsection and eversion endarterectomy of the latter.

either side is to be made to a carotid bifurcation. One is to transsect the common carotid artery and to open the carotid bulb posteriorly (Fig. 6.5). This permits an eversion endarterectomy of the internal and external carotid arteries. The graft is then sewn end-to-end onto this arteriotomy as a patch extending into the internal carotid artery. While this technique is sound, it is sometimes noted in follow-up arteriograms that the elongation inevitably occurring after transsection of the bifurcation and dissection of the internal and external carotid arteries may result in a kink at the tip of the patch. Our preference today is to do a standard endarterectomy of the bifurcation plaque anastomosing the graft end-to-side to it as a patch (Fig.

FIGURE 6.4. Different solutions for the distal anastomosis of a bypass from the ascending aorta to the bifurcation of the innominate artery.

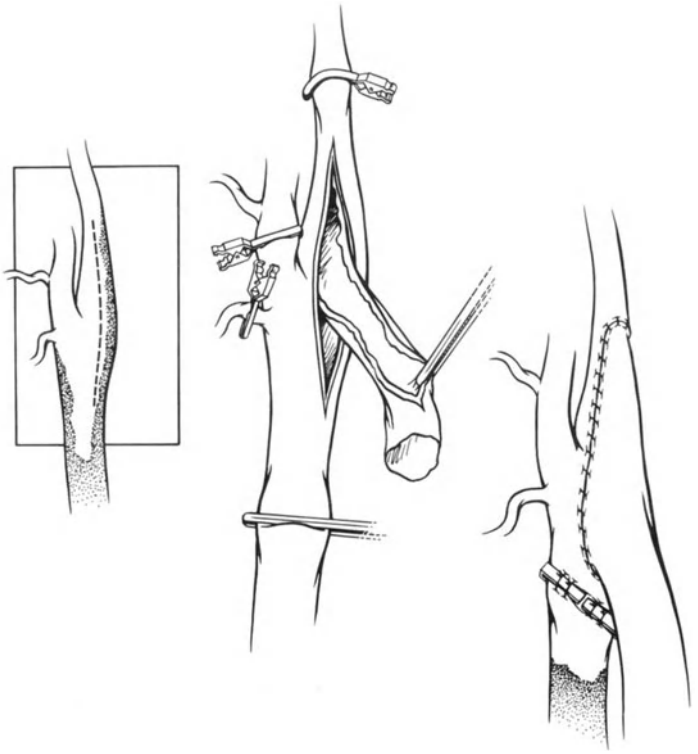


FIGURE 6.6. Distal anastomosis of the prosthesis limb to the carotid bifurcation after a standard arteriotomy and endarterectomy. The distal end of the prosthesis is sewn as a lay-on patch. The end-to-side anastomosis is made functionally an end-to-end by the precise application of two large hemoclips secured with sutures, obliterating the common carotid artery.

6.6). The proximal carotid artery is then excluded with two large hemoclips affixed with sutures transforming this end-to-side into a functional end-to-end anastomosis. Since the bifurcation has not been cut and freed, there is less elongation or tendency to kink in the future.

Once the distal anastomosis of the main graft has been completed, flow is established through it before clamping the opposite carotid. Failure to do so may result in severe brain ischemia. The most frequent reconstruction involves the innominate and left carotid arteries (Fig. 6.7). If all three branches need to be revascularized, the main graft is placed in the innominate artery, and the side branch is extended, usually to the left subclavian artery implanting the left common carotid artery in this branch (Fig. 6.8). Under favorable anatomic circumstances one may also choose to anastomose the side branch to the left common carotid artery, allowing a

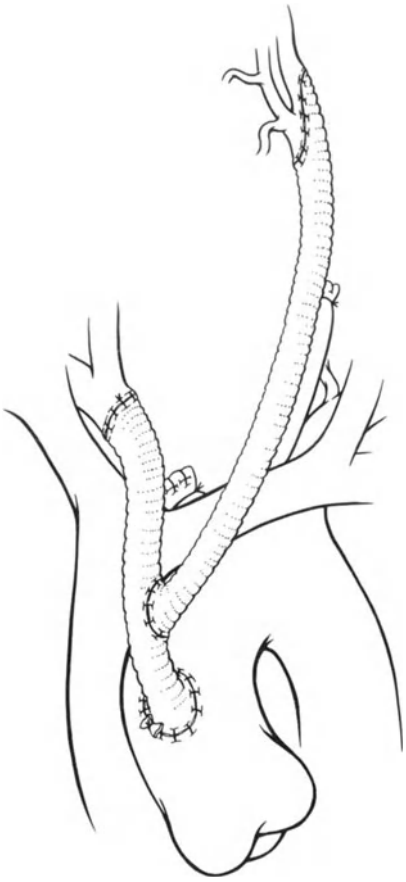


FIGURE 6.7. The most common pattern of revascularization in transthoracic reconstruction of the supraaortic trunks involves the innominate and left common carotid arteries. The anastomosis of the left-sided branch should be low, near the proximal anastomosis of the main bypass graft, so the the prosthesis emerges from the mediastinum on the left side of the trachea.

bit of a loop that will permit an end-to-side anastomosis of the proximal left subclavian to this graft.

The closure of the stump of the IA is done after reestablishing flow through the bypass into the latter artery. The stump is closed with a mattress suture in one direction, completed with an over-and-over suture in the opposite direction.

When side branches extend to the left side, particularly to the subclavian, it is important to loosen the sternal retractor to estimate the optimal length of this branch. Failure to do so may result in redundancy or kinking

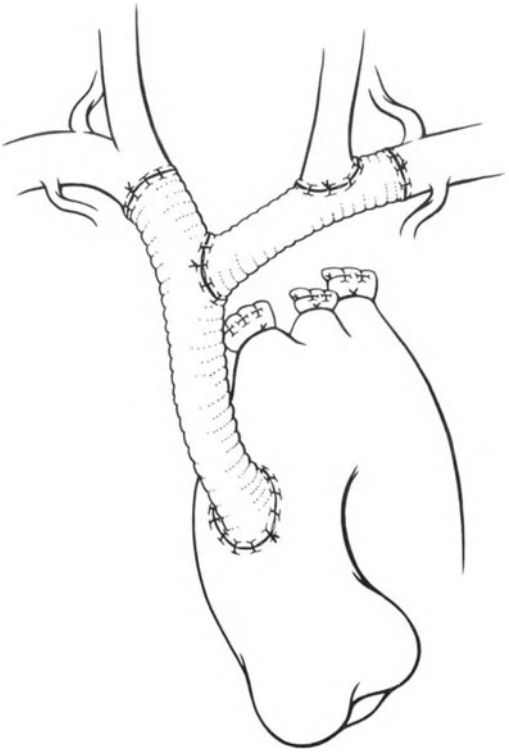


FIGURE 6.8. One of the solutions to the revascularization of the three supraaortic trunks.

of the side branch when the sternum is closed. Once the anastomoses are completed heparin is reversed. The integrity of the pleural space is checked. If the pleura has not been entered, a single mediastinal tube is left in the low mediastinum and brought through a subxiphoid stab wound. The thymus is approximated to cover the graft in the midline, separating it from the sternum. Above the thymus the strap muscles may also be approximated to cover the graft. The sternum is closed with wires, and the skin and subcutaneous tissue are closed with polyglycolic suture.

Transthoracic Reconstruction of the Left Subclavian Artery

The few indications for a transthoracic reconstruction of the left subclavian artery are the need to repair this vessel in a patient in whom a previous neck reconstruction, usually a carotid-subclavian bypass, has failed, and in

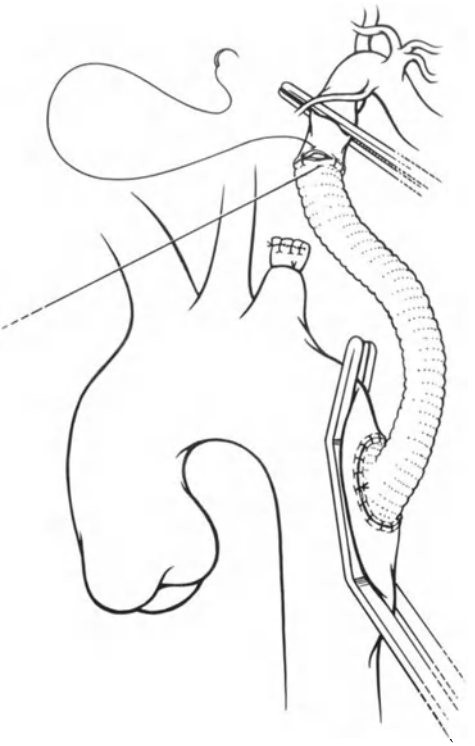


FIGURE 6.9. Revascularization of the left subclavian artery through a left lateral thoracotomy. The proximal anastomosis is constructed into the ascending aorta by partial exclusion clamping. The distal anastomosis is end-to-end to the first segment of the subclavian artery.

individuals who have had neck irradiation or sepsis from a previous supraclavicular operation, making the dissection of the subclavian artery hazardous.

The patient is intubated with a Carlens tube to decompress the left lung during the operation. The approach is a left lateral thoracotomy through the fourth intercostal space. After depressing the lung, the subclavian is identified at the dome of pleura. The first segment of the subclavian artery is dissected beneath the pleura, taking care not to injure the phrenic and vagus nerves that cross in front of the left subclavian artery. The superior intercostal vein runs between these two nerves.

Direct endarterectomy of the subclavian artery is hardly ever used. It requires dissection of the distal portion of the arch and often presents problems with the break-off of the plaque at the level of the aorta. Our preference is a bypass originated in the descending aorta. Once the subclavian has been dissected, a segment of the proximal descending aorta is isolated

for enough length to permit placement of a lateral exclusion clamp (Fig. 6.9). Once the clamp is applied the adequacy of this lateral clamping is checked by a good distal pressure through a femoral arterial line. After a needle aspiration indicates that the exclusion clamping is adequate, an aortotomy is made and a size 8 PTFE graft is anastomosed using a 3-0 polypropylene suture. After testing this anastomosis the patient is systemically heparinized. If a corrugated collagen Dacron graft is used, the anastomosis is tested and the graft is clamped distally, allowing it to distend under pressure to assess the proper length needed to reach the subclavian artery. The subclavian artery is cross-clamped and divided, and the graft is anastomosed to it with 5-0 polypropylene suture. At the conclusion of the anastomosis, flow is reestablished and heparin is reversed. High and low chest tubes are left in the left thoracic cavity, and the thoracotomy is closed.

7
Techniques for Reconstruction of the Vertebral Artery

RAMON BERGUER

The lesion most commonly affecting the vertebral artery (VA) is a plaque stenosing its takeoff from the subclavian artery. This lesion alone is responsible for approximately 70% of all VA reconstructions. In its intraspinal course the VA is affected by atherosclerosis and by other entities; however, from C_2 up to the atlanto-occipital membrane, where the artery becomes intradural, the artery is usually spared from disease. The correction of compression or occlusion of the VA in its intraspinal course is done by accessing the distal vertebral artery at the C_2–C_1 intervertebral space. This segment of the artery, as mentioned above, is seldom involved by disease and is the widest gap between any of the transverse processes. This permits the surgeon to work in the distal VA with relative ease. Very rarely, usually in the course of repairing an aneurysm, will the VA have to be dissected above the transverse process of C_1 in its horizontal portion below the occipital bone.

Although the gist of this chapter is the discussion of technical options for the repair of the VA, it is worth stressing beforehand that the standard 4-vessel arteriogram is often inadequate to identify all the potential lesions of the extracranial VA. A full arteriographic study of the various intrinsic and extrinsic lesions involving the VA often requires selective catheterization of the parent subclavian artery, obtaining films with the neck and body in special positions. It is also pertinent to note that unless the surgeon is dealing with a thromboembolic lesion, unilateral VA lesions in patients who have a normal contralateral VA do not constitute a surgical indication. When patients are operated to correct flow-restrictive lesions, these must be severe and involve both vertebrals or the clearly dominant one.

Technique For Proximal Vertebral Artery Reconstruction

There are two techniques to correct severe lesions of the ostium of the VA. The best and simplest technique is the transposition of the proximal VA to

the posterior wall of the adjacent common carotid artery. In our practice, this technique is used in over 90% of proximal VA reconstructions. There are, however, circumstances where cross-clamping of the common carotid artery is not desirable, such as when the contralateral internal carotid artery is occluded or when the ipsilateral common carotid artery is severely diseased. In such cases the reconstruction of the VA is based on the ipsilateral subclavian, either by tending a subclavian-vertebral bypass or, if there is enough redundancy in the VA, transposing it to another subclavian site.

For a transposition of the proximal VA to the common carotid artery, the VA is approached through the intersternocleidomastoid space. The short oblique incision at the base of the neck, drawn from the head of the clavicle, allows dissection between the two bellies of the sternocleidomastoid as they separate near their lower insertions. The jugular vein encountered at the bottom of the field is retracted laterally with the vagus nerve. The common carotid artery is dissected and slung with silastic tape. The surgeon then exposes the common carotid artery as far down into the mediastinum as feasible. The thoracic duct is identified, ligated, and cut as it emerges from behind the common carotid artery to curve transversely into the left jugulo-subclavian confluent. On the right side accessory ducts are regularly seen. These lymphatic vessels are ligated and then divided to avoid a postoperative lymph leak. The main anatomical landmark for

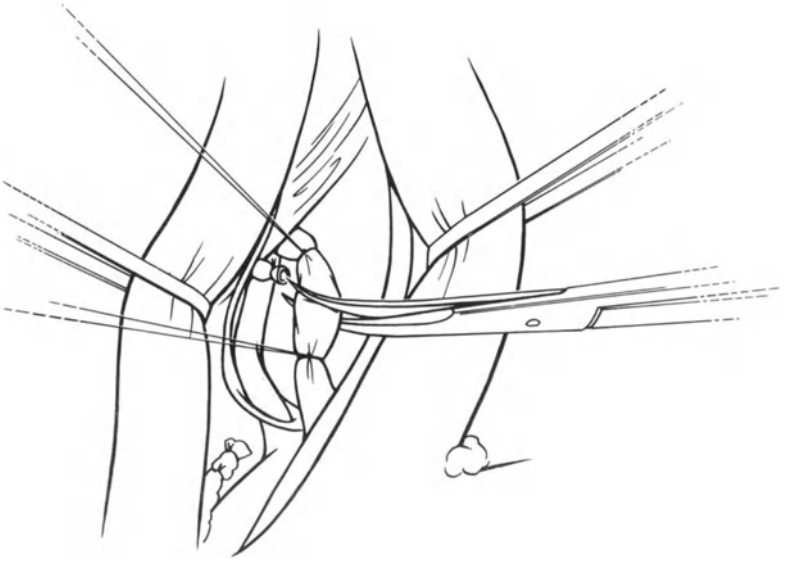


FIGURE 7.1. The approach to the proximal vertebral artery. The division of the vertebral vein permits identification of the underlying vertebral artery.

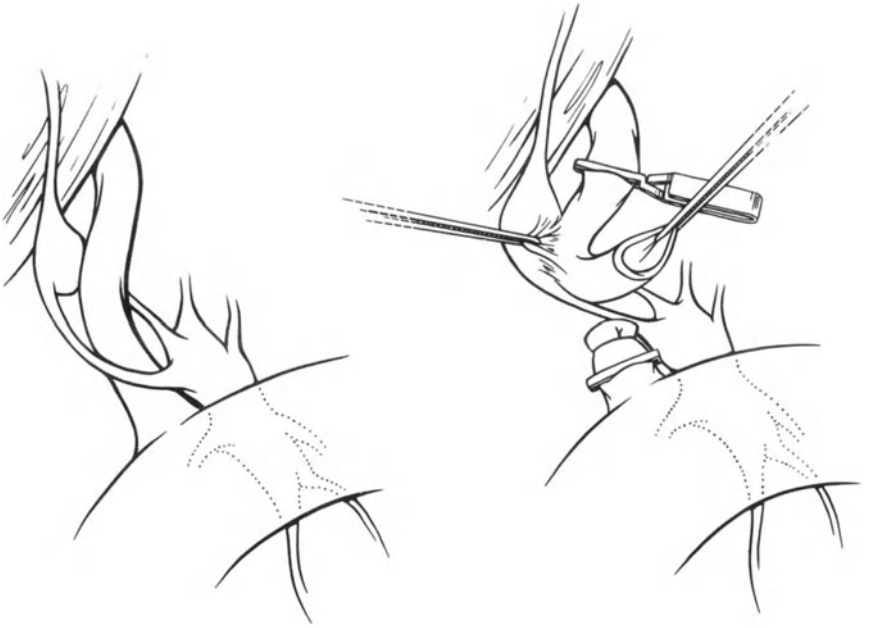


FIGURE 7.2. Left: Relationship of the lower sympathetic ganglia to the vertebral artery. Right: After division of its origin the artery is passed through this loop, freeing any attachments that might exist between the sympathetics and the adventitia of the artery without damaging either.

finding the VA is the vertebral vein. Once this vein is identified it is doubly ligated and divided (Fig. 7.1). The VA lies beneath it.

The anatomy of the sympathetics as they relate to the VA at the base of the neck merits some mention. The middle cervical ganglion when located low in the neck (below C_6) is also called the intermediate ganglion. The lower cervical ganglion in the majority of patients fuses with the first thoracic ganglion to form the stellate ganglion. The connections between the middle and lower (or stellate) ganglia pass both in front and behind the VA and must be preserved during dissection of its first segment. We normally isolate the artery above and below the sympathetic trunks (Fig 7.2). Afterwards, with the patient systemically heparinized, the VA is divided from the subclavian artery pulling its distal end free through this sympathetic loop while freeing whatever adhesions there may be between the sympathetic nerves and the adventitia of the artery. The VA is then brought into proximity of the common carotid artery for anastomosis. The VA is prepared for anastomosis by spatulating its end. The common carotid artery is cross-clamped and an arteriostomy made in its posterior wall with an aortic punch. The anastomosis of the VA to the common carotid is done by open technique using 7.0 polypropylene suture.

Technique for Distal Vertebral Artery Reconstruction

Reconstruction of the distal VA requires techniques that are lesser known. The approach to the artery is at the level of the C_2–C_1 interval between the transverse processes. Although there are three main technical alternatives for reconstruction of the VA, the approach to the C_2–C_1 segment is the same for all of them and is described below.

The incision is anterior to the sternocleidomastoid. The neck is approached through the retrojugular space dissecting between the jugular vein and the sternocleidomastoid muscle and isolating the spinal accessory nerve. This nerve is followed up to the level of C_1. This prominent bony landmark is easily identified by the palpating finger. After removing some of the fibrofatty tissue that covers the levator scapula, its anterior and posterior edges are identified. The anterior ramus of C_2 or its branches are seen emerging from beneath the anterior edge of the levator scapula (Fig. 7.3).

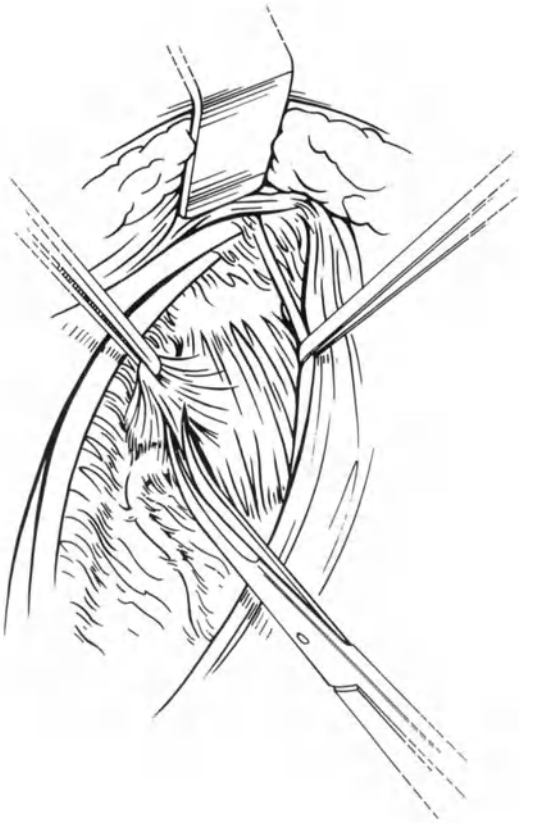


FIGURE 7.3. Identification of the anterior edge of the levator scapula.

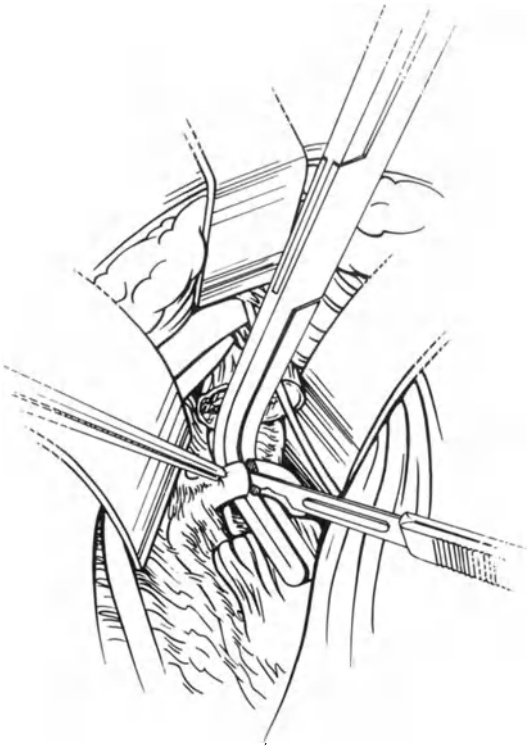


FIGURE 7.4. Once the levator has been divided, the anterior ramus of C_2 can be cut before its bifurcation.

The levator scapula is cut immediately below its insertion at C_1. This permits identification of the trunk of the anterior ramus of C_2. This nerve trunk is cut proximal to its division (Fig. 7.4).

Cutting the anterior ramus of C_2 permits identification of the VA, which runs perpendicularly and beneath it, surrounded by the vertebral veins (Fig. 7.5). The vertebral veins are dissected away from the VA, and the latter is then looped and freed for a sufficient length to construct an anastomosis to it.

Three basic techniques are used in the reconstruction of the distal VA once the segment C_1–C_2 is dissected. The most frequent is a *common carotid to distal VA autogenous saphenous vein bypass*. In this technique, once an appropriate saphenous vein has been obtained, the patient is heparinized and the distal VA is cross-clamped. The anastomosis of the vein graft to the artery is end-to-side (Fig. 7.6) using an open technique and, if the artery is small, interrupted stitches in the upper half. The suture should be 7.0 or 8.0 polypropylene. Once the anastomosis is completed it is back bled and tested. If suitable, the proximal end of the vein graft is

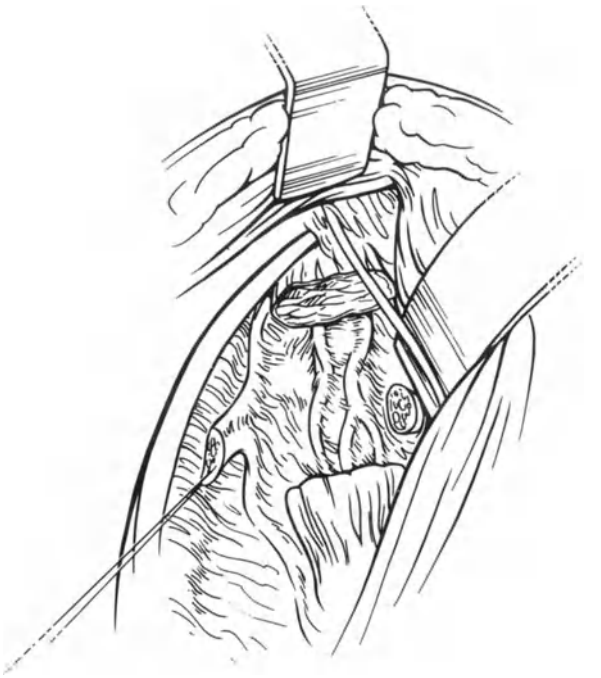


FIGURE 7.5. A suture retracts the distal end of the anterior ramus of C_2. The vertebral artery covered by the vertebral vein plexus is deep to the nerve and perpendicular to it.

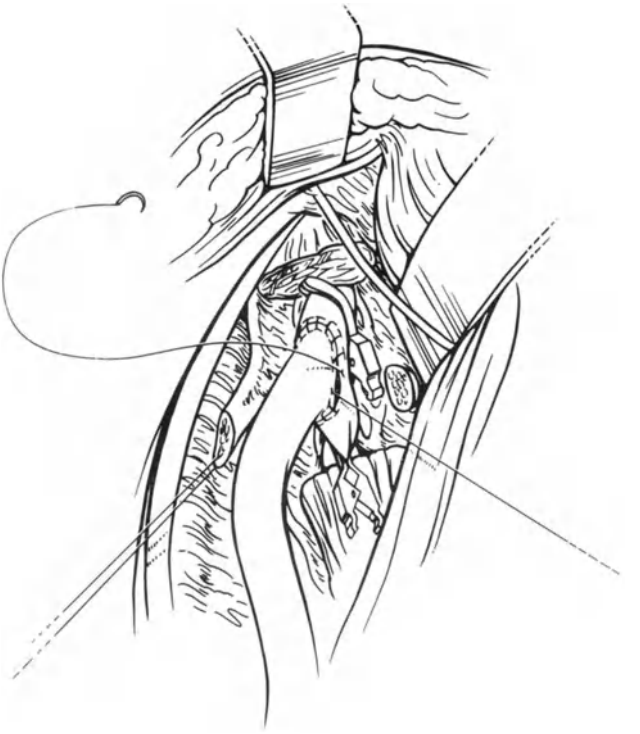


FIGURE 7.7. Schematic representation of the common carotid to distal vertebral bypass. Note the distal anastomosis has been converted functionally into an end-to-end junction by the application of a hemoclip below it.

passed under the jugular vein and anastomosed to the common carotid artery. For this the common carotid artery is cross-clamped clearly below the bifurcation so as not to involve any plaque that may be present there (Fig 7.7).

An alternative technique for reconstruction of the distal VA is to *transpose the external carotid artery to it* (Fig 7.8). This operation is usually done in younger individuals who have extrinsic compression of the VA in the intraspinal portion and carotid bifurcations free from atheromatous disease. The appeal of this technique is that it does not involve clamping of the internal carotid artery. The external carotid artery is dissected to a

FIGURE 7.6. Distal anastomosis of the common carotid to distal vertebral bypass.

FIGURE 7.8. Transposition of the external carotid to the distal vertebral artery.

point beyond the digastric muscle, ligating all of its branches and dividing the end of the dissected segment. The freed external carotid artery is rotated in front of the internal carotid artery, passed under the jugular vein and anastomosed to the distal VA either end-to-side, as described for the vein graft technique or, if the artery is a bit short, end-to-end. The VA in the latter case is swung toward the external carotid artery for an easy reach.

The third technique for reconstruction of the distal VA is the *transposition of the distal VA to the cervical internal carotid artery* (Fig. 7.9) by an end-to-side anastomosis, which will make the distal VA a branch of the distal cervical internal carotid artery. For this the internal carotid artery and the distal VA are freed for an adequate length to permit swinging the latter into the former. The anastomosis is end-to-side. If the arteries are

FIGURE 7.9. Transposition of the distal vertebral artery to the distal cervical internal carotid artery.

small the front half of the anastomosis may be done with interrupted sutures of 7.0 or 8.0 polypropylene. This technique should not be used in a patient with a contralateral internal carotid occlusion because the risk for cerebral ischemia would be prohibitive.

8
Combined Carotid and Coronary Artery Surgery

D.P. HALPIN, S. RIGGINS, J.D. CARMICHAEL, J.H. ISOBE, J.L. MATHEWS, T.R. KAHN, and T.A. GASKIN

Summary

Simultaneous coronary artery bypass grafting (CABG) and carotid endarterectomy (CE) were performed on 133 patients over an eight-year period. Twenty-seven patients (20%) had previous TIAs, 12 (9%) had previous strokes, the remainder were asymptomatic (71%). All asymptomatic patients had greater than 85% stenosis of the internal carotid artery demonstrated by noninvasive ultrasound and four-vessel angiography. CE was performed prior to the sternotomy for coronary artery bypass under the same anesthesia. Nineteen patients had bilateral carotid artery disease.

Postoperatively three patients suffered strokes (2.2%), an additional three patients (2.2%) suffered transient upper extremity weakness; one patient from each of these groups died. There were no post-op strokes or TIAs in patients with bilateral carotid artery disease. Average length cf hospital stay was 10 days. Our experiences lead us to conclude that the morbidity/mortality of the simultaneous procedure is not affected by bilateral carotid artery disease. We believe that in patients with symptomatic coronary artery disease and symptomatic carotid artery disease, or asymptomatic carotid artery disease with a high grade stenosis, simultaneous repair of both lesions should be undertaken.

Introduction

Surgical management of combined carotid and coronary artery disease is controversial. The incidence of carotid artery disease in patients undergoing coronary artery surgery is reported to range from to 6–16%,[1] while the incidence of coronary artery disease in patients undergoing carotid artery surgery in some series is as high as 25–54%.[2] Patient mortality rate for carotid artery surgery for those who have significant coronary artery disease is reported by Ennix et al. to be as high as 18%,[3] coronary-related events accounting for 50–75% of late deaths in this group.[4]

The incidence of stroke is reported to be as high as 10% in patients with significant carotid artery disease who undergo coronary artery bypass surgery. The initial period of hypotension associated with the institution of extracorporeal circulation and the decrease mean and pulsatile flow and pressure may be associated with sudden cerebral hypoperfusion in some patients. In 1972 Bernhard demonstrated a significant reduction in cardiac morbidity in a group of patients undergoing simultaneous carotid endarterectomy and myocardial revascularization compared with a similar group undergoing staged carotid repair followed by coronary artery bypass graft at a later date.[5] This combined approach was subsequently advocated by others.[6,7,8] We present here our experience with the simultaneous approach to this complex lesion.

Materials/Methods

From 1982–1990, 133 patients had coronary artery bypass surgery and carotid surgery performed simultaneously, Data was obtained from patient chart review. A total of 84 men and 49 women were studied with an average age of 66 (range 45–88). A history of cigarette smoking and hypertension was present in 57% and 60%, respectively, 28% were diabetic, and 19% had type II hyperlipidemia. All patients had severe coronary artery disease—30% presenting with unstable angina, 45% with stable angina. Previous myocardial infarction, demonstrated by EKG, was present in 9.7%. Coronary angiography showed that 24% had left main stem disease. One hundred thirty patients (76%) had triple vessel disease. Patients considered to be at risk for a stroke, either by the history of neurologic events or by the presence of a carotid bruit, underwent ultrasonic duplex scanning of the carotid arteries. All patients with a high grade (>85% carotid stenosis) underwent arteriography and carotid endarterectomy, followed by coronary artery bypass during the same anesthesia. Neurologic exams were performed postoperatively in all patients. Cardiopulmonary perfusion techniques were standardized with a bubble oxygenator and moderate hemodilution and flow reduction (1.6 $1/m^2/min$) with cooling to 30°C. Perfusion pressures were not modified unless they were consistently less than 40 mm Hg.

Results

There were 15 postoperative complications in 133 patients (Table 8.1) and three patients (2.2%) suffered permanent neurological deficit, two of whom were able to live independently and one of whom died in the hospital. Three more patients (2.2%) suffered transient upper extremity weakness, which resolved completely. There were six (4.5%) post-op

TABLE 8.1. Postoperative complications.

	Number	%
TIA	3	2.2
CVA	3	2.2
Myocardial infarction	6	4.5
Ventricular dysfunction requiring IABP	3	2.2

Operative morbidity in 133 patients

myocardial infarcts, demonstrated by EKG and enzyme changes. Average hospital stay was 10 days (range 5–30). Nineteen patients were identified with bilateral significant carotid stenosis, nine of whom had one occluded internal carotid artery. None of these patients suffered neurologic morbidity or mortality postoperatively.

Discussion

Various authors have reported their experience in dealing with coexisting carotid and coronary disease. Initial reports of staged and simultaneous procedures indicated a favorable outcome with the simultaneous approach. Bernhard et al.[5] in 1972 reported on 31 patients, 15 having staged procedures, the remainder simultaneous procedures. Three patients (20%) died of cardiac complications prior to myocardial revascularization in the first group, while only one patient (6%) in the second group had a permanent neurological deficit. Morris and associates, in a retrospective study of 79 patients, compared both approaches and found the simultaneous procedure gave the best long-term results.[9]

In contrast Craver et al. reported a series of 68 patients with no deaths and a 7.5% incidence of stroke in patients undergoing simultaneous procedures.[10] Hertzer and colleagues at the Cleveland Clinic[11] echoed this high stroke rate in reporting 115 simultaneous operations. They noted an 8.7% incidence of stroke and a mortality of 4.3%. These results were compared to stroke rate of 3.1% and a mortality of 1.7% in 59 patients who underwent staged repair. It is of note that most of the morbidity occurred in patients with contralateral carotid artery disease. If the contralateral carotid was normal, neurological deficit occurred in only 1.5%. With a contralateral carotid stenosis of greater than 50%, the incidence of permanent neurological deficit was 6.7%.

Similarly, Carey and Cuningnan[12] encountered two postoperative neurological deficits in four patients undergoing simultaneous procedures, which they attributed to disease in the nonoperated carotid artery.

In contrast our series showed 19 patients identified with greater than 70% stenosis of the contralateral carotid artery, nine of whom had contra-

lateral occlusion. There was no neurological morbidity or mortality in this group.

From our experience and the data reported, several conclusions can be reached. Carotid and coronary artery disease frequently coexist. Patients seen for coronary artery surgery require clinical assessment for carotid symptoms or a bruit. If present, neurological review, noninvasive studies and, if necessary, contrast studies are indicated.

The presence of bilateral significant carotid artery stenosis in patients with coronary artery disease does not influence morbidity or mortality adversely.

The approach for patients with coexistent carotid artery and coronary artery disease is not an obvious one. It should probably be tailored to fit individual patients. In our hands simultaneous repair of carotid and coronary arteries has substantial merit, the carotid artery being repaired prior to CABG. The low neurological morbidity and mortality in our series and others support the use of the combined approach.

References

1. Perler BA, Burdick JF, Williams GM. The Safety of CEA at the Time of CABG: Analysis of Results in a High Risk Patient Population. J Vasc Surg 1985;2:558–63.
2. Berkoff HA, Turnipseed WD. Patient Selection and Results of Simultaneous Coronary, Carotid Artery Procedures. Ann Thorac Surg 1984;38:172–5.
3. Ennix CL, Lawrie GM, Morris GC, et al. Improved Results of Carotid Endarterectomy in Patients with Symptomatic Carotid Artery Disease: An Analysis of 1546 Consecutive Carotid Operations. Stroke 1979;10:122–5.
4. O'Donnell TF, Jr, Collow AD, Willet C, et al. The Impact of Coronary Artery Disease on CEA. Ann Surg 1983;198;705.
5. Bernhard VM, Johnson WD, Peterson JJ. Carotid Artery Stenosis Association with Surgery for Coronary Artery Disease. Arch Surg 1972;105:837–40.
6. Urschel HC, Razzuk MA, Gardner MA. Management of Concomitant Occlusive Disease of the Carotid and Coronary Arteries. J Thorac Cardiovasc Surg 1976;72:829.
7. Okies JE, MacManus Q, Starr A. Myocardial Revascularisation and Carotid Endarterectomy. A Combined Approach. Ann Thorac Surg 1977;23:560.
8. Check WA. Simultaneous Coronary Bypass and Carotid Endarterectomy Advocated. JAMA 1978;250:725.
9. Morris GC, Ennix CL, Lawrie GM, et al. Management of Coexistent Carotid and Coronary Artery Occlusive Atherosclerosis. Clev Clin Q 1978;45:125.
10. Craver JM, Murphy DA, Jones E, et al. Con? Carotid and Coronary Artery Revascularization. Ann Surg 1982;195:712–20.
11. Hertzer NR, Loop FD, Taylor PC, Beren EG. Staged and Combined Surgical Approach to Simultaneous Carotid and Coronary Vascular Disease. Surg 1978;84:803–11.
12. Carey JS, Cuningnan RA. Complications of Combined Brachiocephalic and Coronary Revascularization. Ann Thorac Surg 1978;25:385.

9
Anomalous Branch of Cervical Internal Carotid Artery: Embryological and Technical Considerations

CALVIN A. RIDGEWAY, ROBERT D. WILLIAMS, and L. BEATY PEMBERTON

Arterial branches from the cervical portion of the internal carotid artery are unusual. However, surgeons who perform carotid endarterectomies encounter this situation occasionally. These branches may give rise to technical problems. This paper will report a case and discuss embryological and technical aspects of cervical branches of the internal carotid artery.

Case Report

A 70-year-old white woman had a past medical history of a myocardial infarction approximately one year prior to admission and mild hypertension. Her recent complaint of dizziness caused her physician to order a duplex scan, which demonstrated high grade stenotic lesions of the internal carotid arteries bilaterally. A digital subtraction angiogram was subsequently performed (Fig. 9.1), which revealed a high grade stenosis of both internal carotids as well as a possible ulcerated lesion on the left side. Careful examination of this angiogram revealed another vessel, running parallel to the internal carotid artery, which appeared to be an artifact. At operation, with the left common, internal, and external carotid arteries clamped and the shunt in place, there was troublesome back-bleeding. Further dissection revealed that the back-bleeding was caused by an ascending pharyngeal artery arising from the posterior surface of the internal carotid artery approximately one centimeter above the bifurcation. One week later, a right carotid endarterectomy was performed, and a duplicate of this artery was identified.

Discussion

The six aortic arches arise from the aortic sac in the fourth week of gestation.[1] (Fig. 9.2). They are not present at the same time. The first and

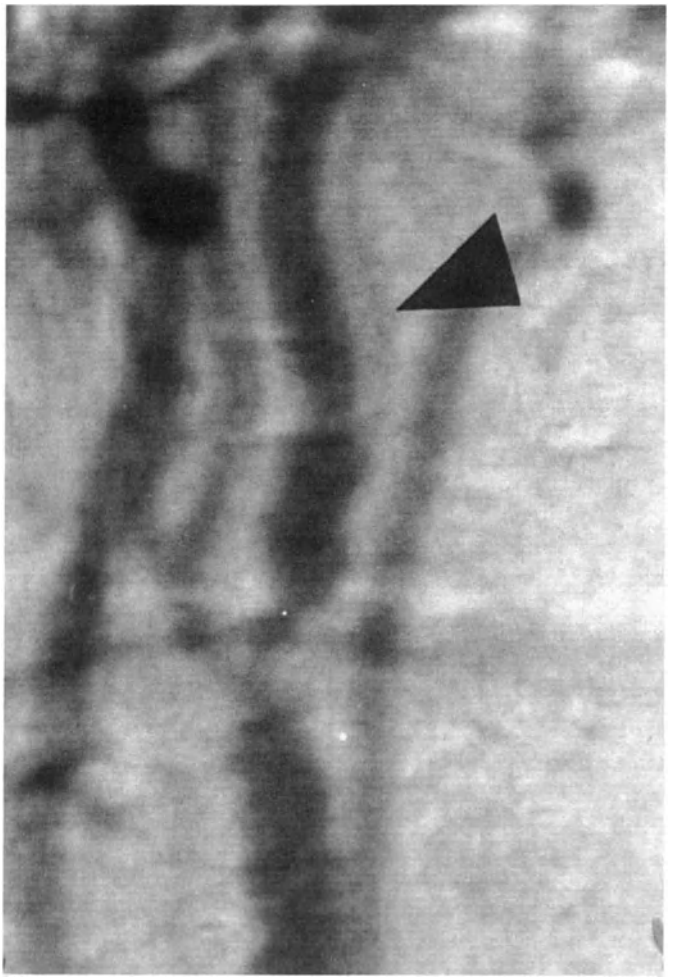


FIGURE 9.1. This angiogram demonstrates a high grade stenosis of the internal carotid artery. The arrow points to the anomalous branch of the cervical internal carotid artery.

second arches are gone by the time the sixth arch appears. By the eighth week, the aortic and brachiocephalic vessels have their adult appearance. The first arch yields the maxillary arteries and may provide portions of the external carotid. The common carotid and portions of the internal carotid arise from the third arch.[10] The dorsal aorta also supplies a portion of the

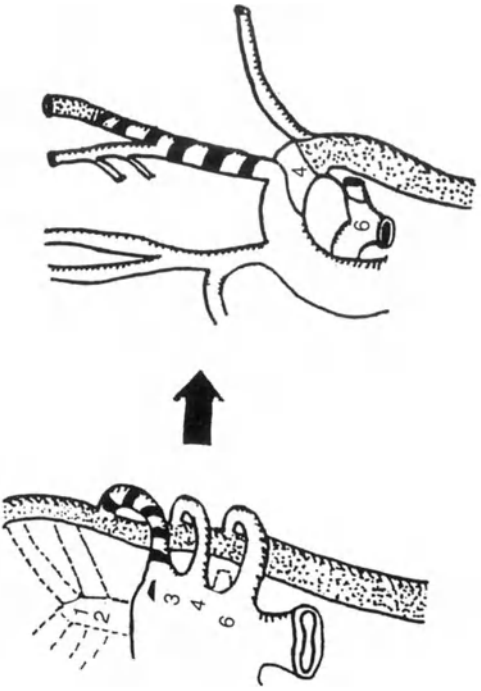


FIGURE 9.2. Transition of the primitive aortic arches to the adult state. The stippled area represents the dorsal aorta; the striped area is the third aortic arch. These combine to form the adult internal carotid artery.

internal carotid. On the left side it continues as the descending aorta. The external carotid artery is a branch from the third arch,[1] which connects to portions of the vascular buds in the subpharyngeal region in order to provide the adult external carotid artery with its branches.[2,3]

Anomalies arise when the vascular buds connect first and second arch remnants directly to the internal carotid instead of using the external carotid as an intermediary.[2,3] The most frequent anomalous branch is the ascending pharyngeal.[4] Several reports have described occipital arteries from the internal carotid.[2] In 1965 Seidel described complete obliteration of the distal external carotid with all of its branches arising from the internal carotid.[5] Surgeons performing carotid endarterectomies will encounter one of these unusual branches of the internal carotid arteries.

These internal carotid branches are important to consider in the diagnosis of carotid occlusive disease as well as in its surgical treatment. Littooy described two cases of anomalous branches from the internal carotid that provided blood flow into the distal internal carotid even though the plaque had completely occluded the artery at its take-off from the common carotid.[6] Both cases required additional dye studies. One required a ver-

tebral injection with double subtraction in order to elucidate further the anatomy of these arteries. He went on to say that patients with an occluded proximal internal carotid artery with reconstitution must have a branch to maintain patency. Usually, a completely occluded internal carotid artery is a contraindication for a carotid endarterectomy. However, if the internal carotid receives blood flow from an anomalous branch, then the internal carotid is not completely occluded. These patients are still physiologically candidates for a carotid endarterectomy.

During the performance of the endarterectomy, back-bleeding from these arteries may be troublesome.[7,8] Brown described a case where the internal carotid bifurcated into two equal vessels with the anterior vessel providing normal circulation and the posterior vessel providing vertebral basilar circulation. Performance of this operation required the use of two Javid shunts, but he still had trouble with back-bleeding. Branches may not arise from the contralateral side.[2] In the case presented they were symmetrical. The branch did need to be occluded to prevent back-bleeding into the operative field. Back-bleeding during a carotid endarterectomy is the most common complication from all of these anomalous branches.

In addition, the surgeon must keep in mind that the vagus nerve travels in the carotid sheath directly posterior to the normal internal carotid artery. Meticulous dissection techniques must be used in order to prevent injury to the vagus nerve with its subsequent complications.

References

1. Moore KL. *The Developing Human*, 3rd Ed., WB Saunders, 1982.
2. Benson MT, Hamer JD. "Anomalous Origin of the Occipital Artery from the Cervical Internal Carotid Artery." J Vasc Surg, Vol 8, No. 5:643–645, 1988.
3. Altman F. Anomalies of the Internal Carotid Artery and Its Branches: Their Embryological and Comparative Anatomical Significance. Laryngoscope, 57:313–39, 1947.
4. Dilenge D, Heon M. "The Internal Carotid Artery." In Newton TH, Potts DG, eds., *Radiology of the Skull and Brain*, Vol 2, Book 2, St. Louis, CV Mosby, 1974;1202–45.
5. Seidel K. Arteriogrophische Beobachtung einer seltenen carotid anomalie. Fortschur Roengenstr 103:390–391, 1965.
6. Littooy FN, Baker WH, Field TC, et al. "Anomalous Branches of Cervical Internal Carotid Artery. Two Cases of Clinical Importance." J Vasc Surg, Vol 8, No 5:634–637, 1988.
7. Brown L, Partridge H, Eckstein M. "Bifurcation of the Cervical Internal Carotid Artery." Arch Surg, Vol 121, No 3:367, 1986.
8. McShane MD, Chant ADBM. "Failure of Duplex-Scanning to Demonstrate an Anomalous Branch of the Internal Carotid Artery" Eur J Vasc Surg, Vol 1, No 3:213–214, 1987.

10
Current Concepts for the Pathogenesis of Abdominal Aortic Aneurysms

JON R. COHEN

Abstract

The classic description of abdominal aortic aneurysms as random variants of atherosclerosis occurring at a weakened site in the aortic wall is now being challenged by an increasing amount of data from recent clinical, genetic, and biochemical studies. The data from these studies suggest that abdominal aortic aneurysms develop in a logical, stepwise progression as related to a complex set of genetic, environmental, and biochemical factors that interact to alter aortic connective tissue metabolism.

Introduction

Abdominal aortic aneurysms (AAAs) occur in approximately 2% of the elderly population.[1] In 1988, 39,000 abdominal aortic aneurysm repairs were performed in this country for a total health cost of approximately $933,000,000, averaging $23,931 per patient.[2,3] Abdominal aortic aneurysms are always found in association with significant aortic atherosclerosis and, because of this association, have been classically described as random variants of atherosclerosis that occur at a weak spot in the arterial wall. The aneurysm is thought to grow according to La Place's Law until it eventually ruptures. In contrast to this belief, clinical observations over the last 30 years and recent genetic and biochemical studies now suggest that aneurysmal pathogenesis is more likely related to an alteration in systemic connective tissue metabolism.

Clinical Observations

If one considers aneurysmal degeneration as a common manifestation of atherosclerosis, then distal aneurysms and aortic aneurysms would be commonplace in the patient with diffuse arterial disease. This, however, is not

the case. It is unusual, if not rare, for a patient with superficial femoral artery disease, distal arterial disease, or aortoiliac obstructive disease to have distal arterial aneurysms. However, abdominal aortic aneurysms commonly occur in association with peripheral aneurysms, and patients with peripheral aneurysms frequently have associated aneurysms at other locations. In a study of 1,488 patients with abdominal aortic aneurysms, 4% of the patients had associated peripheral aneurysms, and multiple aneurysms occurred in 83% of patients who had at least one peripheral aneurysm.[4] In patients with a common femoral artery aneurysm, 95% had a second aneurysm, 92% had an aortoiliac aneurysm, and 59% had bilateral femoral aneurysms. Among patients with popliteal artery aneurysms, 78% had a second aneurysm, 64% had an aortic aneurysm, and 47% had a popliteal artery aneurysm on the contralateral side.[4] In one study of 100 consecutive femoral aneurysms, all patients were men, and 85% of these patients had an AAA.[5]

Inguinal herniation is significantly higher in patients with abdominal aortic aneurysms (26%) compared to patients with aortic occlusive disease (15%). In addition, patients with abdominal aortic aneurysms have more severe herniation in terms of direct hernias, bilateral hernias, recurrent hernias, and early onset, suggesting that a systemic connective tissue disorder causes both the weakening of the abdominal wall and aorta resulting in both hernia and abdominal aortic aneurysm in the same patient.[6] Tortuous carotid arteries are associated with abdominal aortic aneurysms in up to 44% of cases, whereas only 10% of patients with routine carotid atherosclerosis have abdominal aortic aneurysms. Weakness in the arterial wall has been suggested as the cause of both the tortuous carotid artery and the abdominal aortic aneurysm in the same patient.[7]

As opposed to distal occlusive disease, many patients with aneurysms have associated generalized arteriomegaly and rarely develop claudication or rest pain. Vessels remote from abdominal aortic aneurysms are actually 40% to 50% larger in diameter compared to patients with aortic occlusive disease.[8] In one study, three distinct populations of aneurysm patients were defined depending on the degree of associated arteriomegaly. In that study, 430 aneurysms in 91 patients were classified based on the degree and location of the associated arteriomegaly and aneurysms.[9]

Genetic Reports

Several interesting genetic reports strongly suggest that abdominal aortic aneurysms are a familial disease. In one study of 14 families with aneurysms, there was at least one blood relative with an abdominal aortic aneurysm, and in two families, there were two blood relatives with an abdominal aortic aneurysm apart from the proband.[10] In a second study of 250 patients with abdominal aortic aneurysms, 19% of the patients with

aneurysms had a first degree relative with a known aneurysm compared to 2.4% of the control subjects. After adjustment for age and sex, there was an estimated 11.6% increase in abdominal aortic aneurysm risk among patients with an affected first degree relative.[11] Tilson reported on the genetic mechanisms of inheritance of 50 families having 71 affected individuals with abdominal aortic aneurysms. The mechanism of inheritance in this study was suggested to be both x-linked and autosomal dominant forms with x-linked variance as the most common type.[12] Cole found 34 families with abdominal aortic aneurysms in which 17 probands had at least one affected parent and of the siblings, 20% were affected. This study suggested that siblings of patients with abdominal aortic aneurysms have nearly a one in five chance themselves of having an abdominal aortic aneurysm if there is a positive parental history.[13] A fifth study reported 86 families having 209 first degree relatives with abdominal aortic aneurysms and found that the patients with familial aneurysms were more likely to be women and more likely to rupture.[14] In one amazing report, three brothers (the only siblings) of one family have each undergone emergency surgery for a ruptured abdominal aortic aneurysm.[15]

Most recently, a genetic variation on chromosome 16, responsible for haptoglobin phenotype, is associated with abdominal aortic aneurysms. The frequency of the haptoglobin alpha-1 allele was increased in patients with abdominal aortic aneurysms compared to controls. In addition, the frequency of a rare polymorphism at the cholesterol ester transfer protein locus was also increased in abdominal aortic aneurysm patients. Interestingly, haptoglobin containing an alpha-1 chain accelerates the degradation of elastin by elastase two to four times.[18]

To date, six studies have documented the number of first degree relatives with abdominal aortic aneurysms. In combining the results of these studies,[10,11,13,14,16,17] 16% of patients with abdominal aortic aneurysms will have a first degree relative with an abdominal aortic aneurysm (Table 10.1). The results of these reports indicate that the genetics of abdominal aortic aneurysm are very complex and multifactorial, with the involvement of several different genes. The phenotypic expression of these genes affect lipid metabolism, atherosclerosis and elastin metabolism. Currently, we know that specific genes on the long arm of chromosome 16 are associated with abdominal aortic aneurysms.[18] However, other genes are currently under investigation.

Biochemical Data

Elastin Metabolism

The first report suggesting an abnormal increase in proteolytic activity in patients with abdominal aortic aneurysms occurred in 1982 when elastase

TABLE 10.1. Genetic studies of AAA families.

	Number of AAA patients	First degree relatives with AAA	%
Johnansen (11)	250	48	19%
Norrgard (10)	87	16	18%
Cole (13)	305	34	11%
Collin (39)	108	13	12%
Darling (14)	542	82	15%
Powell (16)	60	20	33%
Totals	1352	213	16%
Clifton (15)	3 brothers of same family—all ruptured AAAs		
Tilson (12)	71 relatives with AAAs in 50 families		

was noted to be increased in the aortic wall in patients with abdominal aortic aneurysms.[19] In the same year, circulating serum and leukocyte elastolytic activity in patients with abdominal aortic aneurysms was noted to be significantly increased, whereas serum antiprotease activity was reduced.[20] This was the first report to suggest that an abnormal homeostasis existed between proteolytic and antiproteolytic activity in patients with abdominal aortic aneurysms.

Four years later the relationship between elastase, the major proteolytic enzyme of elastin, and alpha-1-antitrypsin (a-1-AT) inhibitor, the major elastase, was analyzed in the aortic wall in the four groups of patients with the major different types of aortic disease. Aortic elastase was significantly higher in the AAA, multiple aneurysm, and ruptured AAA groups compared to aortic occlusive disease patients, and a-1-AT was significantly lower in patients with multiple aneurysms and ruptured aneurysms. The relationship between these two enzyme systems responsible for aortic elastin breakdown changed in favor of more aortic proteolytic activity as the type of infrarenal aortic disease changed from occlusive to aneurysmal disease.[21] In another study, elastin urine metabolites, specifically the elastin breakdown product L-valyl-proline, was increased in patients with aneurysmal disease compared to patients with aortic occlusive disease.[22]

The elastin content in the AAA wall is also significantly less than in control and aortic occlusive patients, and the increased elastase content of the AAA wall is inversely correlated with the decrease in elastin content.[23] Interestingly, at least two types of elastase have been identified within the aortic wall of abdominal aortic aneurysm patients, including both neutrophil and smooth muscle cell elastase.[24] Recent data indicates that neutrophils are chemotactic to elastin fragments and are very attracted to the elastin peptides of aortic aneurysm tissue. This observation provides a mechanism whereby neutrophil elastase is delivered to the aorta of aneurysm patients.[25] Smooth muscle cells proliferate and secrete elastase in response to atherosclerosis. The increased proteolysis from increased

neutrophil and smooth muscle cell elastase in the aortic wall results in histologically disrupted elastic tissue compared to patients with occlusive disease and controls. Histochemical studies of aneurysmal aortas confirm a significant deficiency of iron-hematoxylin reactive elastin compared to occlusive aortic disease.[26]

Alpha-1-antitrypsin is the major serum inhibitor of elastase, and its phenotype correlates directly with circulating a-1-AT levels. A report of the a-1-AT phenotype in relation to proteolytic activity in AAA patients indicates that the MZ phenotype, which is associated with 35% of the normal inhibitory capacity of a-1-AT, occurs significantly more often in patients with AAAs (11%) than would be expected. The decrease in a-1-AT (elastase inhibitor) that occurs with the MZ phenotype results in increased serum elastase activity and would explain the increased proteolytic activity in some AAA patients, however, it could not be implicated as the cause of protease imbalance in the majority of patients.[27] Cigarette smoke, as an environmental influence, has a major effect on proteolytic activity by inhibiting a-1-AT, thereby increasing serum elastase activity. In a study of the effect of cigarette smoke on rabbit aortic elastase activity, rabbit aortic elastase was significantly higher in cigarette exposed animals compared to controls.[28] In contrast to a-1-AT, haptoglobin with an alpha-1 allele potentiates the effect of elastase on elastin breakdown. Elastase in the presence of haptoglobin with an alpha-1 chain accelerates the breakdown of elastin two-to fourfold.[18]

Collagen Metabolism

Abnormal collagen metabolism has been implicated as a possible cause of AAA development by several authors. In one report an increase in collagen in aneurysmal aorta from 62% to 84% occurred as a result of elastin degradation, and no increase in collagenase activity was noted. Lower amounts of type III collagen were found in patients with familial AAAs, and abnormalities in type III collagen was suggested as a genetic factor contributing to familial clustering of AAAs.[29] Tilson further reported on abnormalities of aortic collagenous peptides based on cleavage with cyanogen bromide in patients with AAAs.[30] In an animal model of post stenotic dilatation, a twofold increase in collagenase activity occurred in the area of dilatation compared to controls with no change in elastase activity. This study suggested that collagenolysis may be induced by local flow and pressure changes and that increased collagenase activity associated with AAAs may be a response to altered hemodynamics and not an intrinsic defect.[31] Deak[32] examined the biosynthesis of type III collagen in AAA patients from cultured skin fibroblasts and reported that several patients with familial AAAs had one normal allele and one allele that coded for a structural mutation in type III collagen which formed an imperfect triple

helical collagen molecule. They suggested that mutations of the type III collagen gene may play a role in AAA development. However, in a more recent study of collagen types in AAA patients and familial AAAs, no collagen type III deficiency was noted in either group. This study suggested that AAAs result from elastin degradation and not a deficiency of type III collagen.[33]

In an animal model, the mechanical properties of dog arteries have been studied before and after exposure to elastase and collagenase. Interestingly, all arteries exposed to elastase and mechanical forces developed aneurysms.[34] In another study, the longitudinal force was measured in dog arteries after treatment with elastase and collagenase. Results showed that elastin provided all of the longitudinal retractive force.[35] The data from these studies indicate that failure of elastin causes blood vessels to elongate and become tortuous, and that elastin breakdown, not collagen, plays the major role in the arteriomegaly and aneurysms seen with age and hypertension in humans.

Ruptured AAAs

AAA rupture may also be related to increased proteolysis and elastin breakdown. One report cited 10 patients who ruptured their AAAs within 36 days after laparotomy and suggested that laparotomy may be a precipitating factor in the rupture of AAAs. This study suggested that laparotomy upsets the dynamic equilibrium between synthesis and lysis of systemic connective tissue.[36] In an animal model operative trauma, such as laparotomy, bowel resection, and mobilization of the aorta without direct aortic injury, resulted in significant increases in rabbit aortic elastase.[37] Furthermore, in human aortic tissue of patients with ruptured AAAs, the balance between elastase and antiprotease is significantly altered in favor of more proteolytic activity in patients with ruptured AAAs.[38]

In a regression analysis of aortic aneurysm rupture, three significant variables were associated with small aneurysm rupture: diastolic blood pressure, initial aneurysm size, and degree of obstructive pulmonary disease. Since the mechanism for severe emphysema in COPD is probably an imbalance between elastase and a-1-AT, it is interesting to speculate on whether the association between COPD and AAA rupture in these patients occurs via the same mechanism of increased systemic proteolysis.[39]

As mentioned above, a positive female marker, that is, identification of a family with AAAs and a female member with an AAA, is strongly correlated with rupture. The term "black widow syndrome" has been suggested to describe this potentially fatal trait of female members of this familial group of patients with AAAs.[14]

The above studies provide enough information to develop a comprehensive unified hypothesis for the pathogenesis of AAA formation (Fig. 10.1). I propose that aneurysms are not merely random variants of atheroscle-

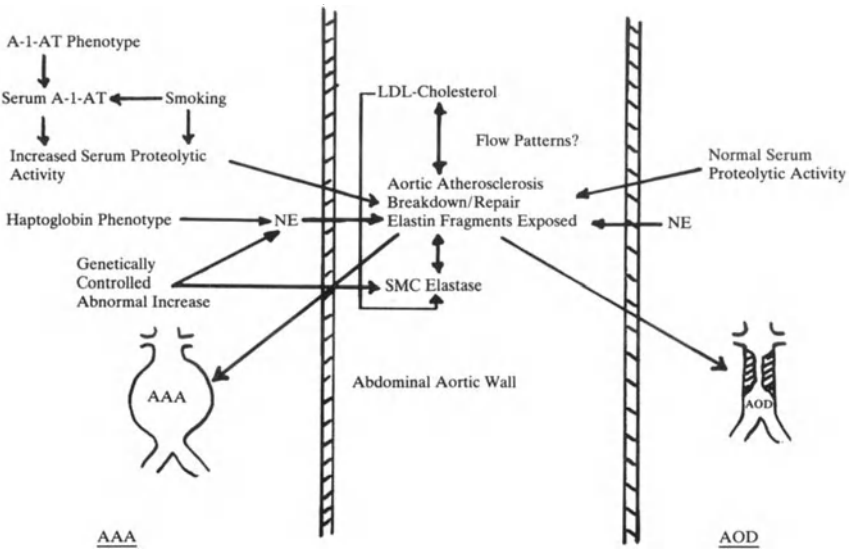


FIGURE 10.1 Unified hypothesis for abdominal aortic aneurysm development based upon current genetic studies, biochemical data, and clinical observations (see text). A-1-AT-Alpha-1-Antitrypsin; NE-Neutrophil Elastase; SMC-Smooth Muscle Cell; AAA-Abdominal Aortic Aneurysm; AOD-Aortic Occlusive Disease; LDL-Low Density Lipoprotein.

rosis, but rather that they develop in a logical stepwise progression in the following manner: (1) aneurysm patients have a genetically determined increase in cellular elastase, specifically an increase in neutrophil and smooth muscle cell elastase; (2) serum proteolytic activity as determined by the genes for elastase production is modified by other genetic factors such as the a-1-AT phenotype, the haptoglobin phenotype, and environmental factors such as cigarette smoke; (3) in the usual course of aortic atherosclerosis, elastin fragments and elastin peptides are exposed to the circulation. During the reparative process, neutrophils are chemotactic to these aortic elastin fragments and the atherosclerotic fragments also act as stimulators of local smooth muscle cell proteolytic production; (4) in aneurysm patients, an increased amount of proteolytic enzymes are delivered to the aortic wall by elastase-laden neutrophils, local smooth muscle cells and increased serum proteolytic activity; (5) during the atherosclerotic process of arterial wall breakdown and repair, the increased delivery of proteolytic enzymes in aneurysm patients results in a chronic increase in aortic elastin breakdown, weakening of the wall, and aneurysm dilatation; (6) the large number of possible combinations of genetic and environmental factors that could occur from patient to patient would then be responsi-

ble for the large diversity of different phenotypes seen clinically, i.e., patients with isolated AAAs, multiple aneurysms or massive arteriomegaly and aneurysms.

This hypothesis suggests that AAAs are indeed a variant of atherosclerosis that could occur only if atherosclerosis is present and would account for the fact that AAAs always coexist with aortic atherosclerosis.

References

1. Melto LJ, III, Bickerstaff LK, Hollier LH, and O'Fallon WM: Changing Incidence of Abdominal Aortic Aneurysms: A Population-Based Study. Am J Epidemiol. 120:379–386; 1984.
2. National Center for Health Statistics. Detailed Diagnoses and Surgical Procedures for Patients Discharged from Short-Stay Hospitals. United States, 1986, Washington, DC: DHHS Publication no. (PHS) 88-1756.
3. Munoz E, Kassan MA, and Chang J. Surgonomics: The Costs of Ruptured Abdominal Aortic Aneurysm. Angiology. 39(9):830–837; 1988.
4. Dent TL, Lindenauer SM, Ernst CB, and Fry WJ. Multiple Arteriosclerotic Arterial Aneurysms. Arch Surg. 105:338–344; 1972.
5. Graham LM, Zelenock GB, Whitehouse WM, et al. Clinical Significance of Atherosclerotic Femoral Artery Aneurysms. Arch Surg. 115:502–507; 1980.
6. Cannon DJ, Casteel L, and Read RC. Abdominal Aortic Aneurysm, Leriche's Syndrome, Inguinal Herniation and Smoking. Arch Surg. 119:387–390; 1984.
7. Mukherjee D, Mayberry JC, Inahara T, and Greig JD. The Relationship of the Abdominal Aortic Aneurysm to the Tortuous Internal Carotid Artery. Arch Surg. 124:955–956; 1989.
8. Tilson MD and Dang C. Generalized Arteriomegaly. Arch Surg. 116:1030–1032; 1981.
9. Hollier LH, Stanson AW, Gloviczki P, et al. Arteriomegaly: Classification and Morbid Implications of Diffuse Aneurysmal Disease. Surg. 93:700–708; 1983.
10. Norrgard O, Rais O, and Angquist KA. Familial Occurrence of Abdominal Aortic Aneurysms. Surg. 95:650–656; 1984.
11. Johansen K and Koepsell T. Familial Tendency for Abdominal Aortic Aneurysms. JAMA. 256:1934–1936.
12. Tilson MD and Seashore MR. Fifty Families with Abdominal Aortic Aneurysms in Two or More First-Order Relatives. Am J Surg. 147:551–553; 1984.
13. Cole CW, Barber GG, Bouchard A, et al. Abdominal Aortic Aneurysms. Consequences of a Positive Family History. The Can J Surg. 31:286–289; 1988.
14. Darling RC III, Brewster DC, Darling RC, et al. Are Familial Abdominal Aortic Aneurysms Different? J Vasc Surg 10:39–43; 1989.
15. Clifton MA. Familial Abdominal Aortic Aneurysms. Br J Surg. 64:765–766; 1977.
16. Powell JT and Greenhalgh RM Multifactorial Inheritance of Abdominal Aortic Aneurysm. Eur J Vasc Surg. 1:29–31; 1987.
17. Collin J and Walton J. Is Abdominal Aortic Aneurysm Familial? Br Med J. 299:493; 1989.

18. Powell JT, Bashir A, Dawson S, et al. Genetic Variation on Chromosome 16 Is Associated with Abdominal Aortic Aneurysm. Clin Sci. 78:13–16; 1990.
19. Busutill RW, Rinderbreicht H, Flesher A, and Carmack C. Elastase Activity: The Role of Elastase in Aortic Aneurysm Formation. Research. 32:214–217; 1982.
20. Cannon DJ and Read RC. Blood Elastolytic Activity in Patients with Aortic Aneurysm. Ann Thor Surg. 34:10–15; 1982.
21. Cohen JR, Mandell C, Chang J, and Wise L. Elastin Metabolism of the Infrarenal Aorta. J Vasc Surg. 7:210–214; 1988.
22. Cohen JR, Dietzek A, Stein T, and Wise L. Urinary L-Valyl-Proline in Patients with Aortic Aneurysms. Surg Gyn Obs. (in press)
23. Campa JS, Greenhalgh RM, and Powell JT. Elastin Degradation in Abdominal Aortic Aneurysms. Atheros. 65:13–21; 1987.
24. Cohen JR, Mandell C, and Wise L. Characterization of Human Aortic Elastase Found in Patients with Abdominal Aortic Aneurysms. Surg Gyn Obst. 165:301–304, 1987.
25. Cohen JR, Keegan L, Sarfati I, Dana D, Ilardi C, and Wise L. The Chemotaxis Distribution and Localization of Neutrophils and Neutrophil Elastase in the Aortic Wall of Patients with Abdominal Aortic Aneurysms. J Vasc Surg. (in review)
26. Tilson MD. Histochemistry of Aortic Elastin in Patients with Nonspecific Abdominal Aortic Aneurysmal Disease. Arch Surg. 123:503–505; 1988.
27. Cohen JR, Sarfati I, Ratner L, and Tilson D. Alpha-1-Antitrypsin Phenotypes in Patients with Abdominal Aortic Aneurysms. Accepted, J Surg Research. 12, 1989.
28. Cohen JR, Sarfati I, and Wise L. The Effect of Cigarette Smoking on Rabbit Aortic Elastase Activity. J Vasc Surg. 9:580–582; 1989.
29. Powell J and Greenhalgh RM. Cellular, Enzymatic, and Genetic Factors in the Pathogenesis of Abdominal Aortic Aneurysms. J Vasc Surg. 9:297–304; 1989.
30. Tilson MD and Roberts MP. Molecular Diversity in the Abdominal Aortic Aneurysm Phenotype. Arch Surg. 123:1203–1206; 1988.
31. Zarins CK, Runyon-Hass A, Zatina MA, Lu CT, and Glagov S. Increased Collagenase Activity in Early Aneurysmal Dilatation. J Vasc Surg. 3:328–348; 1986.
32. Deak SB, Ricotta JJ, Deak ST, Zatina MA, and Boyd CD. Abnormal Type III Collagen Synthesis in Patients with Abdominal Aortic Aneurysms. Clinical Research. 37:33; 1989.
33. Rizzo RJ, McCarthy WJ, Dixit SN, et al. Collagen Types and Matrix Protein Content in Human Abdominal Aortic Aneurysms. J Vasc Surg. 10(4):365–373; 1989.
34. Dobrin PB, Schwartz TH, Baker WH. Mechanisms of Arterial and Aneurysmal Tortuosity. Surg. 104:569–571; 1988.
35. Dobrin PB, Schwartz TH, and Mrkvicka RB. Longitudinal Retractive Force in Pressurized Dog and Human Arteries. J of Surg Research. 48:116–120; 1990.
36. Swanson RJ, Littooy FN, Hunt KT, Stoney RJ. Laparotomy as a Precipitating Factor in the Rupture of Intra-Abdominal Aneurysms. Arch Surg. 115:299–303; 1980.
37. Cohen JR, Schroder W, Mandell C, Wise L. Activation of Rabbit Elastase by Non-Aortic Intra-Abdominal Surgery. Ann Vasc Surg. 2:133–137; 1988.

38. Cohen JR, Mandell C, Margolis I, Chang JC. Altered Aortic Protease and Antiprotease Activity in Patients with Ruptured Abdominal Aortic Aneurysms. Surg Gyn Obst. 164:355–358; 1987.
39. Cronenwett JL, Murphy TF, Zelenock GB, et al. Actuarial Analysis of Variables Associated with Ruptures of Small Abdominal Aortic Aneurysms. Surg. 98:472–482; 1985.

11
Retroperitoneal Aortic Reconstruction: Indications and Pitfalls

DAVID F.J. TOLLEFSON and CALVIN B. ERNST

The first reported use of the retroperitoneal approach for abdominal vessel reconstruction was ligation of an external iliac artery aneurysm by Sir Astley Cooper in 1808.[1] The first successful repair of an abdominal aortic aneurysm was performed via the retroperitoneal approach and was reported by Dubost in 1951.[2] Since then, numerous authors have reported on the advantages of the retroperitoneal approach.[3,4,5] However, retroperitoneal aortic reconstruction still remains a novel and little used approach by most surgeons.

Even though the first aortic reconstructions were through the retroperitoneum,[6] the transperitoneal approach has been the preferred route for aortic reconstruction. This is probably due to the familiarity most surgeons have for the transabdominal route and the presumed concern that the retroperitoneal approach limits operative options.

Rob, in 1962, reporting on 500 aortic reconstructions performed through the retroperitoneal approach, listed the benefits to be less ileus, atelectasis and pain, reduced incidence of wound dehiscence, easier anesthesia, a shorter stay in bed and in the hospital, and faster return to work.[7] Other authors have suggested that the retroperitoneal approach is helpful for large aneurysms, those with suprarenal extension, and those with associated visceral-vessel disease requiring correction. Furthermore, third-space fluid losses appear less, and the operation is safer for patients with limited cardiac or pulmonary reserve.[4,5,8,9] Retrospective studies comparing the transperitoneal and retroperitoneal approaches have confirmed these advantages of retroperitoneal aortic reconstruction in that individuals undergoing the retroperitoneal approach appear to have a smaller intraoperative blood loss, less crystalloid requirements, shorter postoperative ileus, and a shorter hospital stay than those undergoing transperitoneal aortic reconstruction.[10,11] However, in a randomized prospective study of 113 patients, some of the above impressions were not substantiated.[12] No statistical differences were found between the retroperitoneal and transabdominal approaches regarding operative and aortic cross-clamp times, perioperative crystalloid and transfusion requirements, and degree of

hypothermia on arrival at the intensive care unit. Likewise, there were no statistically significant differences in respiratory morbidity, recovery of gastrointestinal function, requirements for narcotics, metabolic parameters of operative stress, incidence of major and minor complications, or length of hospital stay. Despite lack of statistically significant differences, there were some trends that suggested significant differences might exist had more patients been studied. For example, postoperative mechanical ventilation requirements were 39% in the transperitoneal group versus 22% in the retroperitoneal group. Likewise, over twice as many in the transperitoneal group required nasogastric suction compared to the retroperitoneal group.

Recently, we reported our experience with 85 technically challenging aortic reconstructions.[9] These were performed for both complicated aortic problems and complicated patient problems. Aortic pathology included juxta-suprarenal extension of the aneurysm or occlusive plaque, reoperative aortic reconstruction, large inflammatory aneurysms, and associated visceral arterial lesions requiring repair. Patient problems included morbid obesity, the hostile abdomen from previous abdominal operations, abdominal wall stomas, and horseshoe kidneys. One-half of the patients required supraceliac cross-clamping of the aorta. The elective 30-day postoperative mortality was 1.2%. This compared favorably to mortality rates for elective transperitoneal infrarenal aortic aneurysm repair and aortofemoral bypass grafting of 2.6% and 2.9%, respectively, during the same 5 years when the 85 technically challenging operative procedures were performed.

Operative Technique

Proper positioning of the patient is important. The left thorax is elevated 45 to 60 degrees, while the hips should lie as flat as possible to allow access to the right groin should that become necessary. The patient's position is secured by placement on a vacuum styrofoam beanbag. The midpoint between the patient's left costal margin and iliac crest is centered over the table flexion point, and flexing the operating table causes the incision to spiral open. Wound closure is facilitated by flattening the operating table.

The surgeon stands to the left of the patient. Rotation of the table away from the surgeon facilitates retroperitoneal dissection while rotation toward the surgeon facilitates groin dissection.

An oblique left flank incision is used starting midway between the umbilicus and symphysis pubis and extending from the lateral margin of the left rectus sheath into the 11th intercostal space for 8 to 10 cm. The abdominal wall and intercostal muscles are divided in the line of the incision, taking care not to injure the 11th and 12th dorsal neurovascular bundles. Damage to these nerves denervates the abdominal wall, leading to muscle weakness, manifest as an asymmetric abdominal contour with unsightly bulging.

The retroperitoneal space is entered at the tip of the 12th rib, and with blunt dissection the anterior peritoneum is dissected away from the transversalis fascia as far as the rectus sheath. Dissection medial to the rectus is not necessary for exposure and also prevents tearing the peritoneum where it is firmly attached at the lateral border of the rectus.

Posterolaterally, the plane of the flank musculature, psoas, and diaphragm are followed as the peritoneal sac and its contents are dissected and retracted anteriomedially. This plane is developed along the lumbodorsal fascia behind the left kidney, mobilizing the kidney and ureter anteriorly. Alternatively, dissection can be performed anterior to the left kidney and ureter, but an advantage of the retroperitoneal approach is lost since the left renal vein obscures the juxtarenal aorta. However, dissection anterior to the kidney is useful when exposure of the superior mesenteric artery (SMA) beyond its origin is required for endarterectomy or when endarterectomy of the pararenal aorta is anticipated.

The aorta is easily exposed from above the left renal artery to the aortic bifurcation. To prevent injury to the left renal artery, its identification is important. The artery can usually be identified behind the lumbar branch of the left renal vein, which is a fairly constant structure. Ligation of this lumbar branch, which crosses over the aorta, provides good exposure of the aorta and origin of the left renal artery.

Lymphatics and fat overlying the aorta are ligated to minimize lymphorrhea. Blunt dissection of the aorta anteriorly and posteriorly, either above or below the renal arteries, is performed to allow placement of the proximal clamp. Circumferential aortic dissection is not required as long as the tip of the clamp can reach beyond the aortic wall. Inferior vena cava injury is not of concern, as it is not immediately adjacent to the aorta at this level.

If suprarenal aortic control is required, dissection is carried cephalad, with longitudinal division of the diaphragmatic crus. Suture ligation of the areolar tissue surrounding the origin of the SMA minimizes potential lymphatic leaks. With the need for supraceliac exposure, dissection proceeds further cephalad. Investing fascia around the aorta is incised and blunt dissection anteriorly and posteriorly creates tunnels to accommodate the jaws of an aortic clamp. Supraceliac control is often easier to obtain than juxtarenal control because of the relative paucity of lymphatics and fat at the paraceliac level. If juxtarenal or supraceliac aortic clamping is anticipated preoperatively, a 9th or 10th intercostal space incision is recommended.

Distal exposure of the iliac arteries is performed by blunt dissection of the peritoneal sac out of the left iliac fossa. The left iliac artery can be easily exposed over its entire length. Exposure of the right iliac artery is more challenging. Minimal dissection of the distal aorta is required when managing occlusive disease and the inferior mesenteric artery (IMA) is preserved. With aneurysmal disease, however, IMA ligation is often required to facilitate access to the right common iliac vessels. However, even

with division of the IMA, right iliac access can be difficult. To avoid the hazards of ileocaval injury resulting from dissection of the proximal right common iliac artery for clamp application, distal control can be obtained using intraluminal balloon catheter occlusion by threading the catheter into the right iliac arterial ostium through the opened aneurysm.

Anastomoses to the right distal common or external iliac arteries are difficult through the left retroperitoneal approach. Some authors have advocated extending the abdominal incision across the midline into the right lower quadrant to facilitate right iliac arterial exposure.[7,10] We have found that a right lower quadrant counter incision a few centimeters above the inguinal ligament with extraperitoneal dissection of the iliac vessels provides excellent exposure for right external iliac artery anastomoses. This avoids groin dissections with the small but real risk of infection. However, if needed, we do not hesitate to make groin incisions.

Pitfalls of Retroperitoneal Aortic Exposure

Certain pitfalls of retroperitoneal aortic reconstruction deserve comment. These include injury to the vena cava, which is very difficult to manage through the retroperitoneal approach. Vigorous retraction in the upper aspects of the operative field may lead to unrecognized splenic trauma. This should be minimized by using self-retaining retractors. The lumbar branch of the left renal vein must be identified because it serves not only as a marker to the left renal artery but to avoid injuring it as well. Similarly, although rare, a retroaortic left renal vein or circumaortic left renal vein may cause problems if not recognized. During mobilization of the retroperitoneum it is important to identify the left gonadal vein so that, during the course of sweeping the retroperitoneum anteriorally, the gonadal vein is not avulsed from the left renal vein. A left pneumothorax may occur and be unrecognized, particularly if the left 11th intercostal space incision is not made carefully.

Both the IMA and the left renal artery are swept anteriorally when retrorenal dissection is performed and must be identified to prevent injury. In order to preserve continuity of the IMA, it is important to incise the aneurysm posterior to its origin. The left ureter must be identified and swept anteriorally with the kidney and retroperitoneal structures to avoid injury. One should be aware that the left ureter is particularly vulnerable to traction injury during secondary aortic procedures when it may be tethered in the pelvis and not as mobile as it is in a virgin retroperitoneum.

Postoperative problems following retroperitoneal aortic reconstruction include the potential for development of an aortoenteric fistula if the aneurysm sac is not imbricated over the prostheses or if the duodenum lies on the graft following aortic reconstruction for occlusive disease.

Excessive blunt retroperitoneal dissection may cause tearing of small

veins and subsequent oozing with postoperative retroperitoneal hematoma formation. Precise dissection and careful hemostasis prevent this. Some have suggested closed suction drainage for 24 hours to minimize retroperitoneal hematoma formation. However, placing drains near a fresh aortic graft is not recommended. A left flank hernia may develop if precise multiple layer closure of the wound is not performed. Denervation of the abdominal musculature, manifest by asymmetric abdominal wall bulging, is prevented by carefully preserving the intercostal neurovascular bundles.

Summary

Retroperitoneal exposure of the abdominal aorta and its branches has become a well established surgical procedure. Some of the advantages that were originally anticipated have not been realized, however. Nonetheless, there are occasions when retroperitoneal exposure can facilitate difficult aortic reconstructions. Indications for its use include small infrarenal aneurysms localized to the aorta; large complex suprarenal or juxtarenal aneurysms; aortic reconstruction requiring left renal arterial and/or mesenteric revascularization; and for patients with horseshoe kidneys; patients with right-sided ostomies; and some morbidly obese individuals. It is also indicated for patients who have a hostile abdomen resulting from extensive intraabdominal adhesions, radiation therapy, or inflammatory processes, and those undergoing secondary aortic reconstruction.

Contraindications to use of the retroperitoneal approach include need for right renal arterial reconstruction, need to assess intraabdominal organs, and extensive aneurysmal involvement of the right iliac system.

References

1. Cooper A. Case of femoral aneurysm for which the external iliac artery was tied by Sir A. Cooper. Guy's Hosp Rep 1:43, 1836.
2. Dubost D, Allary M, Oeconomos N. Resection of the aneurysm of the abdominal aorta: Reestablishment of the continuity by a preserved human arterial graft, with result after 5 months. Arch Surg. 64:405, 1952.
3. Shumaker, HB. Midline extraperitoneal exposure of the abdominal aorta and iliac arteries. Surg Gynecol Obstet. 135:791, 1972.
4. Williams GM, Ricotta J, Zinner M, et al. The extended retroperitoneal approach for treatment of extensive atherosclerosis of the aorta and renal vessels. Surgery 88:846, 1980.
5. Shepard AD, Scott GR, Mackey WC, et al. Retroperitoneal approach to high risk abdominal aortic aneurysms. Arch Surg. 121:444, 1986.
6. Oudot J. La greffe vasculaire dans les thromboses du carrefour aortique. Presse Med 59:234, 1951.
7. Rob C. Extraperitoneal approach to the abdominal aorta. Surgery 53:87, 1963.
8. O'Mara CS, Williams GM. Extended retroperitoneal approach for abdominal

aortic aneruysm repair. In aneurysms. Diagnosis and treatment. Bergan JJ and Yao JST (eds). Grune and Stratton Inc., pp. 327–343, 1981.
9. Shepard AD, Ernst CB, Tollefson, DFJ, et al. Left flank retroperitoneal exposure: A technical aid to complex aortic reconstruction. J Vasc Surg. (in press).
10. Sicard GA, Freeman MB, VanderWoude JC, et al. Comparison between the transabdominal and retroperitoneal approach for reconstruction of the infrarenal abdominal aorta. J Vasc Surg. 5:19, 1987.
11. Leather RP, Shah MS, Kaufman JL, et al. Comparative analysis of retroperitoneal and transperitoneal aortic replacement for aneurysm. Surg Gynecol and Obstet 168:387, 1989.
12. Cambria RP, Brewster DC, Abbott WM, et al. Transperitoneal versus retroperitoneal approach for aortic reconstruction: A randomized prospective study. J Vasc Surg. 11:314, 1990.

12
Difficult Aortic Aneurysms: Pararenal and Suprarenal Aneurysms, Inflammatory Aneurysms, and Concomitant Renal or Visceral Revascularization

JOSE MENA and LARRY H. HOLLIER

General Considerations

An aneurysm of the abdominal aorta is defined as any dilatation greater than 1-1/2 times the normal diameter of the native vessel,[1] while dilatations of lesser dimensions are termed ectasia.[1,2] They usually represent the degenerative result of aging of the aorta. Abdominal aortic aneurysms (AAA) occur in about 5% of the population over the age of 60 years in the United States.

There exists an apparent increase in the incidence of aortic aneurysms, which has been attributed to the increasing age of the population and the widespread use of improved diagnostic techniques such as ultrasound, computed tomography, magnetic resonance imaging, and angiography.[3]

Preoperative Evaluation

Preoperative assessment of a patient with an aortic aneurysm begins with the history and physical examination. The majority of patients do not experience symptoms unless there is rapid expansion or rupture. Approximately 75% of abdominal aortic aneurysms are incidentally found on physical examination, on roentgenographic examination being performed for other reasons, during celiotomy, or on autopsy studies. Upon palpation of the abdomen, the examiner can feel a pulsatile mass. Since the aortic bifurcation is at the level of the umbilicus, the aneurysmal mass is located above this point in the epigastrium. In infrarenal aneurysms, the lateral borders can be felt below the costal margins. If the cephalad border of the aneurysm appears to extend above the costal margin, one must suspect a pararenal or suprarenal aneurysm.

Ultrasound examination of the abdomen will reveal the size of infrarenal abdominal aortic aneurysms but has severe shortcomings when evaluating aneurysms that extend up to or above the level of the renal arteries.[4] Com-

puted tomography (CT) with intravenous contrast provides excellent visualization of the aneurysm, accurately defines the extent of involvement, and will identify complicating factors such as venous anomalies of the inferior vena cava and renal veins, horseshoe or ectopic kidneys, or the presence of an inflammatory abdominal aortic aneurysm.

Aortograms do not constitute part of our routine preoperative assessment of abdominal aortic aneurysms. Since the clot that lines the lumen of the aneurysm may give the false impression that there is no dilatation, especially if only the anteroposterior projection is used, angiography may be misleading. However, aortograms (Fig. 12.1) may be obtained in the face of:

1. renovascular hypertension;
2. impairment of renal function;
3. suspected pararenal or suprarenal extension;
4. intestinal angina;
5. suspicion of horseshoe kidney;
6. suspected femoral, popliteal or other aneurysm;
7. abdominal or flank bruit;
8. peripheral arterial occlusive disease.

All patients undergoing repair of AAA usually have a complete blood count, serum electrolytes, blood urea nitrogen and creatinine, liver function tests, coagulation studies, electrocardiogram, chest X-ray, and urinalysis. Based upon findings from the patient's history and physical examination, one may proceed with other tests such as pulmonary function tests, carotid duplex scanning, complete coagulation evaluation, or cardiac evaluation. Since 50% of patients with AAA have coronary artery occlusive disease,[2,3,5] cardiac evaluation by noninvasive means may be helpful in assessing operative risk.

The dipyridamole-thallium scintigraphy of the heart or exercise stress electrocardiography will identify those patients with significant coronary artery disease. Because suprarenal and juxtarenal extension of the aneurysm, as well as other conditions, may require clamping above the visceral arteries, which places additional strain upon the heart and increases the risk of myocardial dysfunction and infarction, noninvasive evaluation may assume vital importance. If these studies suggest significant coronary artery disease, coronary angiography should be carried out, with subsequent myocardial revascularization if critical stenoses are found.[4] Aneurysmorrhaphy should then follow in approximately 6 weeks.

Abdominal aortic aneurysm (AAA) repair is one of the more frequently performed operations in vascular surgery. Even though the majority of those are repaired by straightforward techniques familiar to most surgeons, certain conditions may exist that will require a change in strategy. The rest of this chapter will deal with some of those conditions, and our method of management.

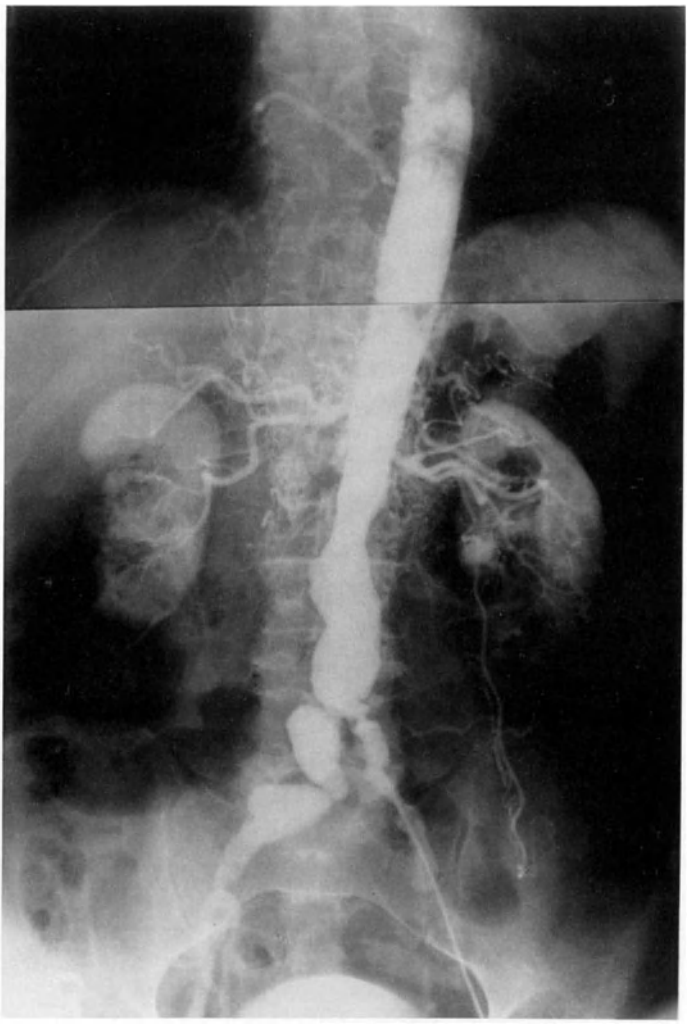


FIGURE 12.1. Aortogram of a patient with an infrarenal abdominal aortic aneurysm, right common iliac aneurysm, occluded visceral arteries, and stenotic renal arteries. This patient was treated by transaortic endarterectomy of the visceral and renal vessels, replacement of the infrarenal aorta and iliac arteries with an aorta-bi-iliac Dacron graft, and reimplantation of the IMA.

Intraoperative Monitoring

Strict monitoring of cardiovascular, pulmonary and renal function is beneficial for assuring a successful outcome. There must be close cooperation between the surgical and anesthetic teams.

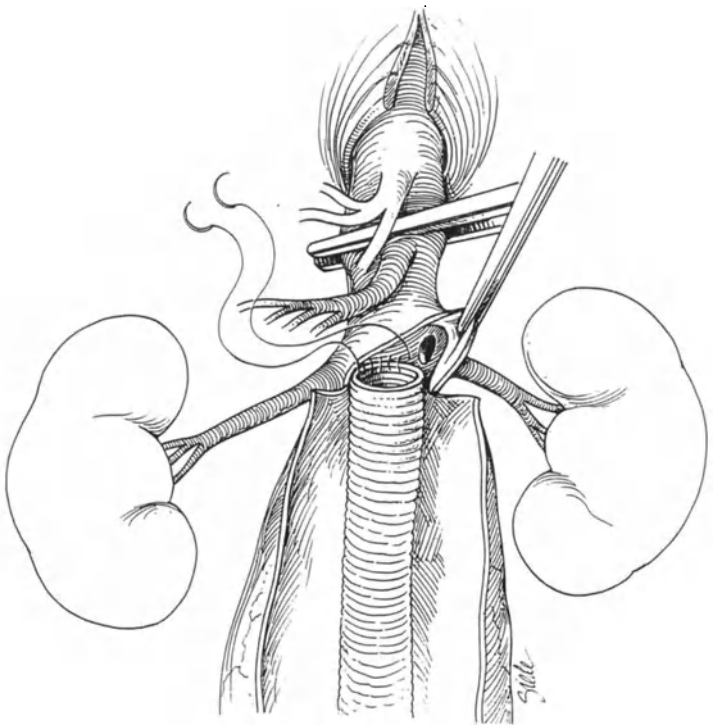
We routinely employ the use of a radial artery line for continuous monitoring of arterial blood pressure and for drawing of blood for hematocrit and electrolyte determinations. In high-risk patients, an oximetric Swan-Ganz catheter is used for serial monitoring of cardiac indices and cardiac filling pressures, and continuous display of mixed venous oxygen saturation. When supraceliac cross-clamping is anticipated or there is preexisting cardiac dysfunction, real-time B-mode transesophageal echocardiography is often employed, as this is the quickest mode of detecting cardiac dysfunction, allowing either fluid or pharmacologic intervention. Because of the concern about the transmission of bloodborne disease, we routinely employ the autotransfusion device to minimize the need for banked blood. Additionally, we ask that the patient donate two units of autologous blood prior to surgery. An indwelling urinary bladder catheter is placed to continuously monitor urine output. We begin antibiotics in the preoperative period and continue this for 48 to 72 hours after surgery or until all central lines have been removed.

Pararenal (Juxtarenal) and Suprarenal Abdominal Aortic Aneurysms

Although the vast majority of AAAs occur in the infrarenal location, approximately 2% extend proximally to involve the renal and/or visceral arteries.[2] Juxtarenal aneurysms are those in which there is no normal segment of aorta below the renal arteries, while suprarenal aneurysms involve the orifice of at least one renal artery and may extend cephalad to involve the visceral vessels.

The operative approach to suprarenal and pararenal aneurysms depends on the proximal and distal extent of the disease and associated risk factors, especially chronic obstructive pulmonary disease. For juxtarenal aneurysms in good risk patients, especially if the aneurysmal disease involves the iliac arteries, a midline transperitoneal approach is employed. An extraperitoneal flank approach is used if the aneurysm extends suprarenally or if significant chronic obstructive pulmonary disease is present.

To gain access to the suprarenal aorta via the transperitoneal route, a xiphoid-to-pubis incision is made. The small bowel is mobilized to the right, wrapped with sterile towels, and gently retracted. Division of the ligament of Treitz is carried out for further mobilization of the duodenum. The inferior mesenteric vein, where it crosses the base of the mesentery, is doubly ligated and divided. The left renal vein is identified and mobilized after the left adrenal vein is doubly ligated and divided. If one anticipates significant traction on the renal vein, one may elect to identify, doubly ligate and divide the posterior tributary of the renal vein that goes to the lumbar venous plexus, as this can be a site of troublesome bleeding if avulsed. Although some authors advocate division of the left renal vein, we



A

FIGURE 12.2A. Technique for replacement of the aorta in a patient with juxtarenal abdominal aortic aneurysms. The proximal anastomosis is placed along the inferior margin of the renal artery orifices. (From Hollier LH, and Moore WM: Surgical Management of juxtarenal and suprarenal aortic aneurysms. Reprinted with permission from Acta Chir Scand Suppl 555:117–122, 1990.)

do not generally find this necessary. If transection of the left renal vein is necessary, the left adrenal vein and the posterior tributary to the left renal vein must be left intact, as these provide alternate routes for venous drainage from the left kidney.

Application of gentle downward traction on the left renal vein and division of the preaortic fatty tissue will provide exposure of the suprarenal aorta. The crus of the diaphragm, which can be felt as a tight fibromuscular band to the left of the aorta, is divided. The left adrenal artery, found 2 to 3 cm caudad to the crus, is doubly clipped and divided. Digital blunt dissection cephalad to this point will develop a plane posterior to the pancreas. Dissection anterior and posterior to the aorta will allow placement of clamp above the superior mesenteric artery, or celiac artery if needed. This approach is usually restricted to pararenal aneurysms (Fig. 12.2A) or involvement of the aorta no higher than the superior mesenteric artery.[6]

An aortic occlusive clamp is placed above the superior mesenteric artery

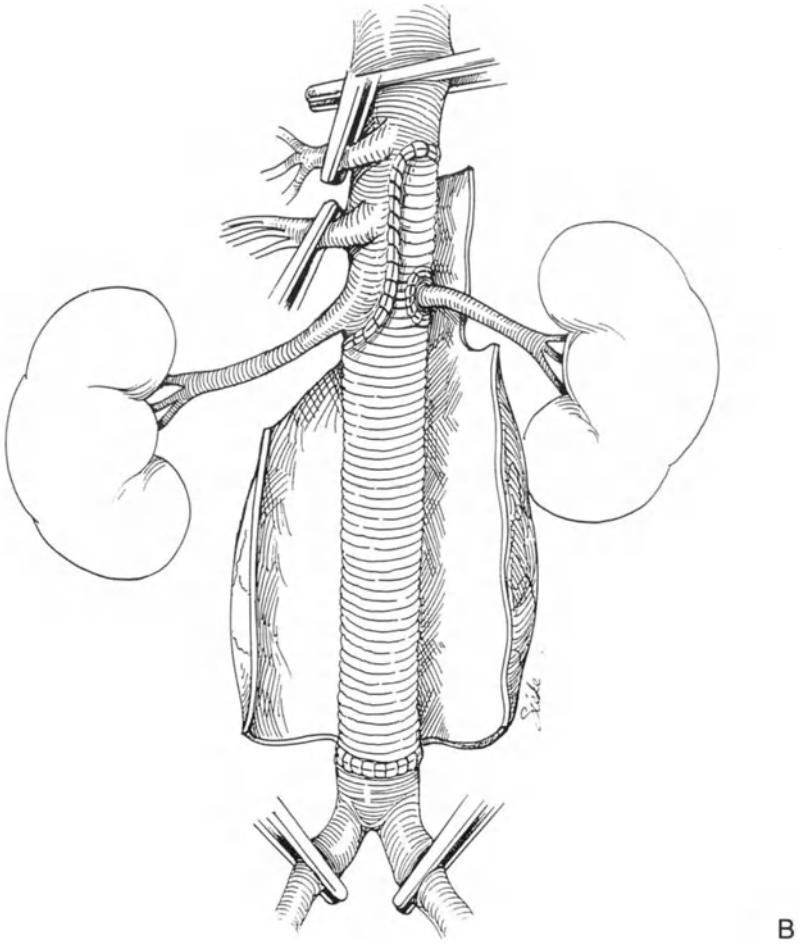


FIGURE 12.2B. Technique for reimplantation of visceral and renal arteries in a patient with suprarenal abdominal aortic aneurysms. The celiac superior mesenteric and right renal arteries are implanted as a single anterior tongue. The left renal artery is reimplanted separately with a Carrel patch technique. (From Hollier LH, and Moore WM: Surgical management of juxtarenal and suprarenal aortic aneurysms. Reprinted with permission from Acta Chir Scand Suppl 555:117–122, 1990.)

and a second clamp is applied above the inferior mesenteric artery. The infrarenal aorta is transected, and after the left renal vein is gently retracted downwards, a vertical opening is made on the anterior surface of the aorta with the cephalad extent of the aortotomy placed between the left renal artery and the left side of the superior mesenteric artery. A graft is then placed with reimplantation of the superior mesenteric artery and both

renal arteries as a single anterior tongue in the proximal anastomotic suture line. Once the proximal anastomosis is done, the proximal clamp is replaced on the graft below the renal arteries. This restores flow to the kidneys and the superior mesenteric artery within a short period of time, usually less than 15 minutes occlusion time. The distal anastomosis is then carried out in the usual fashion.

Aneurysms that extend proximal to the superior mesenteric artery, especially in a patient with compromised respiratory status, are best approached via a left flank incision, with the dissection carried out extraperitoneally. The patient is placed supine on the operating room table and then the chest is rotated approximately 45 degrees toward the patient's right. The incision is started at approximately the left 11th intercostal space and carried down obliquely across to the right paramedian area of the abdomen.

The fascia and muscles are divided in the direction of the skin incision, but the peritoneum is not entered. Mobilization is begun at the level of the left iliac crest and continued superiorly, with the plane of dissection being maintained anterior to the left kidney and renal vessels. In the superior portion of the wound, the peritoneum is gently dissected from the undersurface of the diaphragm; mobilization of the abdominal viscera is completed by sweeping them anteriorly and medially. This exposes the crus of the diaphragm, which may be sharply incised if necessary. Ligation and division of the left adrenal vein and mobilization of the left renal vein facilitates visualization of the left renal artery. The distal aorta is then exposed. If adequate collateral circulation through the superior mesenteric artery and the hypogastric arteries is evident, further exposure is made possible by doubly ligating and dividing the inferior mesenteric artery. The iliac arteries can be exposed fairly easily with a little extra distal dissection. A clamp is then placed at the level of the supraceliac aorta, and a vertical aortotomy is made anterior to the left renal artery and lateral to the superior mesenteric and celiac arteries. A single anterior tongue can be used to reimplant the celiac, superior mesenteric and right renal arteries as part of the proximal anastomosis. The left renal artery is reimplanted separately as a Carrel patch (Fig. 12.2B). The distal anastomosis is then carried out in standard fashion after flow to the visceral and renal vessels has been reestablished by replacing the aortic clamp below the renal arteries.

Potential Problems

Potential problems that may ensue include hemorrhage, thrombosis, distal arterial occlusion, pseudoaneurysm formation, infection, aorta-enteric fistula, pulmonary or cardiac failure, and erectile dysfunction in males. Renal failure and paraplegia may result from repair of complicated aneurysm, and steps to prevent them should be implemented.

Adequate hydration preoperatively and judicious administration of fluids during surgery are vital. We employ the use of 25 grams of mannitol and 20 mg of furosemide approximately 20 to 30 minutes prior to interrupting renal perfusion. Perfusion of the kidneys with iced heparinized electrolyte solution is used occasionally, but since renal ischemia time is usually brief, we generally do not utilize this method of renal protection. Stenoses of the renal arteries are treated either by transaortic endarterectomy or bypass grafting. Postoperatively, the urine output is maintained at approximately 50 ml/hr or greater. Many patients will develop a high output state, and replacement of urinary output on a deciliter per deciliter basis every hour is necessary if outputs greater than 500 cc per hour exist. This measure can generally be discontinued within 8 to 12 hours of surgery.

Paraplegia and paraparesis are exceedingly rare after repair of uncomplicated pararenal or suprarenal aneurysms. Nevertheless, if clamping is required at or above the diaphragm, the artery of Adamkiewicz can be deprived of flow. Paraplegia appears to be directly related to decreased blood flow in the spinal cord during aortic clamping.[7,8] To ascertain adequate spinal cord perfusion, we generally maintain systolic arterial blood pressure in the 160–180 mmHg range, and cerebrospinal fluid (CSF) pressure below 10 mmHg. The difference between these two variables constitutes the spinal cord perfusion pressure; increasing systemic arterial blood pressure while decreasing CSF pressure should theoretically maintain adequate spinal cord perfusion.

At the time of surgery, an indwelling intrathecal catheter is placed at the L3 to L4 level. Enough CSF, generally 10 to 20 cc, is removed to bring the pressure to under 10 mmHg as monitored through the catheter connected to a transducer. CSF pressure is continuously measured throughout the procedure and additional volumes of fluid withdrawn to maintain the pressure below 10 mmHg. The catheter is left in place for approximately 24 to 48 hours after operation; in the intensive care unit, CSF is removed to maintain pressure below 15 mmHg. We employ this technique on all aneurysms that require clamping of the aorta above the level of the diaphragm.

Inflammatory Abdominal Aortic Aneurysm

Inflammatory abdominal aortic aneurysms (IAAA) have been reported to comprise up to 15.1% of abdominal aortic aneurysms (AAA),[9] although larger series have documented a lower incidence. In a review of 2,816 patients operated upon for AAA over a 30-year period, Pennell et al.[10] reported an incidence of 4.5%. Crawford et al.[11] reported an incidence of 2.9%, while Goldstone et al.[12] found IAAA in 5% of 200 AAA.

This condition is characterized by intense fibrosis and thickening of the aortic wall.[13] The aneurysm is surrounded by a thick, shiny, highly vascu-

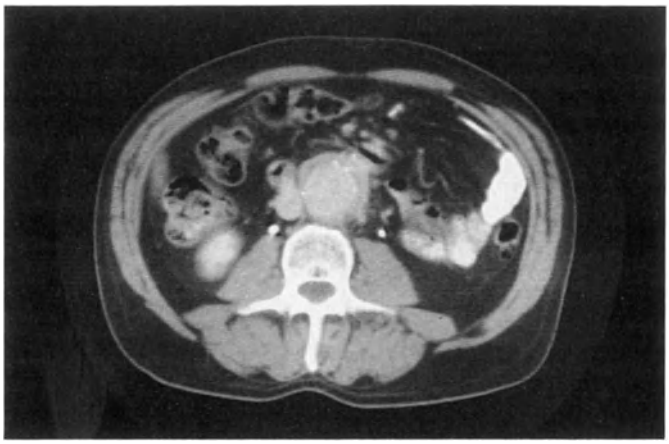


FIGURE 12.3. CT scan showing inflammatory abdominal aortic aneurysm. Note the prominent periaortic inflammatory process.

lar, whitish/pink fibrotic process. This reaction may extend away from the aneurysm to involve the left renal vein, the ureters, the inferior vena cava, the duodenum and other portions of the bowel. The anterior and lateral walls of the aorta are thickened, measuring from 1 to 4 cm. The thickening is not uniform, with the posterior wall and, to a lesser extent, the lateral wall, being thinner; it is at these locations that rupture may occur.[14]

Patients having IAAA present with symptoms more frequently than their noninflammatory counterpart. In Pennell et al.'s review, abdominal or back pain was present in 60%, 20% exhibited weight loss, while 10% experienced anorexia. The erythrocyte sedimentation rate (ESR) was elevated in over 70% and is the only blood test that suggests the diagnosis.

CT scan is diagnostic when it shows the contrast enhanced thickened inflammatory wall surrounding the aorta (Fig. 12.3). The abdominal ultrasound may show a sonolucent "halo" around the aorta. Angiography does not assist in making the diagnosis of this disorder. Intravenous pyelography may suggest the presence of the inflammatory process by revealing hydronephrosis and/or medial deviation of the ureters.

If an IAAA is suspected, ureteral catheterization and placement of indwelling ureteral stents should be undertaken prior to surgery. The stents will make it easy to identify the ureters, by palpation, during the operative procedure. If obstruction is present, it is prudent to delay operation after placement of the stents since a postobstructive diuresis may ensue, which may cause severe alteration in the fluid and electrolyte balance.

At surgery, an inflamed retroperitoneal mass is found. The duodenum is usually adherent at the right anterolateral aspect of the mass—DO NOT ATTEMPT TO DISSECT THE DUODENUM AWAY FROM THE

MASS. Exposure of the aorta can be obtained by incising the base of the transverse mesocolon, cutting the ligament of Treitz, and exposing the left renal vein. The left renal vein may be mobilized, exposing the underlying aorta if it is not severely involved by the inflammatory process. Dissection is then carried out very carefully on each lateral wall so that placement of an aortic occluding clamp may be safely accomplished.

If the left renal vein is stuck to the aortic wall, it means that the inflammatory process extends at least up to the renal arteries. In this situation, it is safer to approach the aorta by mobilizing the left colon, spleen, pancreas, and left kidney, rotating them medially.

One should generally avoid dissection of the common iliac arteries since these are usually involved to some degree by the inflammation. The distal anastomoses are best made in the distal external iliac arteries, which are almost invariably free of the inflammatory process. Tunnels for each limb of the bifurcation graft to the distal external iliac arteries are made either lateral or posterior to the inflammatory mass and either medial or lateral to the iliac arteries as long as they are deep to the ureters.

A vertical aortotomy is then made after proximal and distal occlusion is achieved. Proximal control may be necessary just below or above the superior mesenteric artery depending on the extent of the process, while in the most severe cases, supraceliac clamping may be necessary. The orifices of the common iliac arteries are oversewn, unless the distal anastomoses are to be made at this level. We routinely perform endarterectomy in an IAAA, since it has been proposed that the inflammatory process is the result of an autoimmune reaction to the diseased atheromatous intima of the aneurysm. The proximal anastomosis is then performed in a standard "inlay" fashion. The distal anastomoses are performed in an end-to-side fashion to provide retrograde flow into the hypogastric arteries. Flow is then restored through the graft, and it is covered with the aneurysm wall.

Concomitant Renal Revascularization

Five to 10% of all patients undergoing repair of abdominal aortic aneurysms have significant renal artery stenosis, but only 50% of these require concomitant revascularization.[15] Hollier et al.[15] reported that 3.1% of 1112 AAA repairs required concomitant renal revascularization. In a multicenter study, 2.1% of 666 patients required renal artery revascularization at the time of aneurysm repair.[18]

Renal artery revascularization should be considered when there is poorly controlled arterial hypertension, deterioration in renal function, and when preservation of renal function is pursued. Additionally, renal artery aneurysms should be repaired at the time of AAA repair. The latter is very uncommon and will not be discussed.

We perform concomitant revascularization of the renal arteries for ste-

nosis greater than 70% in a patient with severe or progressive hypertension, or renal dysfunction secondary to the stenosis. Revascularization in an asymptomatic patient is also carried out when high grade stenosis, greater than 95%, is present. This is done because of the progressive nature of the atherosclerosis, which threatens with complete occlusion and loss of renal function. A concomitant procedure should obviate the need for reoperation.

If there is proximal bilateral renal artery stenosis, transaortic renal endarterectomy is generally performed. If bilateral nonorificial renal artery stenoses exist or there is unilateral stenosis, bypass to the renal arteries is undertaken from the aortic graft to the distal renal artery. Alternatively, nonorificial stenoses may be treated with balloon angioplasty prior to aneurysm repair.

A midline transperitoneal abdominal approach is employed for patients with AAA and bilateral renal artery stenosis. The aorta is exposed up to the left renal vein, which is subsequently mobilized. The left adrenal and gonadal veins are doubly ligated and transected. This allows for safe traction on the left renal vein, which need not be divided. At the level of the superior mesenteric artery to the left of the aorta, the crus of the diaphragm is found. This is divided for more proximal exposure. The overlying pancreas is elevated with the use of a Wylie renal vein retractor.

A transverse aortic clamp is placed above the SMA, and the iliac arteries are individually cross-clamped. A vertical aortotomy is done from just left of the SMA down to the aortic bifurcation. Bleeding lumbar arteries are oversewn. Endarterectomy of the aorta and renal arteries is then performed. An attempt is made to stay in the superficial medial plane to avoid creation of intimal flaps in the renal arteries. Constant, gentle, central traction is applied to the plaque, and eversion of the renal arteries will permit better visualization and control of the distal endpoints. Once the endarterectomy is completed, No. 3.5 or 4 Fr. dilators are passed through each renal artery to verify patency of the vessel. The aortotomy is then closed with a running 3.0 prolene suture from above down to the neck of the aneurysm. Renal perfusion is then reestablished by moving the clamp to the aorta just below the renal arteries. The infrarenal aorta is then replaced with a prosthestic graft.

The adequacy of the revascularization is then tested with either the intraoperative Doppler or the electromagnetic flowmeter. Flows of 100 to 450 cc/min are expected; flows of less than 100 cc/min raise the suspicion of an intimal flap.

Aortorenal bypass is used for the treatment of unilateral or bilateral nonorificial renal artery stenosis. A midline transperitoneal approach is used, the left renal vein is mobilized as previously described, and the renal arteries are fully exposed out to the branch vessels. The AAA is replaced with a prosthetic graft in the standard manner. Once flow through the graft is restored, a partial occluding clamp is placed approximately 1 cm below

the proximal anastomosis. For a right renal bypass, the clamp is placed on the right lateral aspect of the graft; it is placed on the left anterolateral portion of the aortic graft for a left renal artery bypass, since a graft to the left renal artery will generally loop anterior to the left renal vein. A 6 mm knitted velour dacron graft is sutured end-to-side to the aortic graft using 5.0 prolene sutures in a running fashion. The renal artery is suture ligated at its origin and then divided. Spatulation of the distal end of the renal artery is carried out on the anterior aspect, and the dacron graft is then sutured end to end with 7-0 prolene suture to the renal artery. Once bypass is complete and flow reestablished, the aneurysm wall is closed over the graft. Partial excision of the side of the aneurysm sac is performed to allow holes for exit of the bypass.

Concomitant Visceral Revascularization

Patients who have AAA and symptomatic chronic intestinal ischemia are best treated by aneurysm repair and revascularization of the visceral vessels. When both celiac and SMA are occluded, an extraperitoneal approach is preferred.

Exposure of the aorta, with proximal and distal control, and the aortotomy are carried out as described for renal vascularization; the proximal clamp, however, is placed on the supraceliac aorta and the aortotomy is carried just cephalad and to the left of the celiac axis. Transaortic endarterectomy of the visceral arteries is then performed and the infrarenal aorta replaced with a dacron graft. If endarterectomy is not possible, bypass with a bifurcated 14×7 mm dacron velour graft from the supraceliac aorta to the celiac and superior mesenteric arteries, respectively, is done. The infrarenal AAA is then repaired with a prosthetic graft in the usual manner.

In the patient with ASYMPTOMATIC celiac artery and SMA occlusion, we revascularize, at the time of AAA repair, at least one major visceral vessel. If the inferior mesenteric artery (IMA) is large and tortuous, reimplantation of this vessel is carried out.

Concomitant revascularization of only one visceral artery is done by a short segment graft placed end-to-side to the supraceliac aorta for celiac artery bypass, or to the aortic graft for SMA bypass. A Carrel patch technique is usually used for IMA reimplantation. If the orifice of the IMA is occluded upon opening of the aneurysm, it may be oversewn. If the IMA is open, it should be occluded and one should reevaluate the collateral blood supply to the colon at the end of aneurysm repair, since restoration of flow to the hypogastric arteries may provide excellent collateral flow up to the IMA and left colic artery.

No matter what the condition of the IMA and other visceral vessels, revascularization of at least one of the hypogastric arteries is carried out. A

tube graft of the aorta or a bifurcated graft to the common iliac arteries provide direct flow to the hypogastric arteries. The latter, however, are not perfused if bypass to the distal external iliac or femoral arteries is carried out in the face of bilateral proximal external iliac artery occlusion. In this scenario, a separate bypass is performed directly to one of the hypogastric arteries.

Once the AAA is repaired, back-bleeding from the IMA's orifice is evaluated; the latter is oversewn if pulsatile backflow is present. If one is concerned about the adequacy of collateral circulation, IMA stump pressure is measured. Although others accept 40 mmHg as being adequate,[17] we prefer to see a pressure greater than 50 mmHg since we have seen mucosal ischemia with pressures of 40 mmHg. Additionally, colonic perfusion can be evaluated by injecting one or two ampules of fluorescein intravenously and then examining the bowel with the ultraviolet lamp. If the perfusion of the colon seems to be inadequate, a Carrel patch technique is used for reimplantation of the inferior mesenteric artery. Alternatively, a short segment vein interposition graft may be used to revascularize a critically needed IMA.

Summary

Aneurysm repair is usually uncomplicated and with very low morbidity and mortality. However, some anatomic variations or inflammatory changes, as described above, can be of significant importance and concern if encountered unexpectedly by the unwary surgeon. Careful preoperative evaluation can alert the surgeon to such problems and allow better preparation for operation or for judicious referral of the patient.

References

1. Johnston KW, Rutherford RB, Tilson MD, et al. Suggested standards for reporting on arterial aneurysms. J Vasc Surg. 13:452–458, 1991.
2. Hollier LH, Rutherford RB. Infrarenal aortic aneurysms. In Rutherford RB (ed): *Vascular Surgery*. Philadelphia, WB Saunders Co, 1989, 902–927.
3. Bickerstarff LK, Hollier LH, Van Peenen HJ, et al. Abdominal aortic aneurysms: The changing natural history. J Vasc Surg. 1:6–12, 1984.
4. Budden J, Hollier LH. Management of aneurysms that involve the juxtarenal or suprarenal aorta. Surg Clin N Am. 69:4, 837–844, 1989.
5. Reigel MM, Hollier LH. Surgical repair of abdominal aortic aneurysms. Cardiac Surg: State of the Art Reviews 1:453–465, 1987.
6. Hollier LH. Technical modifications in the repair of thoracoabdominal aortic aneurysms. In Greenhalgh RM (ed): *Vascular Surgical Techniques*. Kent, England, Butterworths, 1984, 98–106.
7. Bower TC, Murray MJ, Gloviczki, et al. The effects of thoracic aortic occlusion

and cerebrospinal fluid drainage on regional spinal cord blow flow in dogs: correlation with neurologic outcome. J Vasc Surg. 9(1):135–44, 1989.
8. McCullough JL, Hollier LH, Nugent M. Paraplegia after thoracic aortic occlusion: Influence of cerebrospinal fluid drainage. J Vasc Surg. 2:859–869, 1985.
9. Barr H, Cave-Bigley DJ, Harris PL. The management of inflammatory abdominal aortic aneurysms. J R Coll Surg Edinburgh 30:27–220, 1985.
10. Pennell RC, Hollier LH, Lie JT, et al. Inflammatory abdominal aortic aneurysms: a thirty-year review. J Vasc Surg. 2:859–869, 1985.
11. Crawford JL, Stowe CL, Safi HJ, et al. Inflammatory aneurysms of the aorta. J Vasc Surg. 2:113–124, 1985.
12. Goldstone J, Malone JM, Moore WS. Inflammatory aneurysms of the abdominal aorta. Surgery 83:425–430, 1978.
13. Hollier LH. Special problems in aortic aneurysm surgery. In Bergan JJ and Yao JST (eds): *Techniques in Arterial Surgery*. Philadelphia, WB Saunders Co, 1990 56–69.
14. Goldstone J. Inflammatory aneurysms of the aorta. Seminars in Vascular Surgery 1(3):165–173; 1988.
15. Hollier LH, Plate G, O'Brien PC, et al. Late survival after abdominal aortic aneurysm repair: Influence of coronary artery disease. J Vasc Surg. 1:290–299, 1984.
16. Johnston KW, Scobie TK. Multicenter prospective study of nonruptured abdominal aortic aneurysms. I: Population and operative management. J Vasc Surg. 7:69–81, 1988.
17. Ernst CB, Hagihara PF, Daugherty ME, et al. IMA stump pressure: a reliable index for safe IMA ligation during abdominal aortic aneurysmectomy. Ann Surg. 187:641–645, 1978.

13
Mycotic Aneurysm: In Situ vs. Extraanatomic Repair

MIRALEM PASIC

Summary

Twenty-eight patients with 30 mycotic aneurysms were operated on between January 1971 and December 1990. There were 24 males and 4 females ranging in age from 6 to 84 years (mean for adults, 63 years). Sixteen aneurysms from 30 (53%) were ruptured. In situ repair was undertaken in 22 (73%) patients. Four patients died during hospital stay and 24 survived. Two of the surviving patients died as a direct result of aneurysm. The estimated 1-year and 5-year survival rates were 62% and 36%, respectively. In situ repair after an extensive debridement of the aneurysmal wall and infected tissue with antibiotic therapy is a satisfactory method for treatment of mycotic aneurysm.

Introduction

Mycotic aneurysm is a rare life-threatening pathological entity.[1] It grows rapidly with subsequent rupture and high mortality.[2] To prevent these complications, aggressive antimicrobial and surgical therapy should be promptly undertaken.

Standard surgical treatment consists of aortic ligation and extraanatomic bypass.[3,4] However, when lesion is well circumscribed, in situ graft interposition with radical excision and retroperitoneal debridement may be undertaken.[5,6] In spite of antibiotic and surgical therapy, the morbidity and mortality remain great.[7] In this study we report the surgical treatment, results and long-term follow-up of a group of patients with mycotic aneurysms operated on at the University Hospital Zuerich between 1971–1990.

TABLE 13.1. Location of mycotic aneurysm.

Location	No. of patients		
	In situ		Extraanatomic
Ascending aorta	6		
Descending aorta	4		1
Suprarenal abdominal aorta	1		
Infrarenal abdominal aorta	6		5
Iliacal artery	1		1
Innominate artery	1		
Axillary artery	1		
Extracranial carotid artery	1		
Popliteal artery	1		
Radial artery		ligation	
Total	22	1	7

Patients and Methods

During the 20-year period from January 1, 1971, to December 31, 1990, we treated 28 patients with 30 mycotic aneurysms at the Cardiovascular Clinic of University Hospital Zuerich. All but one were adults, the mean adult age was 63 years, range 28–84 years. There were 24 males and 4 females. The diagnosis was made on clinical, microscopic and microbiologic findings. Patients who had elective aneurysm repair with positive findings on culture of aneurysm contents, incidentally discovered in the absence of other signs of infection, were not included. Likewise, patients treated for infection of prosthetic graft, with or without a false aneurysm, were not included. Sixteen aneurysms (53%) were ruptured. In four cases rupture occurred in the hospital. The location and method of treatment are summarized in Table 13.1. In situ repair was undertaken in 22 (73%) pa-

TABLE 13.2. Causative agent.

Agent	No. of patients
Salmonella	1
Pneumococcus	3
Streptococcus viridans	2
beta-hemolitic Streptococcus	1
Candida albicans + *Escherichia coli*	1
Escherichia coli	3
Mycobacterium TBC	2
Staphylococcus aureus	8
Unknown	7
Total	28

TABLE 13.3. Primary site of infection.

Site	No. of patients
Pneumonia	3
Sepsis	5
Fever	3
Sore throat	1
Active TBC	2
Osteomyelitis	3
Arthritis	1
Endocarditis	2
Pericarditis	1
Prostatitis	1
Intraabdominal abscess	1
Diverticulitis	1
Unknown	4
Total	28

tients (oversewing of the aneurysm was done in one, patch in 10, prosthetic graft in 9 and autologous venous graft in 2 patients). Simple ligation of the peripheral artery proximal and distal to the aneurysm and excision without grafting was our therapy in cases with mycotic aneurysm of the radial artery. Aortic or iliacal aneurysm required an axillofemoral bypass graft prior to excision of the aneurysm in six cases, and in one case was performed ascending aorta to infrarenal aortic bypass. Twenty-two patients were treated by antimicrobial therapy preoperatively. All patients received appropriate antibiotic treatment during and after surgical procedure.

Table 13.2 summarizes the pathogenic agents cultured from aneurysm or by positive blood-culture. The most common pathogen was *Staphylococcus aureus* (in 8 patients, 38% from known agents) followed by *Escherichia coli* (in 3 patients, 14%) and pneumococcus (in 3 patients, 14%). In one of our

TABLE 13.4. Factor predisposing to immunoincompetency.

Factor	No. of patients
Severe alcoholism	4
Chronic renal failure	2
Chronic obstructive pulmonary disease	2
Steroids	6
Aortic isthmus stenosis	1
Chronic pancreatitis	1
Active tuberculosis	1
Irradiation (Hodgkin's disease)	1
Total	18

TABLE 13.5. Cause of death.

Cause	Type of repair	Location
A. Early death:		
1. Sepsis, multisystemic failure	Extraanatomic	Infrarenal
2. Sepsis, multisystemic failure	In situ	Infrarenal
3. Sepsis, multisystemic failure	In situ	Infrarenal
4. Sepsis, multisystemic failure	In situ	Suprarenal
B. Late death:		
1. Renal insufficiency Heart insufficiency	In situ	Axillary artery
2. Sudden death	In situ	Ascending aorta
3. Renal insufficiency Heart insufficiency	In situ	Ascending aorta
4. Bleeding, aorto-duodenal fistula	Extraanatomic	Infrarenal
5. Bleeding, aorto-duodenal fistula	In situ	Infrarenal
6. Systemic candidiasis	Extraanatomic	Infrarenal
7. Unknown	Extraanatomic	Iliacal artery
Total		11

28 patients multiple organisms were cultured. Primary site of infection was known in 24 patients (Table 13.3).

Eighteen patients had immunodeficiency caused by corticosteroid therapy or had chronic disease such as pancreatitis, chronic obstructive pulmonary disease, tuberculosis, renal insufficiency or hematological disorder (Table 13.4).

Results

Four of the 28 patients died during hospital stay and 24 survived. Two of the surviving patients died as a direct result of aneurysm (Table 13.5). Five patients died as a result of unrelated causes. Tables 13.6 and 13.7 summa-

TABLE 13.6. Complications related to in situ repair.

Location	Type of complication	Frank suppuration during operation	Radical debridement
Iliacal artery	Y-graft infection	No	No
Ascending aorta	Dehiscence	Yes	Yes
Infrarenal	Y-graft infection	No	No
Infrarenal	Aortoduodenal fistula	No	No
Infrarenal	Embolization	No	No

TABLE 13.7. Complications related to extraanatomic reconstruction.

Location	Type of complication	No. of patients
Infrarenal	Aortoduodenal fistula	1
Iliacal	Graft thrombosis	1
Infrarenal	Graft thrombosis	3*

*Three times by the same patient

rize complications related to operative procedure. The estimated 1-year and 5-year survival rates for the whole group were 62% and 36%, respectively (Fig. 13.1).

Comment

Medical and operative treatment of patients with mycotic aneurysm differ due to localization of pathologic process and to causative agent. It depends also on a patient's age, general condition and underlying disease. Treatment of mycotic aneurysm is surgical, combined with appropriate pre- and postoperative antibiotic therapy. Classical treatment avoids the use of

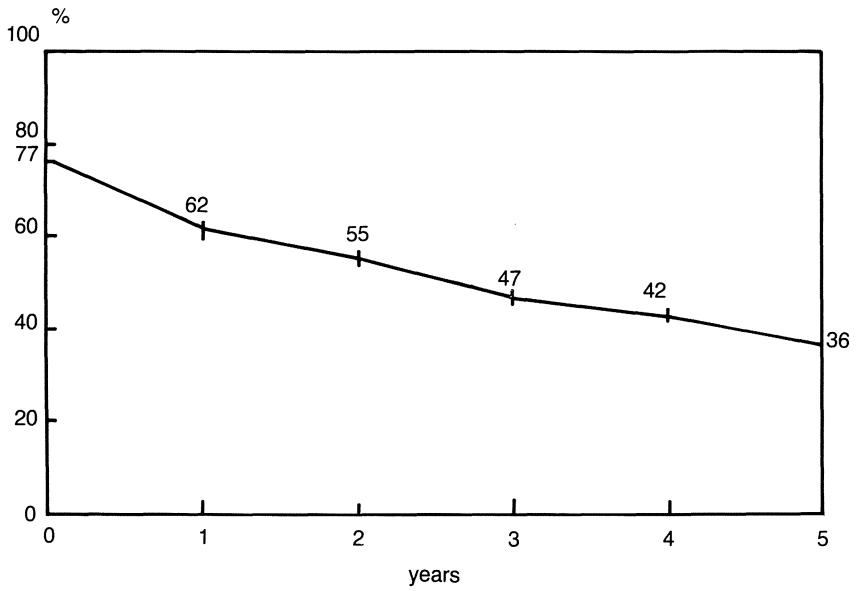


FIGURE 13.1. Actuarial survival of 28 patients with mycotic aneurysms.

bypass procedures in the contaminated region because of the great risk of graft sepsis. Ligation of the involved periphery or visceral artery above and below the aneurysm is effective. If ischemia develops, however, a bypass procedure through uninfected tissue planes should be performed. Treatment of mycotic abdominal aortic aneurysm consists of a two-stage procedure, with primary resection of the aneurysm and the creation of an extraanatomic bypass. Secondary grafting on the aorta from descending thoracic aorta to iliacal arteries could take place later if no signs of infection are present. Treatment by excision and ligation of the extracranial carotid artery may result without complications,[8] but there is a great risk from stroke.[2] In case of poor retrograde flow through the internal carotid artery, it is necessary to restore arterial circulation by end-to-end anastomosis or to bridge the defect in the excised artery by autogenous vein graft.[8] Contrary to conventional treatment by aortic ligation, aneurysmal excision and extraanatomic bypass grafting, several reports suggest that mycotic aortic or external carotid mycotic aneurysm may be successfully treated by in situ grafting.[6,7] Mycotic aneurysm of the ascending aorta and aortic arch should be treated by in situ graft replacement because of the location of the aneurysm. In these regions excision, local debridement and extraanatomic bypass is not possible in most patients.

Aortic aneurysm should be completely excised. Incomplete resection of aortic disease may be the major factor for recurrent infection and subsequent complications.[7] Absence of purulence and negative intraoperative gram stain may decrease danger from infection of in situ bypass.[9] Rupture occurs more frequently and earlier, and survival after surgery is also less common after infection with gram-negative bacilli than for those with gram-positive cocci.[10] In our patients with graft inclusion, it is one possible reason for persistent subfebrile temperature several weeks postoperatively.

Our study revealed that patients had underlying malignant or other chronic disease, or on immunosupressive therapy, exhibited more complications and had shorter life expectancy. Although early results are good, the late results are unsatisfactory. The risk of reinfection, potential rupture and fatal hemorrhage remains great, independent of type of operation. A great number of patients die either from complications of primary disease or from complications of mycotic aneurysm. The risk of complications is great. Optimal surgical therapy and length of antimicrobial therapy is not well established. Recommended length of antibiotic therapy varies from 2 to 6 months postoperatively,[5] or for the rest of the patients' lives.[7] In treating patients with mycotic aneurysm by in situ bypass grafting, it is necessary to continue antibiotic therapy, probably for a lifetime, although this differs from case to case depending on causative agent and general state of the patient. Patients treated for mycotic aneurysm, especially by in situ bypass grafting, should be followed closely and frequently.

Our study shows that in situ repair is the treatment of choice for mycotic

aneurysms located opposite the origin of a major vessel or in close proximity to the heart. Mycotic aneurysm of the extracranial carotid artery should be treated by in situ reconstruction if revascularization is necessary. In situ reconstruction could be undertaken for other localization of mycotic aneurysm by younger patients in good condition. Aggressive local debridement of the aneurysmal wall and infected local tissue is essential to minimize postoperative infection. Patients with prosthetic graft should be treated by lifetime oral antibiotic therapy.

References

1. Leu HJ. Die Aneurysmen der grossen Arterien. Pathologische Gesichtpunkte. Helv Chir Acta. 1986;53:381–386.
2. Borris LC, Nohr M, and Petersen K. Case report. Rapid growth and early rupture of a primary mycotic aneurysm of the abdominal aorta. Eur J Vasc Surg. 1989;3:461–463.
3. Blaisdel FW, and Hall AD. Axillary-femoral artery bypass for lower extremity ischemia. Surgery. 1963;54:563–568.
4. Taylor LM Jr, Deitz DM, McConnell DB et al. Treatment of infected abdominal aneurysm by extraanatomic bypass, aneurysm excision, and drainage. Am J Surg. 1988;155:655–658.
5. Johansen K and Devin J. Mycotic aortic aneurysm. A reappraisal. Arch Surg. 1983;118:583–588.
6. Chan FY, Crawford ES, Coselli JS, Safi HJ, and Williams TE Jr. In situ prosthetic graft replacement for mycotic aneurysm of the aorta. Ann Thorac Surg. 1989;47:193–203.
7. Bitseff EJ, Edwards WH, Mulherin JL Jr, and Keiser AB. Infected abdominal aortic aneurysm. South Med J. 1987;80:309–312.
8. Gossi RJ, Onofrey D, Tvetenstand C, and Blumental J. Mycotic carotid aneurysm. J Vasc Surg. 1987;6:81–83.
9. Brown SC, Busuttil RW, Baker JD, Machteder HI, Moore WS, and Barker WF. Bacteriologic and surgical determinants of survival in patients with mycotic aneurysms. J Vasc Surg. 1984;3:541–547.
10. Jarett F, Darling C, Mundth ED et al. The management of infected arterial aneurysm. J Cardiovasc Surg. 1977;18:361–366.

14
Replacement of Infected Aortic Prosthetic Grafts with Vascularized Musculofascial Flaps

ANITA K. LINDSEY, BRET C. ALLEN, ROBERT S. RHODES, and JOHN A. GRISWOLD

Infected aortic prosthetic grafts are associated with a high rate of morbidity and mortality. In order to examine an alternative to extraanatomic bypass, a canine model of infected aortic Dacron grafts was used to evaluate a vascularized musculofascial pedicle flap fashioned into a cylinder for aortic reconstruction. The flap consists of rectus muscle, posterior rectus fascia and underlying peritoneum, based on the inferior epigastric artery. Infection was established by direct inoculation of the Dacron grafts after insertion, with 1×10^8 CFU of *Staphylococcus aureus*. All dogs had swab, culture, and histologic proof of infection of the prosthetic Dacron grafts within 72 hours prior to construction of the flap. Three of the four dogs ecovered after flap insertion with normal activity and normal neurovascular status. Aortogram demonstrated patency of the flap with excellent arterial runoff in all dogs 24 days after construction. None of the flaps showed evidence of infection. One animal is now seven months after flap construction with no difficulties and is being maintained long-term for histological evaluation of the flap. A vascularized musculofascial pedicle flap can be successfully used for replacement of infected aortic tube grafts without the need for extraanatomic bypass.

Introduction

Since the development of prosthetic vascular grafts in the early 1950s by Voorhees and associates, many complications have been recognized.[1] One of the most serious and frequently life-threatening is infection involving an aortic prosthesis. The incidence ranges from 1 to 6%, with mortality rates from 16 to 90%, and limb loss occurring in approximately 25% of patients.[2] Outcome with infected aortic grafts is strongly influenced by the timing and accuracy of diagnosis as well as the aggressiveness of surgical intervention.

Once the diagnosis of prosthetic graft infection is made, the mainstay of therapy involves excision of the entire infected graft, broad spectrum anti-

biotics, and a reconstructive procedure. Reconstruction usually involves extraanatomic bypass with prosthetic graft material with less than optimal long-term results. In addition, aortic stump disruption remains a threat despite aggressive surgical treatment.

Muscle pedicle flap grafts are widely used for various reconstructive procedures, and the use of autologous tissue in an infected field is obviously far superior to prosthetic material. Studies in 1952 by Cousar and Lam evaluated the use of rectus fascia as a patch for aortic defects or as a short segment fashioned into a tube for aortic replacement.[3] There was a high incidence of graft failure secondary to rupture, thrombosis, and aneurysmal dilatation. In 1987 Coltharp, in this lab, evaluated the use of a vascularized musculofascial graft consisting of the rectus muscle, posterior fascia, and peritoneum for infrarenal aortic graft replacement.[4] He showed this to be highly successful for aortic reconstruction. The success with this autologous tissue reconstruction is the basis for this study using these grafts in an infected field.

Methods

Adult mongrel dogs (fed and housed according to National Institutes of Health guidelines), 20 to 28 kgs, were anesthetized using sodium pentobarbital (40 mg/kg). After sterile betadine preparation of the abdomen, a right paramedian incision was made to avoid injury to the rectus sheath. The retroperitoneum was entered and the aorta dissected free. Branches between the renal vessels and the bifurcation were ligated with 4-0 silk and efforts were made to preserve the inferior mesenteric artery. The animal was then heparinized (100 U/kg) and proximal and distal control of the aorta was obtained. A 1-2 cm segment of aorta was resected, and an 8 mm woven Dacron graft (Meadox) was interposed with anastomoses performed using a 5-0 proline suture (Ethicon) in running fashion (Fig. 14.1). Once patency was established and flow restored, the graft was directly inoculated with 1×10^8 CFU *Staphylococcus aureus* suspended in a 1 cc solution, and the retroperitoneum closed. The bacterial inoculum was prepared by obtaining colonies of *S. aureus* isolated from an infected wound, courtesy of the Microbiology Laboratory at the University of Mississippi Medical Center. These colonies were inoculated in Trypticase Soy broth for 24 hours at 37°C, then washed three times in saline. The solution was then standardized in a spectrophotometer to the desired cell concentration. After inoculation, the abdominal wall was closed in layers with 2-0 and 0 Vicryl suture (Ethicon). No peri- or postoperative antibiotics were used.

After an observation period of 72 hours, the animals were again anesthetized, prepped, and draped, and the abdomen was entered through a vertical midline incision. The retroperitoneum was reopened, cultures were taken around the graft with sterile swabs, and the Dacron prosthesis was

FIGURE 14.1. Completed anastomosis with Dacron graft (8 mm) interposed between transsected ends of the aorta.

dissected free from surrounding tissues. Next, the rectus abdominus flap was constructed beginning with the development of a plane between the anterior fascia and the underlying rectus muscle on the left, away from the previous incision, using scalpel dissection. Dissection continued until the muscle was exposed along the full length of the incision and extending 1 to 2 cm lateral to the lateral border of the rectus. The pedicle flap was then divided superiorly at the level of the costal margin, and the superior epigastric vessels were ligated. Separation continued laterally to create a muscu-

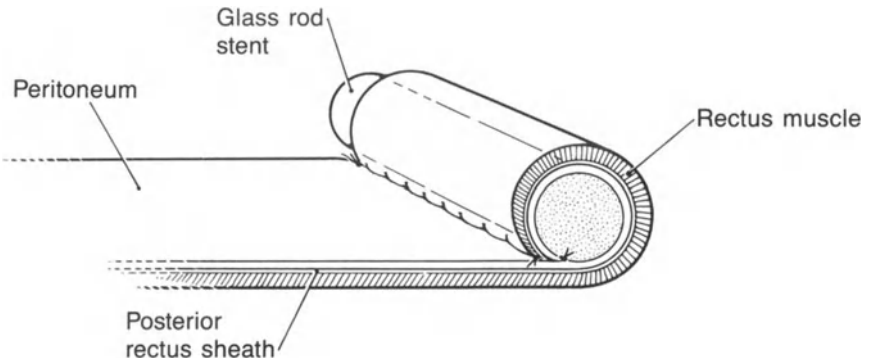


FIGURE 14.2. Illustration showing construction of tube graft with free end wrapped around glass rod stent and sutured to itself in two layers with 5-0 proline suture.

lofascial pedicle flap based on the inferior epigastric vessels. The underlying peritoneum was left attached to the posterior fascia. The superior end of the muscle, distal from the base, was then wrapped around a glass tube stent approximating the size of the aorta. The graft was sewn to itself using a double row of running 5-0 proline suture, resulting in a tube graft lined with peritoneum (Fig. 14.2). The infected prosthetic graft was then excised after heparinization and establishment of proximal and distal control. A longitudinal section through the anastomosis was excised, placed in Trypticase Soy broth for culture, and the remainder of the specimen was placed in formalin for histologic study. The muscle flap was then rotated into the aortic bed, taking care not to allow kinking of the muscle at the abdominal wall with subsequent compromise of blood supply. The flap was then anastomosed to the transsected aorta with running 5-0 proline suture (Fig. 14.3). Blood flow was restored and an aortogram was performed by injection of 20–30 cc Hypaque via a 16-gauge butterfly inserted into the aorta just above the proximal anastomosis. The peritoneal cavity was then irrigated with saline and closed in layers approximating the left anterior fascia and the right anterior and posterior fascial layers. The animals were treated preoperatively with a single dose of Monocid, 500 mg IV (SK&F), and then placed on a 5-day course of Monocid, 500 mg IM q 24 hours.

After an observation period of 24 days, the dogs were again anesthetized and reexplored through the midline incision. The aorta and the muscle flap were exposed. Cultures of the retroperitoneal space were repeated. An aortogram was again obtained to insure flap patency. Proximal and distal control was obtained, and the flap excised in continuity with the anastomoses (Figs. 14.4 and 14.5). A longitudinal segment through one anastomosis was placed in Trypticase Soy broth for culture, with the remaining specimen placed in formalin for histologic study.

Microbiologic studies performed on the graft included plating of the re-

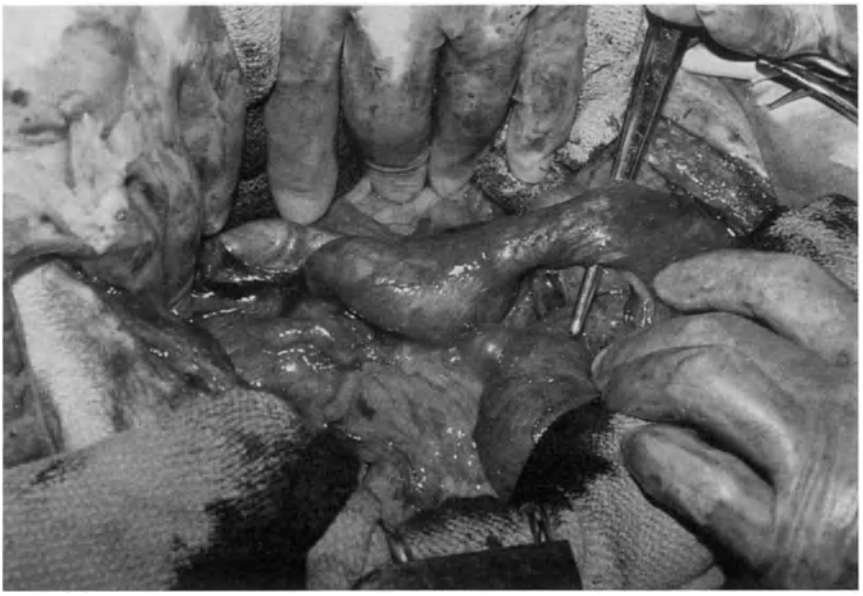


FIGURE 14.3. Completed anastomosis with rectus musculofascial pedicle flap replacing excised infected Dacron graft. Forceps under muscle pedicle illustrating base from anterior abdominal wall.

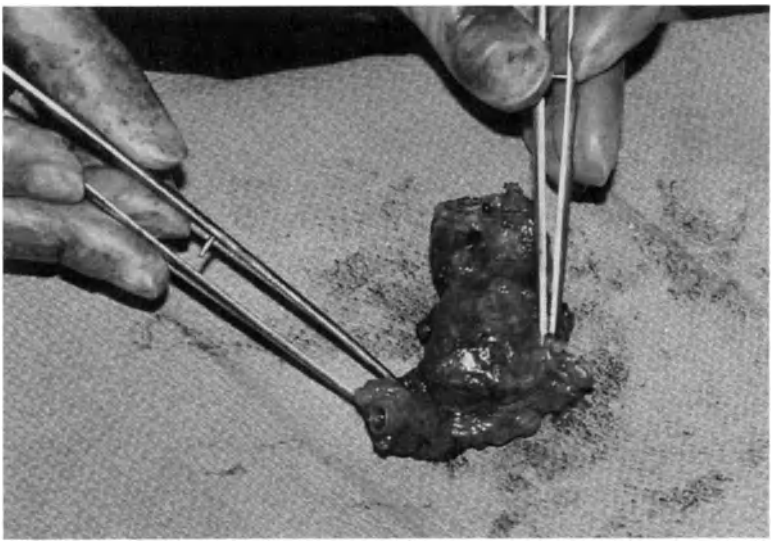


FIGURE 14.4. Muscle flap excised with small segment of native aorta. Forceps pointing to each end.

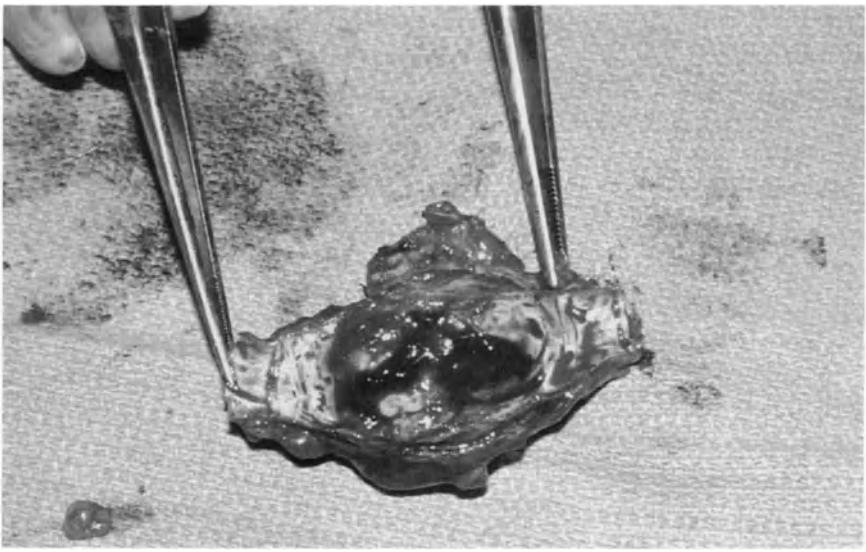


FIGURE 14.5. Muscle flap opened longitudinally.

troperitoneal swabs on Trypticase Soy agar and incubation for 24 to 72 hours. The sections taken through the anastomosis were incubated in Trypticase Soy broth for 24 hours.

Specimens for histologic study were fixed in 10% neutral buffered formalin. Longitudinal sections were taken from each specimen, including one anastomosis. Sections were all stained with hematoxylin and eosin and McCullum-Goodpasture gram stain. On histologic examination, inflammatory cells were identified and quantified subjectively from most to least predominant in each section, as well as overall intensity of the cell infiltration. The specimens were also examined for presence of gram positive cocci.

Results

A total of four adult mongrel dogs had infected aortic prostheses replaced with muscle pedicle flaps. The dosage of 1×10^8 CFU/cc of *Staphylococcus aureus* provided positive cultures of the graft within 72 hours that were consistently reproducible without causing overwhelming peritonitis and/or sepsis. Retroperitoneal cultures were positive in three of four animals. All graft cultures were positive at 72 hours and identified as pure *S. aureus* (Table 14.1).

Survival was 75% (three of four) in animals receiving the muscle flap.

TABLE 14.1. Microbiologic results.

Dog no.	Dosage of inoculum (CFU/cc)	Dacron graft (3 days)		Muscle graft (24 days)	
		Retroperitoneal swab[1]	Graft culture[2]	Retroperitoneal swab[1]	Graft culture[1]
1	1×10^8	+	+	−	−
2	1×10^8	−	+	−	−
3	1×10^8	+	+	−[2]	−[2]
4	1×10^8	+	+	NA[3]	NA[3]

[1]Results of all cultures given as (+) or (−) for *Staphylococcus aureus* after incubation for up to 72 hours.
[2]Autopsy specimen at 6 days.
[3]Not harvested at present—maintained for long-term histopathologic evaluation of the muscle flap.

The three survivors had no neurovascular deficits and in the immediate postoperative period were active and appeared healthy. One animal died six days after placement of the muscle graft. Immediate autopsy was performed and revealed a dissection between the rectus muscle and peritoneum that resulted in graft occlusion. This was felt to be related to technical error in an area of thinned peritoneum. Arteriograms performed on the three survivors showed excellent patency and arterial runoff (Fig. 14.6).

Two of the surviving animals had the musculofascial pedicle flap harvested 24 days following replacement of the infected Dacron graft. One animal is being maintained for long-term histopathologic evaluation of the muscle flap at one year. All cultures of the pedicle flap graft taken at 24 days were negative.

TABLE 14.2. Histopathologic results.

Dog no.	Dacron grafts (3 days)		Muscle grafts (24 days)	
	Predominant inflammatory cell types	Intensity (cellularity) of inflammatory reaction	Predominant inflammatory cell types	Intensity (cellularity) of inflammatory reaction
1	N	Marked	L	Sparse
2	N	Marked	L	Sparse
3	N	Marked	L[1]	Sparse[1]
4	N	Marked	NA[2]	NA[2]

Results of histopathologic study of infected Dacron grafts and musculofascial flaps; N = Neutrophils, L = Lymphocytes.
[1]Autopsy specimens at 6 days.
[2]Not harvested at present.

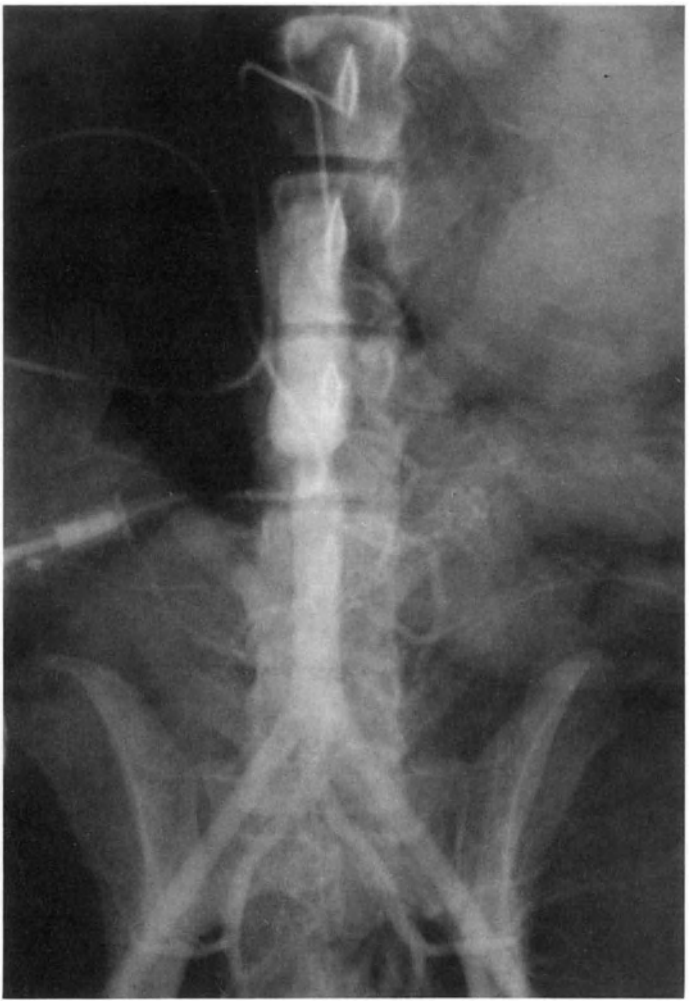


FIGURE 14.6. Aortogram at 24 days after muscle pedicle flap insertion.

The results of histopathologic examination of the infected Dacron grafts and the musculofascial grafts following harvest are shown in Table 14.2. Pathologic studies of the harvested infected Dacron graft revealed an acute inflammatory reaction with marked cellular infiltration, a predominance of neutrophils, and an occasional microabscess (Figs. 14.7 and 14.8). Examination of the excised musculofascial flap revealed viable muscle cells and a sparsity of inflammatory cells, predominantly lymphocytes (Figs. 14.9 and 14.10). No gram positive cocci were identified in any of the musculofascial specimens.

FIGURE 14.7. Histologic section of infected Dacron graft demonstrating marked acute inflammatory reaction.

Discussion

Despite progress made in the early diagnosis of prosthetic aortic graft infections and the realization that aggressive surgical intervention is mandated, the mortality and morbidity of this serious complication remains significant. No synthetic material has been developed to-date that can be

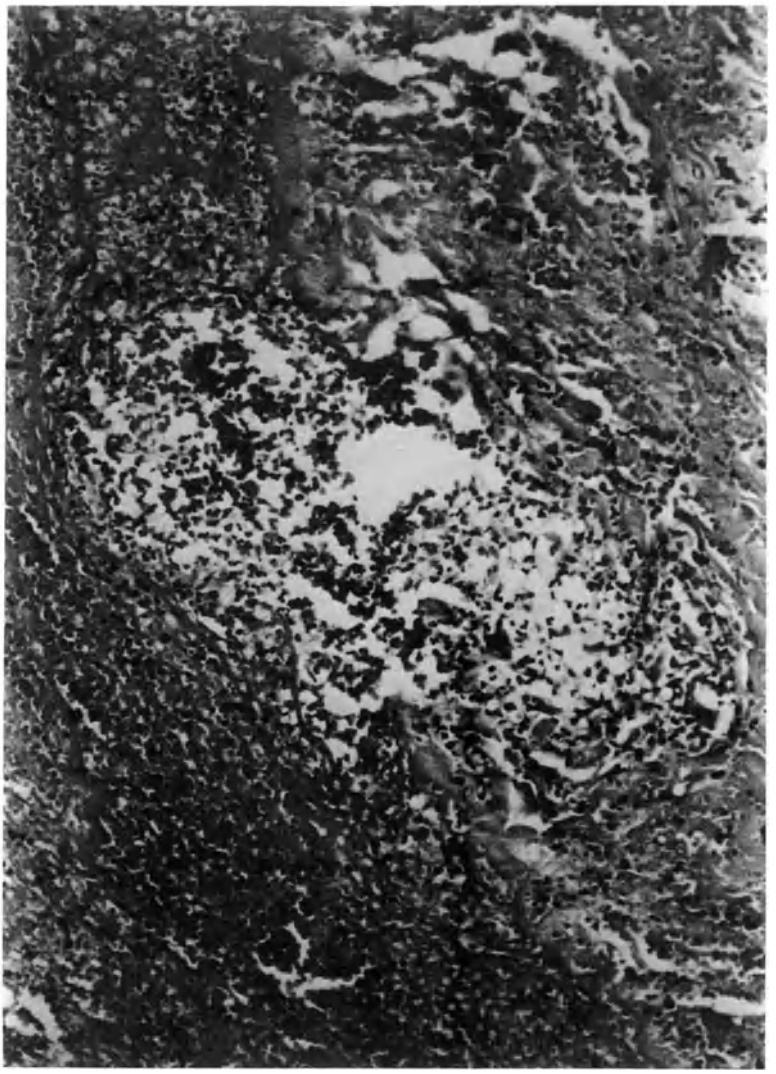
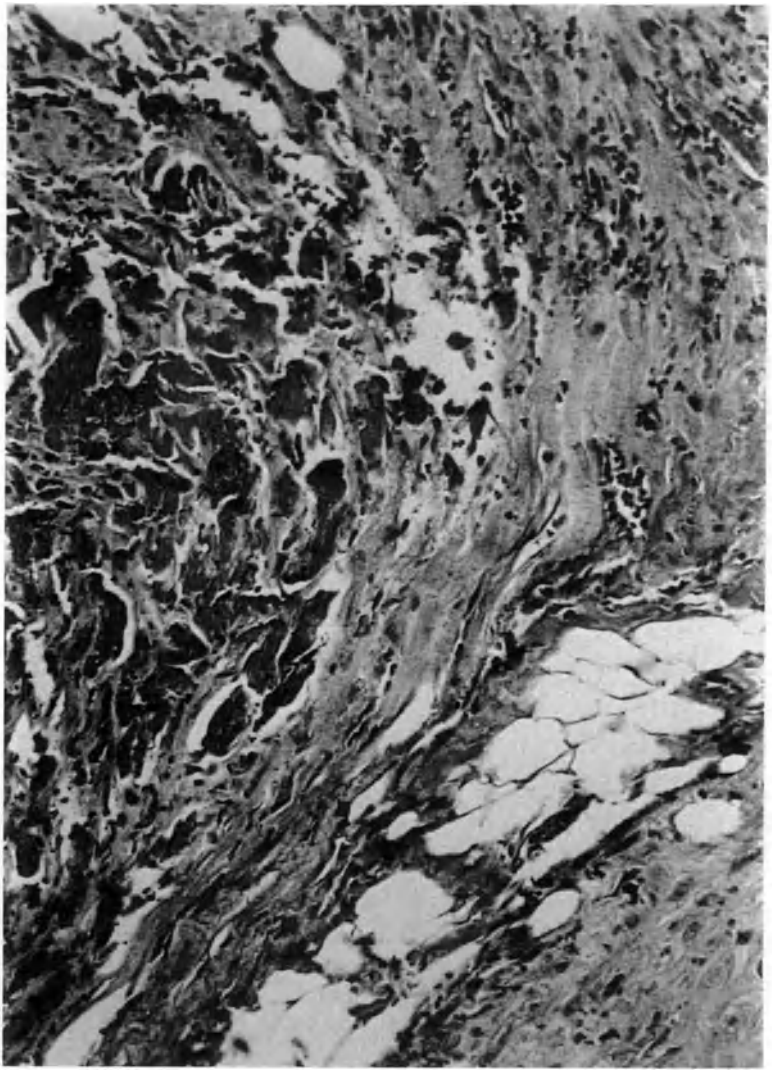


FIGURE 14.8. Histologic section of infected Dacron graft with microabscess.

utilized in an infected field. Extraanatomic bypass, with its less than optimal long-term patency rate, frequently may lead to eventual loss of limb. In addition, when radical surgical removal of all infected prosthetic graft material is performed, the threat of aortic stump disruption remains present.[5]

The use of autogenous material in an infected field has long been recognized as yielding superior results over the almost certain failure of prosthe-



FIGURES 14.9 and 14.10. Histologic sections of muscle flap showing paucity of chronic inflammatory cells.

tic materials. However, in dealing with large vessels, suitable autogenous material has been difficult to construct. The supply of homografts and problems with degenerative changes have slowed major advances toward this end. Previous efforts to evaluate the rectus fascia without attached vascular supply as an aortic replacement met with significant complications of thrombosis, dilatation, and anastomotic disruption.[3,6,7] In 1987 Col-

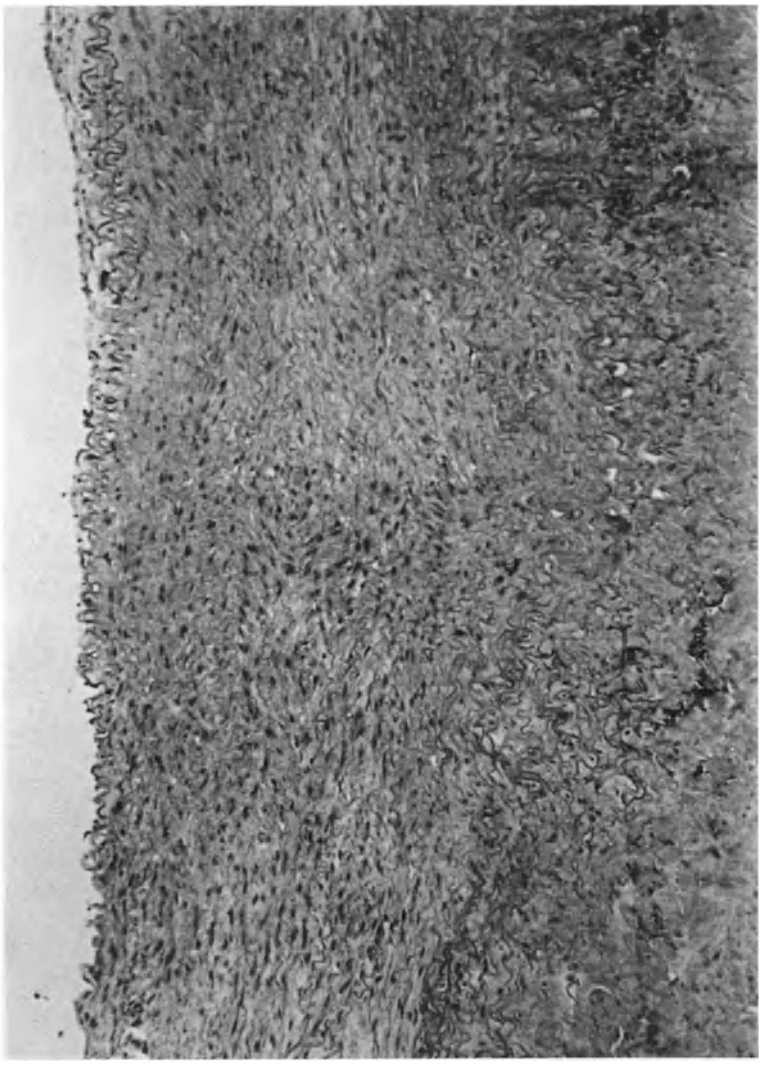


Fig. 14.10

tharp showed that a vascularized pedicle flap, utilizing the rectus fascia and underlying peritoneum based on the inferior epigastric vessels, had marked advantages over its nonvascularized counterpart.[4] Evaluation of the autologous muscle flap in the face of infection, the situation we feel to be most clinically useful, was the basis for this study.

In this study, the bacterial inoculum of 1×10^8 CFU/cc of *Staphylococcus aureus* was consistently able to produce microbiologic and histopathologic

evidence of infection in the Dacron graft three days after inoculation without causing overwhelming sepsis or widespread peritonitis. Examination of the grafts at time of reoperation also revealed clinical evidence of infection with grossly purulent fluid encountered upon opening the retroperitoneum. There was also a marked perigraft inflammatory reaction in the form of fibrinous exudate and adhesions. In contrast, when the abdomen was reexplored 24 days after placement of the muscle flap, no free fluid was encountered. Adhesions were noted from the external muscle layer of the flap to other retroperitoneal structures.

The study reconfirms that the rectus musculofascial flap can be successfully utilized as a tube graft for aortic replacement. However, meticulous surgical technique in the preparation of the flap is essential as evidenced by the single death from technical error. Furthermore, the results indicate that these flaps appear to be quite successful in a setting of infected prosthetic grafts demonstrating no evidence of infection 24 days after construction while maintaining excellent blood flow.

The consistent availability of the flap in most patients and the inherent superiority of autologous vascularized tissue in infected fields makes it useful, not only in infected prosthetic tube grafts, but possibly also in other problem areas, such as buttressing the aortic stump after excision of infected prosthetics to lessen the risk of aortic stump disruption.

References

1. Voorhees AB, Jaretzki A, Blakemore AH. The use of tubes constructed from Vinyon "N" cloth in bridging arterial defects. Ann Surg. 1952;135:332.
2. Bunt TJ. Synthetic vascular graft infections. I: Graft infections. Surgery. 1983;93(6):733–746.
3. Cousar JE, Lam CR. Rectus sheath grafts in vascular repair. Arch Surg. 1952;65:471–476.
4. Coltharp WH. Experimental aortic replacement with a vascularized tissue graft. Arch Surg. 1989;124:1331–1334.
5. Moore WS. Infection in prosthetic vascular grafts, in Rutherford RB (ed): *Vascular Surgery*. Philadelphia, WB Saunders Co, 1984, pp 413–424.
6. Kehne JH, Sensenig DM. Arterial replacement with rectus fascia. Arch Surg. 1956;72:516–519.
7. Holderfer WF, Edwards S, Dowling ER. Autologous rectus fascia as an arterial replacement. Arch Surg. 1968;97:124–327.

15
Is It Worthwhile To Operate on Patients with Infrarenal Abdominal Aortic Aneurysms in China? A Review of 30 Years' Experience at Zhongshan Hospital

YUQI WANG, JIANRONG YE, FUZHEN CHEN, WEIGUO FU, YOUXIAN FENG, and XIULING YAO

During the 30 years between 1960 and 1989, 100 patients with atherosclerotic infrarenal abdominal aortic aneurysms (AAA) were admitted into Zhongshan Hospital. Twenty-eight were treated nonsurgically and 72 surgically. The operation was resection of AAA with prosthesis replacement. The operative mortality was 2.78%. Sixty-two surgical patients are in the postoperative follow-up group. The 5-year survival rate is 83.98% ± 1.71%. Eighteen nonsurgical patients are in the follow-up group. Eight of them died directly from ruptured AAA. The nonsurgical 5-year survival rate is 47.74% ± 12.96%. Ten surgical patients died from ruptured anastomotic false aneurysms. This mortality is higher than what has been reported in the English literature. It may relate to the durability of the prostheses that were inserted. Even so, if all postoperative late deaths from ruptured aorta and other causes are taken into consideration, the 5-year survival rate of the surgical group, 77.51% ± 6.21%, is still higher than that of the nonsurgical group. The data in this paper show that the operation for AAA in this hospital is safe and the postoperative long-term results are comparable to what has been reported in the literature.

It has been routine to treat infrarenal abdominal aortic aneurysms with resection and prosthetic graft replacement since the first operation was done in 1951.[1] There is no accurate epidemiological investigation of the incidence of atherosclerotic abdominal aortic aneurysm in China, but it is evidently lower in this country than in the Western countries. During the last 30 years, 100 cases were admitted into Zhongshan Hospital and about two-thirds of them were treated surgically. The questions that need to be answered are whether the operation is safe and what the long-term results are.

Patients and Methods

All the patients presenting with infrarenal AAA in Zhongshan Hospital from 1960 to 1989 have been reviewed. Surgical management has been resection of the aneurysm and replacement with a synthetic prostheses. In the early days, knitted pure silk and occasionally woven Dacron were used, then knitted blend silk with Dacron, and recently EPTFE and woven Dacron. Perioperative prophylactic antibiotics were used. Mortality and morbidity have been defined as postoperative when death and complication occurred during or subsequent to resection during the initial hospital admission. Long-term survival rate in both surgical and nonsurgical patients available for follow-up at the end of 1989 was achieved by a questionnaire to the patients or by follow-up in the outpatient clinic. Analysis of long-term survival rate was calculated by the life table method.

Results

During the 30-year period, 100 patients with infrarenal abdominal aortic aneurysms were hospitalized at Zhongshan. The patients were from 22 of 30 provinces and municipalities all over China. The pathogenic diagnosis was atherosclerosis for all of the patients. Eighty-seven cases were male, 13 were female, for a male to female ration of 6.69 to 1. The ages ranged from 27 to 76 with a mean age of 59 years. All the patients came for medical consultation because of an abdominal pulsatile mass. Forty-four patients complained of abdominal pain or back pain (44%). Ten complained of claudication, but only 2 had evidence of occlusive vascular disease. In their histories, 73 of them had a history of hypertension (73%), 30 of coronary heart disease (30%), 8 of diabetes mellitus (8%), and 6 of cerebral vascular disease (6%). Thirty-five of 50 recorded cigarette smoking (70%). Thirteen of 63 with records of blood lipids tests had hyperlipidemia (20.63%); 13 of 46 with records of renal function tests revealed elevated creatinine and/or urea nitrogen (28.26%). Four of 47 with records of hepatic function tests showed chronic insufficiency (8.51%). Four of 13 with respiratory function tests had evidence of chronic obstructive pulmonary disease (30.8%) (Table 15.1). B-mode ultrasound examination (68 cases), computerized tomography (15 cases), and angiography (58 cases) confirmed the diagnosis of infrarenal AAA.

Twenty-eight patients, 25 males and 3 females, were treated nonsurgically because of acute myocardial infarct (AMI) (2 cases), stroke (1 case), insufficiency of renal function with the aneurysm just below the renal arteries (1 case), bilateral occluded iliac arteries (1 case), ruptured AAA denying surgery (1 case), and refusing surgery (20 cases). The diameters in all the nonsurgically treated patients were 5 cm or greater with the greatest

TABLE 15.1. Concomitant diseases.

Diseases	No. of patients	%
Hypertenision	73/100	73.0
Coronary heart disease	30/100*	30.0
Diabetes	8/100	8.0
Cerebral vascular disease	6/100	6.0
Hyperlipidemia	13/63	20.63
Renal insufficiency	13/46	28.26
Hepatic insufficiency	4/47	8.51
Chronic obstructive pulmonary disease	4/13	30.80
(Cigarette smoking)	35/50	70.0

*2 with confirmed MI

being 15 cm. Nine of this nonsurgically treated group were followed up only in the first year following admission and 5 were lost to follow-up at the fifth year, making a 64.29% follow-up rate. At the latest follow-up, 1 is alive at the first year postadmission, 1 at the third year, 3 at the fourth year, and 3 at the fifth year. Except for these cases, no one has been followed up over 5 years. The rest of the nonsurgically treated patients either died or were lost to follow-up. Eight of 28 patients died from ruptured AAA with 3 cases during the first year, 2 during the second year, and 1 each during the third, fourth, and fifth years. The incidence of ruptured AAA in this group is 28.57%. One patient died from acute myocardial infarct during aortic angiography, 1 died from stroke during the first year, and 1 from AMI during the third year (Table 15.2). The cumulative survival rate is 86.36% ± 6.84% at the first year, 73.07% ± 9.54% at the second year, 66.12% ± 11.8% at the third year, 58.38% ± 11.99% at the fourth year, and 47.73% ± 12.96% at the fifth year (Fig. 15.1).

Seventy-two of 100 patients underwent elective resection of the AAA and in situ prosthetic graft. There were 64 males and 8 females. Twenty-six of them were under 60 years of age, 35 were ages 60 to 69, and 11 were

TABLE 15.2. Reasons for undertaking nonsurgical treatment.

Reasons	No. of patients	%
AMI	2	7.14
Stroke	1	3.57
Renal insufficiency with AAA near renal arteries	1	3.57
Bilateral occluded iliac arteries	1	3.57
Ruptured AAA denying operation	1	3.57
Denying operation	22	78.57
Total	28	100.00

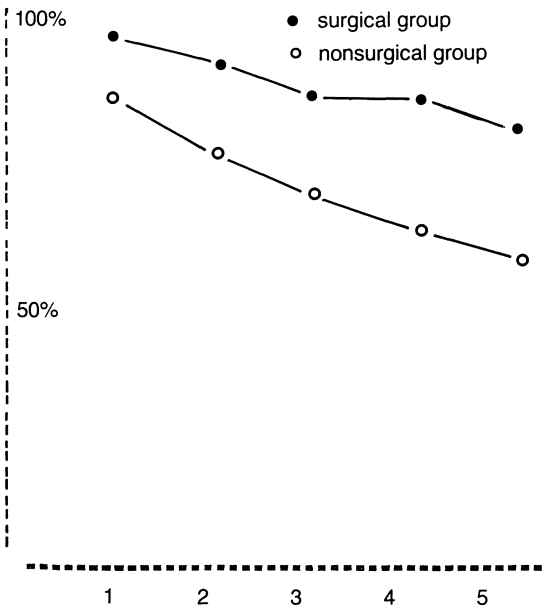


FIGURE 15.1. Five-year survival of two groups. Only deaths from ruptured aorta are taken into consideration.

over 70. The youngest was 27 and the oldest 76. No ruptured AAA was operated on. In 2 cases, the aneurysm diameter was 4 cm. In the remaining 70 cases, the diameter of AAAs were greater than 5 cm with the greatest 13 cm. Knitted pure silk prostheses were used before 1983 in 48 cases. Knitted Dacron was used in 2 cases. Knitted silk and Dacron blend prosthesis was used in 6 cases between 1983 and 1987. EPTFE prosthesis was used in 14 cases, and Gelsoft knitted Dacron was used in 2 cases in 1988 and 1989. Among them, 60 bifurcated and 12 tube grafts were inserted (Table 15.3).

The AAA neck was dissected thoroughly back through the aorta, and the anastomosis was completed using a "two-point" method in 67 cases. The AAA neck was only bilaterally dissected, and the anastomosis was

TABLE 15.3. Prosthesis used.

Material	Period	No. of patients	%
Pure silk	1960–1983	48	66.67
Silk & Dacron blend	1983–1987	6	8.33
Dacron	1960–1987	2	2.78
Gelsoft Dacron	1989	2	2.78
EPTFE (Gore-Tex)	1988–1989	14	19.44

TABLE 15.4. Operative mortality.

Causes of death	No. of patients	%
Hemorrhage from anastomosis	1	1.39
AMI	1	1.39
Total	2	2.78

performed with continued "parachute" method from the back wall of the aorta in the latest 5 cases in 1989. The inferior mesenteric artery was ligated in all of the 72 cases because of its involvement in the aneurysm. In the postoperative stage, the patients who had surgery done between 1960 and 1987 were routinely kept in bed for 3 weeks. From 1988, all of the 17 patients were up and about in 3 to 7 days postoperatively as routine. There was no wound or deep infection. There were 2 operative deaths. One died 2 weeks postoperatively from blood leakage of the upper anastomosis that was not corrected by reoperation. The other died from AMI on the fourth day after the operation. The operative mortality was 2.78% (Table 15.4).

Intraoperative bleeding happened in 2 cases, both occurring from laceration of iliac veins when dissecting the iliac arteries. Postoperative bleeding happened in 3 cases (4.17%), one of which has been mentioned above: the death from blood leakage of the upper anastomosis. One had a blood leakage from the upper anastomosis, and exploration stopped the bleeding by an additional stitch in the suture line of the back anastomotic wall. Two months later, the patient eventually recovered from multiple organ failure. Another experienced bleeding from an unligated lumbar artery; this was ligated by a second laparotomy. Other vascular complications were: thrombosed right branch of the graft in one case (1.39%), which was evacuated by thrombectomy; ischemic left leg in one case; and a femorofemoral vein bypass was done on the second day in one case. The operative vascular morbidity was 9.72%. Other complications were AMI in one case (mentioned above); arrhythmia cordis in one case; bronchopneumonia in 3 cases, one of which was complicated by pulmonary edema; gastric retention in 2 cases; hemorrhage from the upper digestive tract in 1 case; adhesive intestinal obstruction in 3 cases—one had gangrenous intestine resected, one underwent plication of the intestine, and division of adhesion relieved the obstruction in the third; melena in 3 cases; and long-lasting flatulence and constipation in 4 cases. The operative morbidity rate of other diseases was 25%. The overall operative morbidity rate was 34.72% (Table 15.5).

Eleven of 72 surgical patients were lost to follow-up, 2 each at the first, second, third, and fourth years, and 1 each at the eighth, ninth, and eleventh years postoperatively. The rate of follow-up is 84.73%, higher than for nonsurgical patients ($p < 0.001$). One patient had a second opera-

TABLE 15.5. Operative complications.

Complication	No. of patients	%
Intraoperative bleeding	2	2.78
Postoperative bleeding	3	4.17
Thrombosed graft branch	1	1.39
Ischemic foot	1	1.39
Total vascular complications	7	9.72
AMI	1	1.39
Arrhythmia	1	1.39
Bronchopneumonia	3	4.17
Gastric retention	2	2.78
Hemorrhage upper digestive tract	1	1.39
Adhesive bowel obstruction	3	4.17
Flatulence and constipation	4	5.56
Melena	3	4.17
Total other complications	18	25.00
Total operative complications	25	34.72

tion for anastomotic false aneurysm 3 years and 3 months after the first operation. Another patient had a second operation for a proximate infrarenal aortic aneurysm just above the previous graft 2 years and 4 months after the first operation. Eight patients died from ruptured aorta or "graft failure" during follow-up, diagnosed by the symptoms of sudden deaths given by the patients' doctors or relatives. Two died at the second year, 3 at the third year, and 1 each at the fifth, sixth, and eighth years. Rupture was considered to have happened in an anastomotic false aneurysm or in the graft. Six of the 8 could not get to a hospital. The incidence of ruptured aorta in the surgical group is 11.11%, significantly lower than that of the nonsurgical group ($p < 0.05$). Eleven surgical patients died from other diseases, i.e., 2 from cardiac problems, 2 from stroke, 1 from hemorrhage of the digestive tract, 1 from obstruction of the intestine, 1 from carcinoma of the cardia, 1 from "high fever," 1 from complications of a gangrenous foot, and 2 from unknown causes. The mortality of other diseases in the two groups were about the same ($p > 0.05$) (Table 15.6).

Except for the cases lost to follow-up, 56 patients lived over 1 year, 29 lived over 5 years, 8 lived over 10 years, and 3 lived over 15 years. The patient who has the longest life postoperatively has entered the 20th year and is 78 years in age. The postoperative survival rate is $96.92\% + 2.13\%$ at the first year, $93.20\% + 1.04\%$ at the second year, $86.69\% + 1.53\%$ at the third and fourth years, $83.98\% + 1.71\%$ at the fifth year, $80.93\% + 1.90\%$ at the sixth and seventh years, $76.43\% + 2.31\%$ at the eighth year and beyond (Figure 15.1).

The surgical group survival rate in each year is significantly higher than that of the nonsurgical group, namely, $p < 0.05$ at the first year, $p < 0.01$

TABLE 15.6. Causes of late deaths.

Causes	Nonsurgical	Surgical
Cardiac	2	2
Stroke	1	2
Obstructive intestine	0	1
Carcinoma	0	1
"Gangrenous foot"	0	1
"Fever"	0	1
Hemorrhage of digestive tract	0	1
Unknown	0	2
Total	3 (10.71%)	11 (15.28%, $p > 0.05$)
Rupture of aorta or graft failure	8 (28.57%)	8 (11.11%, $p < 0.01$)

from the second to the sixth years. If all deaths from ruptured aorta and other causes are taken into consideration, the survival rate of the surgical and nonsurgical groups are, respectively, $95.42\% \pm 2.58\%$ and $78.26\% \pm 8.59\%$ at the first year, $88.22\% \pm 4.61\%$ and $66.22\% \pm 10.69\%$ at the second year, $82.06\% \pm 5.49\%$ and $54.18\% \pm 11.65\%$ at the third year, $82.06\% \pm 5.49\%$ and $48.16\% \pm 11.90\%$ at the fourth year, and $77.51\% \pm 6.21\%$ and $41.28\% \pm 12.57\%$ at the fifth year (Figure 15.2).

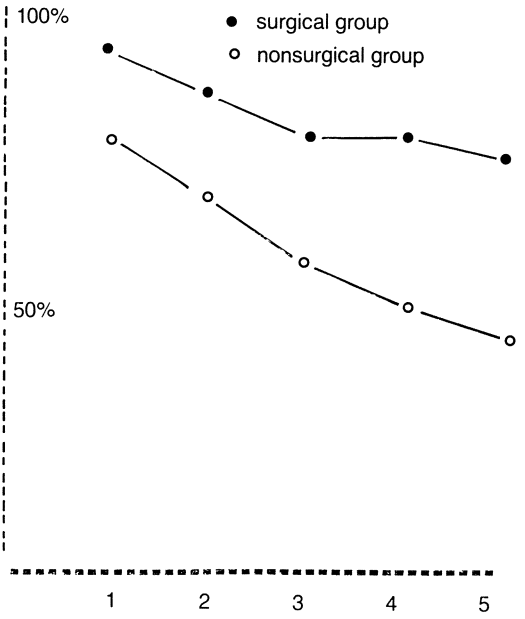


FIGURE 15.2. Five-year survival of two groups. All deaths are taken into consideration.

Discussion

AAA patient numbers are increasing in Western countries.[2] An increasing rate has also been encountered at Zhongshan Hospital, Shanghai, China. There were 100 cases of infrarenal AAA during the last 30 years at this hospital, but there were 25 cases, of which 20 were treated surgically, during just the last 3 years. That is 25% of the total cases in only the last 10% of the time period. It is time to analyze the safety of the operation, the long-term results, and any related subjects.

All the cases with atherosclerotic suprarenal AAA or thoracoabdominal aortic aneurysms, AAAs from Marfan's disease, mycotic AAA, and traumatic AAA, etc., have been excluded from the present data. Only atherosclerotic infrarenal AAAs are reported. Moreover, the reasonable overall follow-up rate of 79% and the follow-up rates of 84.73% and 64.29% in surgical and nonsurgical groups, respectively, make comparisons possible and relatively reliable.

The mean age of our patients is about 10 years younger than that of patients in the literature. This may reflect the difference in life expectancies. The mean age of patients during the last 2 years (1988 and 1989) was 67, older than the mean age of 59 for the whole group, as also life expectancy has increased over the last 3 decades.

The incidence of concomitant diseases is about the same as in the literature. One obvious difference from the data in the English literature is that there was no ruptured infrarenal AAA operated on in this group. The operative mortality for elective AAA varies from unit to unit during the last 20 years, ranging from 0 to 11% in British teaching hospitals and in major vascular units of the USA.[3] In a 2-year prospective study conducted between 1985 and 1987 in Oxford, the operative mortality for elective aneurysmal surgery was 1.4%.[3] In another prospective survey conducted in Toronto and Ottawa, the mortality was 4.8%.[4] These results of prospective studies may represent the current level of surgical treatment of AAA. Therefore, we could say that an operative mortality rate of 2.78% in the present data is acceptable, or that the operation for AAA is safe at Zhongshan Hospital. It should be pointed out that one patient died from anastomotic hemorrhage 2 weeks postoperatively. This patient had an embolized left dorsalis pedis on the third day after resection of AAA and severe back pain and vomiting at the same time. These were the signs of postperitoneal hemorrhage. If the exploration had been performed at this time, rather than 10 days later, the patient would have been saved. In fact, prompt exploration of the operative field found the causes of bleeding and saved 2 lives in this group. It is definitely wrong to have unnecessary "investigation" or just to wait for the appearance of hemorrhagic shock.

Other operative complications were not as often reported in the literature.[5] Among them, pulmonary complication was dominant as well as vascular complications. Seventy percent of patients were smokers and

30.8% had obstructive pulmonary disease, making them liable to have postoperative hypoxia. The key to protect from this complication is keeping the respiratory tract patent. We found that leaving a epidural catheter for adding analgesic and using an ultrasonic atomizer was very effective in helping patients cough up phlegm. Flatulence, melena or fresh blood in the feces might be from ischemic colitis because of ligation of the inferior mesenteric arteries. But no case needed colectomy for relieving these symptoms. Though 13 of 46 had evidence of renal function damage preoperatively, no renal failure occurred postoperatively except in the case of multiple organ failure. This good result might be because no suprarenal aortic clamping was used during surgery. Cardiac complication was not often seen but could be fatal in this group. Adequate prophylactic antibiotics ensured the success of surgery without infection. Antibiotics were used for more than 10 days in most patients in the surgical group. But this sort of irrational prolonged administration should be avoided. Now we just give the patients antibiotics intravenously twice before and after surgery. We feel that early extraction of the gastric tube and the catheter in 2 to 3 days, or even eliminating intubation, as well as early ambulation all help patients recover sooner.

Long-term results of surgical treatment of our patients is comparable to that reported in the literature,[6–8] even if the deaths from other causes are included in the postoperative deaths. As control, the follow-up of the nonsurgical group showed a much lower five-year life expectancy than the surgical group did. But it still seems higher than that reported in the literature. Before surgical procedure of resection and prosthesis replacement for AAA was put into practice, Estes[9] showed that the 5-year survival rate following diagnosis was only 18.9%. Clinical investigations after his study showed quite similar results.[10,11] It should be noted that the relatively high 5-year life expectancy of our nonsurgical group may have to do with the loss of 9 patients from follow-up during the first year after admission. Some of them might have died from ruptured AAA, but their families didn't answer the questionnaires. This could make the mortality lower than the actual mortality rate. In contrast, significantly fewer were lost from postoperative follow-up in the surgical group ($p < 0.05$). As a result, the surgical group rupture rate is a better reflection of the truth, and the rupture rate would be even higher in the nonsurgical group than in that of the surgical group. This shows that, from the point of view of long-term results, surgical treatment for AAA is the better choice to avoid rupture of the aorta.

There is no doubt that most vascular surgeons have accepted the principle that AAA should be resected when its diameter is over 5 cm.[12] In fact, AAA with a diameter of 4 cm or less did rupture at the rate of 9.5% to 20% per year.[13–15] This means that AAA with a diameter of 4 cm or less should not be excluded from surgery. In other words, even small, asymptomatic AAA should be operated on unless the concomitant diseases are

very serious. We did see a few small AAAs with very thin front walls rupturing during surgery. No factor seems to predict the natural history for the individual patient.[10] If these patients are not operated on for some reason when diagnosis of small AAA is made, regular follow-up with B-mode ultrasound or CT scan can be undertaken.

By follow-up, 11 nonsurgical and 8 surgical patients died from ruptured aneurysms or false aneurysms. The term "rupture of the aorta" can cover both these pathological situations. Sixteen of these 19 patients lived in different provinces far away from Shanghai, with the farthest 3,000 km away. It was impossible to come to Shanghai by train or ambulance to undergo an emergency operation for ruptured AAA. In addition to poor transportation, the lack of vascular surgical service available in those areas where the patients lived is another hindrance to prompt treatment. Two cases of ruptured anastomotic false aneurysms did reach the hospital. Unfortunately, no active surgical procedure was taken to save their lives because of the presumed operative difficulties and risks. It seems important that we have trained vascular surgeons here, and that both doctors and patients' families adopt a more aggressive approach in electing surgery to save the lives of dying patients.

Pure silk prostheses are exclusively used in China.[16] Except for 2 operative deaths, 8 patients who suddenly died from ruptured aorta had pure silk prostheses. There was no late aortic rupture in 24 cases with Dacron, blended silk and Dacron, Gel-seal Dacron, or PTFE. However, the results are not statistically comparable at the present time because half of the patients with material other than silk are still in the first year. Meanwhile,

TABLE 15.7. Results of treatment of non-ruptured AAA.

Authors	Year	Operative mortality %	Surgical 5-yr survival %	Nonsurgical 5-yr survival %
Estes [9]	1950	–	–	19
Schatz [19]	1962	–	–	36
DeBakey [6]	1964	9	58	–
Levy [18]	1966	11–35	34	–
Szilagyi [17]	1966	6.1–39.1	55.2	24.0
Fielding [7]	1981	8.1	68.1	–
Crawford [9]	1981	4.76	63	–
Johnston [4]	1988	4.8	–	–
Collin [3]	1989	1.2–4.2	–	–
Cronenwett [12]	1990	–		35
Johansson [20]	1990	7.5	70	40*, 15**
Present data		2.78	77.51	41.28

* diameter of AAA 5 cm
** diameter of AAA 5 cm
all deaths are included

all 48 cases with pure silk are past 3 years postoperatively. In a retrospective study, Sieswerda et al. found incidence of 2.6% and 12.3% of anastomotic aneurysms at the aorta and iliac artery, respectively, after aortoiliac or iliofemoral reconstruction. Szilagyi et al.[17] in an early series of surgical treatment of AAA, had 10 cases of "graft failure" in 434 postoperative cases (2.3%). In a study of 100 operated cases of AAA, Levy[18] found 4 deaths from hemorrhage due to disruption of the anastomotic suture line. The incidence of anastomotic aneurysms, or "graft failure," in the present group is obviously higher than that reported in the literature. The higher incidence is likely to be related to the durability of pure silk. Pure silk aortic prostheses played an historic role in the development of vascular surgery in Zhongshan Hospital and in China. Now it needs to be reevaluated.

As time goes on, both short- and long-term results of AAA resection are getting better (Table 15.7). We hope that our knowledge and technique will quickly catch up with the advances in other parts of the world.

References

1. Dubost C, Allary M, Oeconomos N. Resection of an aneurysm of the abdominal aorta: Re-establishment of the continuity by a preserved arterial graft with result after five months. Arch Surg. 1952, 64:405–408.
2. Collin J. Ruptured aortic aneurysms: The coming storm. Br J Surg. 1987, 74:332.
3. Collin J, Murie J, Morris PJ. Two year prospective analysis of the Oxford experience with surgical treatment of abdominal aortic aneurysm. Surg Gynecol Obstet. 1989, 169:527–531.
4. Johnston KW, Scobie TK. Multicenter prospective study of nonruptured abdominal aortic aneurysms: I. Population and operative management. J Vasc Surg. 1988, 7:69–81.
5. Johnston KW. Multicenter prospective study of nonruptured abdominal aortic aneurysms: II. Variables predicting morbidity and mortality. J Vasc Surg. 1989, 9:437–447.
6. DeBakey ME, Crawford ES, Cooly D, Morris GC Jr, Royster TS. Aneurysm of abdominal aorta: Analysis of results of graft replacement therapy one to eleven years after operation. Ann Surg. 1964, 160:622–639.
7. Fielding JWL, Ashton F, Slany G, Campbell DJ. Diagnosis and management of 528 abdominal aortic aneurysms. Br Med J. 1981, 283:355–359.
8. Evans WE, Hayes JP. Tube graft replacement of abdominal aortic aneurysm. Amer J Surg. 1988, 156:119–121.
9. Estes JL. Abdominal aortic aneurysms: A study of 102 cases. Circulation 1950, 2:258–263.
10. Jensen EB, Vestersgaard-Anderson T. The natural history of abdominal aortic aneurysm. Eur J Vasc Surg. 1989, 3:135–139.
11. Crawford ES, Saleh SA, Babb JW III, Glaeser DH, Vaccaro PS, Silvers A. Infrarenal abdominal aortic aneurysm: Factors influencing survival after operation performed over a 25 year period. Ann Surg. 1981, 193:699–709.

12. Cronenwett JL, Sargent SK, Wall MH, Hawkes ML, Freeman DH, Dain BJ, Cure JK, Walsh DB, Zwolak RM, McDaniel MD, Schneider JR. Variables that affect the expansion rate and outcome of small abdominal aortic aneurysms. J Vasc Surg. 1990, 11:260–269.
13. Darling RC, Messina CR, Brewster DC, Ottinger LW. Autopsy study of unoperated abdominal aortic aneurysms. Cardiovasc Surg. 1976, 56(Suppl 2 Circ):161–164.
14. Szilagyi DE, Elliott JP, Smith RF. Clinical fate of the patient with asymptomatic abdominal aortic aneurysm and unfit for surgical treatment. Arch Surg. 1972, 104:600–606.
15. Cronenwett JL, Murphy TF, Zelenock GB. Actuarial analysis of variables associated with ruptured small abdominal aortic aneurysms. Surg. 1985, 98:472–483.
16. Feng Y, Zheng J, Li J, Shi Q. Clinical application of pure silk vascular prostheses. Chi Med J. 1982, 95:565–568.
17. Szilagyi DE, Smith RF, DeRusso FJ, Elliott FJ, Sherrin FW. Contribution of abdominal aortic aneurysmectomy to prolongation of life. Ann Surg. 1966, 164:678–699.
18. Levy JF, Kouhoukos NT, Walker WB, Butcher Jr HR. Abdominal aortic aneurysmectomy. Arch Surg. 1966, 92:498–503.
19. Schatz IJ, Fairbairn JF, Juergens JL. Abdominal aortic aneurysms: A reappraisal. Circulation 1962, 26:200–205.
20. Johansson G, Nydahl S, Olofsson P, Swedenborg J. Survival in patients with abdominal aortic aneurysms: Comparison between operative and nonoperative management. Eur J Vasc Surg. 1990 4:497–502.

16
Patency Improvement and Neointimal Inhibition of Vena Caval Dacron Prosthesis by Endothelial Cells Sodding in Canine

GUANGDI LI, ZHONGGAO WANG, JIDONG WU, WEI DU, JIAN YU, DAJIE WANG, LIQUN PU, and HAI ZHANG

Thirty-seven infrarenal vena caval (IVC) and 10 carotid arterial knitted dacron grafts sodded with microendothelial cells (MECs) were investigated in canines. MECs were procured from omentum by enzymatic digestion through a special process of density gradient extraction with lymphocyte separation medium. A total of $1.32 \pm 0.39 \times 10^6$ MECs/g of omentum was obtained. The grafts were sodded with MECs (a cell density of greater than $10^5/cm^2$ surface of graft) by a preclotting technique. The control grafts received a sham sodding with only autogenous plasma. Three experimental groups were designed.

In Group I (GI) animals, a 6 cm length of IVC was replaced by Dacron graft of the same dimension and an additional distal arterial-venous fistula (AVF) was created and functioned for one week. In Group II (GII), a 4 cm length graft was inserted by the same method except without distal AVF supplementation. In Group III (GIII), bilateral carotid arteries were partially replaced with 6 cm length Dacron grafts. The grafts were explanted at 7 days and 100 days in GI, at 7, 14, and 60 days in GII, and all at 7 days in GIII. All sodded grafts and 2 of 9 controls harvested at 100 days in GI were patent ($p < 0.001$). Four of 5 sodded grafts and none of 5 controls explanted at 60 days in GII were patent ($p < 0.05$). In GIII, both the sodded and control grafts were patent. Light microscopy revealed that the thickness of neointima in the sodded GI grafts was significantly thinner than that in the control GI grafts ($p < 0.001$) and in the GII grafts as well ($p < 0.01$). Scanning electron microscopy demonstrated that confluent endothelial cells appeared on the inner surface of the sodded implants beginning at 7 days in GI and 14 days in GII. No MEC lining was detected in GIII grafts. The production of extrinsic arachidonic acid, 6-keto PGF_1-alpha, metabolized by sodded cells in the 100-day-old grafts was much greater than that metabolized by cells of spontaneous endothelialization in the control ($p < 0.001$) and vice versa for the Thromboxane B_2 ($P < 0.02$). It is con-

cluded that MEC sodding significantly improves the patency rate and inhibits neointimal thickening of the vena caval knitted Dacron prosthesis, presumably through a process of rapid endothelialization on the sodded grafts. A temporary distal AVF promotes the maturation process of the endothelial lining and contributes to the inhibition of neointimal hyperplasia of implants. Successful MEC sodding is revealed only in the venous system in this study.

A significant limitation of the seeding technique in the venous system is the time necessary for seeded ECs to adhere and proliferate properly and eventually cover the entire graft surface. The high density seeding technique, the sodding technique, was initiated in the hope of finding a stringent model for studying methods of venous grafting leading to better, more rapid endothelialization of the prosthesis.

Key Words

Inferior vena cava, endothelial cells, sodding, Dacron prosthesis, arachidonic acid, thromboxane A_2 (TXA_2) and B_2 (TXB_2), prostaglandin (PGI_2 and PGF_1).

Introduction

Previous studies have shown an increasing tendency in the patency rate of endothelial cell (EC) seeding in arterial grafts compared with EC nonseeding ones.[1–5] However, recent studies found that these results were not as favorable in the seeding grafts implanted in the venous circuit.[6,7] The slow and erratic venous flow occurring in a thrombogenic conduit leads almost invariably to an undesirable early thrombosis in venous grafts. One significant limitation of the seeding technique used in the venous system is the time lag necessary for the seeded ECs to stick firmly, expand smoothly, proliferate properly, and eventually cover the entire graft surface. The high density seeding technique, i.e., to seed more than 10^5 ECs per cm^2 of graft surface, or the so-called "sodding" technique, was initiated[7–9] in the hope of finding a better, more rapid endothelialization of the prosthesis.

Our study has demonstrated that confluent endothelialization of the prosthesis could be achieved within 5 to 10 days after implantation into the IVC of dogs by sodding of microvascular endothelial cells (MECs) derived from omentum.[10] More work needs to be done, however, to confirm the feasibility of this approach for clinical situations. This study was designed to evaluate the possibility of increasing patency and inhibiting neointimal hyperplasia of the venous prosthesis by the MEC sodding technique and to observe morphologically and functionally the newly-formed cell lining on the neointimal surface.

TABLE 16.1. Experimental design.

Group	approaches	Graft dimension (cm)*	Explantation days**			
			7	14	60	100
I	IVC implantation with distal AVF	1.0/6.0 ± 0.5	3/0	—	—	9/9
II	IVC implantation only	1.0/4.0 ± 0.3	3.0	3.0	5.5	—
III	Bilateral carotid implantation	0.4/6.0 ± 0.5	5/5	—	—	—

*Diameter/length.
**Sodding/control.

Materials and Methods

Animal Subjects and Preoperative Care

Forty-two adult male mongrel dogs weighing 17.5 kg to 23 kg were cared for in accordance with the guidelines of the "Principles of Laboratory Animal Care" and the "Guide for the Care and Use of Laboratory Animals" (1983 revision of NIH publication No. 8023). Aspirin, 300 mg, and dipyridamole, 50 mg, were given once daily to all animals beginning three days preoperatively and continued for seven days postoperatively.

Experimental Design

The animals in this study were allocated to 3 groups (Table 16.1).

Isolation of Microendothelial Cells

MECs were isolated according to procedures we used previously.[5] The dogs were anesthetized with intraabdominal sodium pentobarbital (30 mg/kg). The omental tissue was collected through an upper midline abdominal incision and rinsed in normal saline with heparin (0.1 mg/ml). Ten grams of omentum were cut into pieces about 2 mm × 2 mm × 2 mm, which were then placed into 10 ml of 2.4 mg/ml collagenase (Type 1, Sigma Chemical Co., St. Louis, MO, USA) in calcium- and magnesium-free Hanks balanced saline solution (HBSS-CMF) and incubated in a 37°C water bath with intermittent gentle agitation for 35 minutes. The specimen was then centrifuged at 1000 g for 5 minutes at 22°C to remove adipocytes. The pellet was resuspended in HBSS-CMF at 22°C and filtrated successively through a nylon mesh of 500 microns pore size and a stainless steel mesh of 54 microns pore size so as to remove undigested tissue debris. The filtrated

fluid was layered into a centrifugation tube on top of the centrifugation solution with a specific density of 1.065, which was made by mixing a polysucrose lymphocyte separation medium, a density of 1.077 ± 0.002, (Shanghai Second Reagent Factory, Shanghai, China) with HBSS-CMF in a 1 to 0.2 volume ratio. After centrifugation at 200 g for 5 minutes at 22°C, a milky-white cellular layer was formed at the top of the separation medium, and it was then aspirated and washed in the HBSS-CMF and centrifuged at 200 g for 5 minutes. The collected cells were resuspended in 2 ml autologous plasma made from 3.8% sodium citrate anticoagulated whole blood (1:9) after centrifugation at 500 g for 10 minutes.

Graft Sodding

A modified four-staged preclot method was used before implantation.[11] Twice the graft was preclotted with a mixture of 0.4 ml of autologous plasma and 0.05 ml of 2.5% calcium chloride from the external wall of graft. Then 0.6 ml of the EC suspension in plasma, mixed with 2.5% calcium chloride solution, was instilled into the graft's luminal surface, which was constantly rotated and intermittently stretched. Twice the same process was repeated with another 0.6 ml aliquot of EC suspension in plasma. The control graft was treated in an identical fashion except no ECs were employed. The Meadox knitted double-velour Microvel prosthesis was the only graft material used in this study.

Operative Procedure

In the venous groups (GI, GII), the IVC was isolated from the renal vein level to its bifurcation. Following systemic heparinization intravenously (1 mg/kg), a segment of 6 cm (GI) or 4 cm (GII) of IVC was resected and replaced with an equal dimension Dacron prosthesis. The proximal and distal anastomoses were completed with continuous 6-0 Prolene everting sutures in an end-to-end manner. An additional 1 cm length side-to-side femoral AVF was then constructed in the right thigh and closed 7 days following surgery in GI animals.

In the arterial group (GIII), through a midline cervical incision, both carotid arteries were exposed; 6 cm length segments were resected and replaced by similar length Dacron prostheses, of which the left grafts were sodded with MECs and the opposite ones were only clotted with autologous plasma.

Postoperative Observation and Explanation

All animals in the IVC groups were examined by B-Mode ultrasonography for collaterals on the abdominal wall to evaluate the patency of the prostheses. When occlusion of the graft was suspected from the ultrasonog-

raphy or at the scheduled explantation time, the dogs were sacrificed and the grafts, together with 3 cm length adjacent IVC segments, were removed in Groups I and II.

Evaluation of Specimens

Immediately following their removal, the grafts were flushed with normal saline, opened longitudinally, and photographed. Neointimal samples were taken from the midportions of the prostheses and fixed in 2.5% buffered glutaraldehyde for 2 hours or longer. After fixation in 1% aqueous osmium tetroxide, the tissue samples were dehydrated in graded ethanol and then flatly embedded in the epoxy resin (Epon 812) routinely. Sections 2 microns thick were prepared and stained with hematoxylin-eosin for light microscopic evaluation. The thickness of neointima was measured morphometrically. The best-oriented blocks were further trimmed for ultrathin sections. These sections were doubly stained with uranyl acetate and lead citrate for transmission electron microscopic examination. Other specimens prepared by the same graded dehydration process in acetone were dried at the critical point and were then coated with a gold-palladium target for scanning electron microscopic investigation.

Fluorescence Microscopic Examination

The cells collected from 0.2 ml harvested suspension were resuspended in Medium 199 (Life Technologies Inc., Grand Island, NY, USA) with 20% fetal calf serum and incubated at an atmosphere of 37°C at 95% air humidity and 5% carbon dioxide for 24 hours. The cells were then examined by phase contrast light microscopy. Cultured cells of a 4 mm^2 area of the neointima, taken from each patent midgraft, were fixed with 0.12 Mol polyformaldehyde for 24 hours. Frozen sections 5 microns thick were made from each graft sample. The thawed sections and the cultured cell specimens were reacted with indirect immunofluorescence staining for Factor VIII-related antigen as described by Watkins et al.[12]

Radioimmunoassay

Segments larger than 1 cm^2, taken from the middle part of patent grafts together with the distal parts of the IVC in GI animals, were placed in two specially designed chambers. The chamber surface fixed an incubating vial composed of a metal cylinder and a base connected to each other with a screw device. The vial, which contained exactly 1 cm^2 of surface, was designed and made in our laboratory for use in measuring arachidonic acid metabolites. The samples were placed between the metal cylinder and the base while the screw device was loosened. The samples were then stretched on the original surface by traction from different directions to flatten them,

while the screw device was fully tightened to a water-seal, forming real vials. After the vials were rinsed with Hanks Solution three times, one ml of Medium 199 containing 20 micron Mol of arachidonic acid (Sigma Chemical Co., St. Louis, USA) was added to the chambers, which were then placed in the cell culture incubator at 37°C for 20 minutes. The medium was repeatedly bubbled and then collected into ampullae that were stored in liquid nitrogen. Metabolites of PGI_2 and TXA_2, 6 Keto-PGF_1 Alpha, and TXB_2 were assayed by a tritiated radioimmunoassay technique.[13]

Results

Cell Harvesting

An average harvesting of MECs was $1.32 \pm 0.39 \times 10^6$/g of omentum. The MEC seeding density was $7.0 \pm 2.1 \times 10^5/cm^2$ of the graft surface, $10.5 \pm 2.5 \times 10^5/cm^2$, and $17.5 \pm 2.5 \times 10^5/cm^2$ in Groups I, II, and III, respectively. Thus the density of all grafts was greater than 10^5 cells/cm^2, in accordance with the criteria of sodding. The trypan blue stain showed a viability of more than 90% of harvested cells.

Gross Observation of the Implanted grafts

All the prostheses harvested within 2 weeks of implantation remained patent except one in ^{3}GII at 14 days, which was occluded because of neointimal hyperplasia at the proximal anastomosis. The luminal surfaces of the grafts were covered with a layer of smooth, white, semitransparent neointima without clot formation. Neointimal thickness of the sodded grafts in GI was thinner than that of GII grafts. All the grafts in GIII remained patent at 7 days. Their surfaces were lined with thrombus or fibrin coagulum, and there was no visible difference between the sodded and unsodded grafts. The patency rates of the late harvested grafts at 60 and 100 days following implantation are shown in Table 16.2. The neointimal surface of the late patent sodded grafts appeared comparable to ones seen in the early stage (Fig. 16.1). The two patent control grafts in GI formed a thicker neointima than the sodded grafts. Prostheses in both groups, in which an occlusion

TABLE 16.2. Graft patency.

Group	Days of implantation	Sodded*	Unsodded*	p
I	100	9/9	2/9	<0.001
II	60	4/5	0/5	<0.05
III	7	5/5	5/5	NS

*No. of patent grafts/no. of experimental grafts

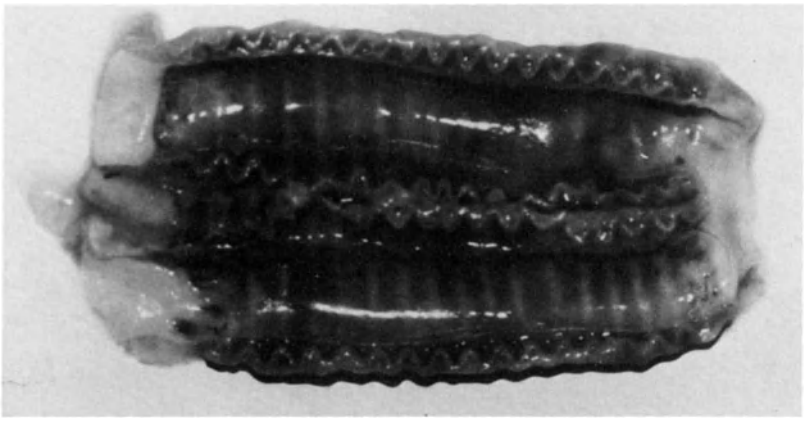


FIGURE 16.1. Gross photograph of sodded graft removed 100 days after implantation showing an intact, smooth, glistening and semitransparent layer of neointima without evidence of gross thrombus.

was suggested by B-Mode ultrasonography, were confirmed to be thrombosed.

The results of immunoassay study are listed in Table 16.3 and Fig. 16.2.

Light microscopy revealed that an intact neointima was established in all the patent venous grafts, and the inner lining of grafts was composed of a continuous monolayer of flat cells. The subendothelial layer consisted of amorphous material and some unidentified cells. Predominant vasa vasorums were seen in the subendothelial layer in two patent control grafts in GI and four patent sodded grafts in GII. The neointimal thickness of GI sodded grafts was markedly thinner than GI control grafts ($p < 0.001$) and GII sodded grafts ($p < 0.01$) as listed in Table 16.4.

TABLE 16.3. Metabolites of extrinsic arachidonic acid in GI grafts.

Samples	No.	6-Keto-PGF_1Alpha*	%	TXB_2*	%
Sodded	1	4745/12825	37.0	1740/3965	43.7
(graft/vein)	2	2980/7125	41.6	1024/2970	34.4
	3	2310/4950	47.1	821/1120	73.3
	4	976/5380	18.1	1510/2120	71.3
	5	810/5455	14.8	1980/2813	70.0
	6	1385/5725	24.2	1900/2900	65.5
	7	2215/5160	42.9	1270/2640	48.1
Control	1	465/17490	2.7	4638/5662	81.9
(graft/vein)	2	300/11650	2.6	2972/3020	98.4

*Unit: cpm

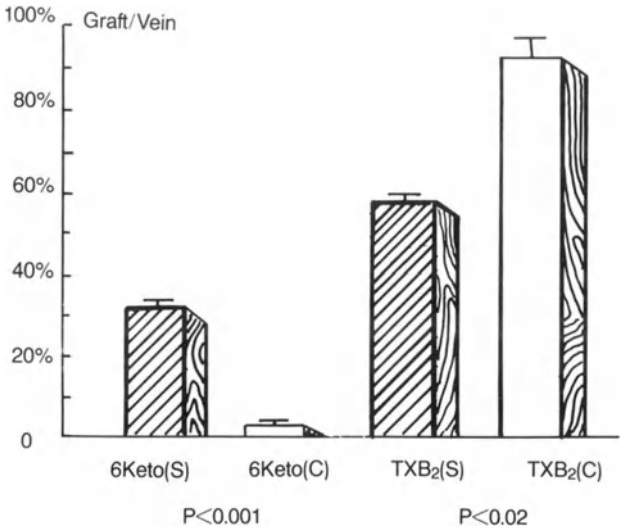


FIGURE 16.2. Histogram showing metabolites of the arachidonic acid produced by newly formed endothelial layers expressed with a ratio of graft to vein in GI at 100 days.

Histoimmunological fluorescent study demonstrated that a specific fluorescent staining for human Factor VIII-related antigen appeared in the harvested cells after 24-hour culture and also in the lining of neointima of sodded venous grafts, but did not appear in the arterial grafts.

Scanning electron microscopy showed a complete endothelialization seen on the sodded midgraft surface in GI, both at 7 and 100 days (Fig. 16.3). The endothelial cells were slender, shuttle-shaped, and aligned parallel to the direction of the blood flow. The cell axial ratio, longitudinal to transverse, was about 8 to 1. The cells were closely confluent. Two patent control grafts of 100 days in GI were also well-endothelialized, with their cells polygonal or shorter shuttle-like. The cellular axial ratio was about 3 to 1 (Fig. 16.4). The inner surface of the GII sodded grafts procured at 7

TABLE 16.4. Intimal thickness (micron).

	Group I			Group II		
	Sodded		Control	Sodded		
Explant day	7	100	100	7	14	60
No.	3	9	2	3	3	4
Mean	229.3	302	783.1	1228.6	1832.7	1908.3
±SD	35.4	106.1	84.2	394.2	352.8	459.6

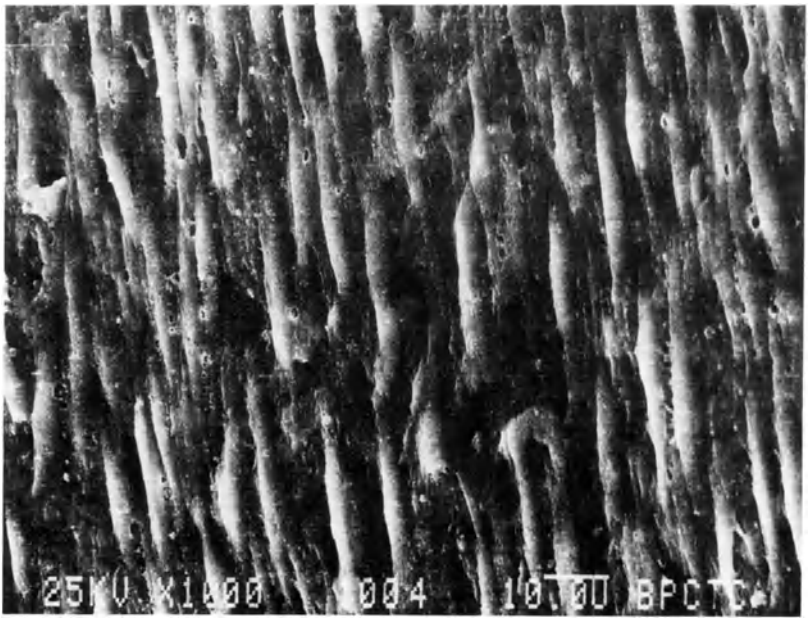


FIGURE 16.3. SEM demonstrating slender shuttle-shaped ECs closely aligned and well polarized in a GI sodded graft at 100 days. (Original magnification ×1000.)

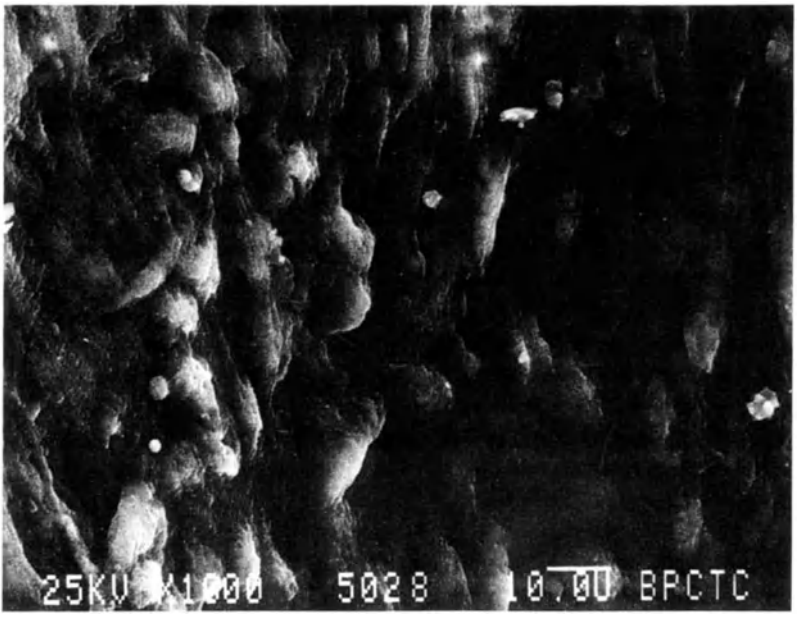


FIGURE 16.4. SEM demonstrating short shuttle-like and polygonal ECs with disorderly alignment and poorly polarized in a GI control patent graft at 100 days. (Original magnification ×1000.)

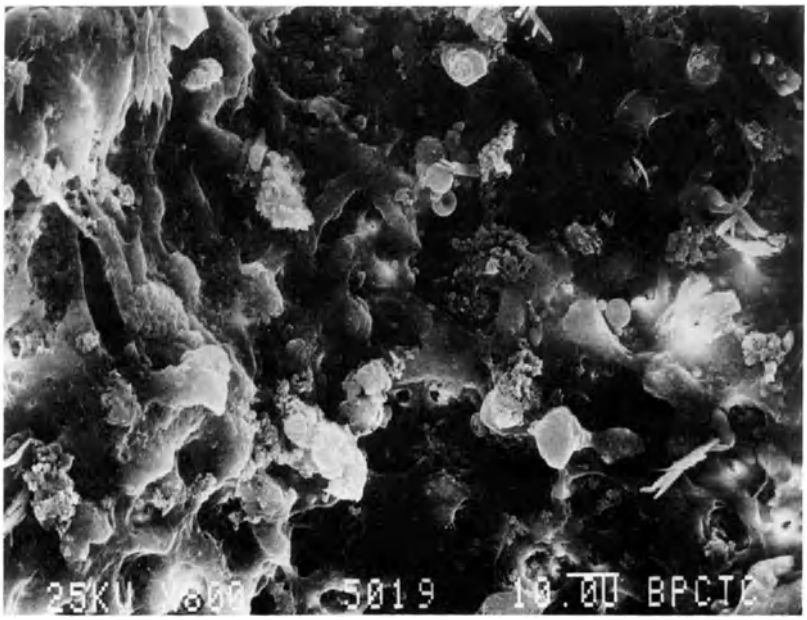


FIGURE 16.5. SEM showing irregular and star-shaped cells unevenly aligned with deposition of some platelets and other blood components. (Original magnification ×800.)

days consisted of star-shaped and other irregular cells that were disordered, uneven, and poorly aligned, with a covering of some platelets and occasional other blood components (Fig. 16.5). Surface cells of patent grafts harvested at 14 and 60 days were shorter, shuttle-like cells with a better alignment parallel to the blood flow. The ratio between the two axes was also about 3 to 1, and under high magnification, microvilli could be seen (Fig. 16.6). In GIII, the inner capsule was composed only of an accumulation of fibrin and blood components in both sodded and control grafts. Typical endothelial cells were scarcely identified on the sodded grafts.

Transmission electron microscopy revealed that the luminal lining cells had morphologic characteristics of endothelial phenotype: microvilli on the cell surface and pinocytes in the plasma as we found before.[10]

Discussion

Since porous prostheses were used as vascular substitutes in the 1950s, modern vascular surgery has been established. After the passive conduits are implanted and healed in vivo, they have a certain biocompatibility

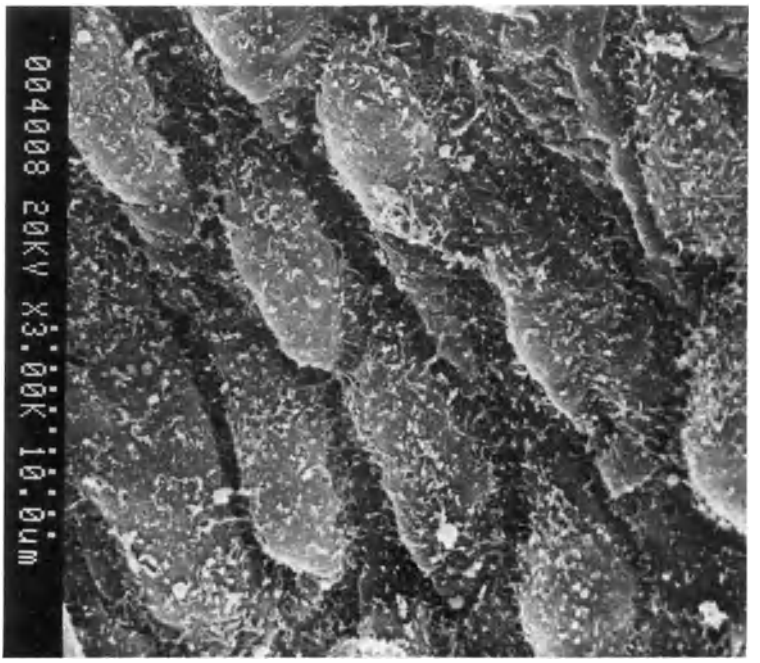


FIGURE 16.6. SEM showing short shuttle-shaped ECs with visualization of microvilli in GII grafts at 60 days. (Original magnification ×3000.)

while protein deposits and fibroblasts migrate into the lumen of the grafts. The patency rate is acceptable in the large caliber and high-blood-flow circumstances. Nevertheless, the intrinsic thrombogenicity is one of the major factors limiting their application in venous and small arterial vessels because of the low velocity and blood flow. The only reasonable solution for this problem seems to be to seed the graft inner surfaces effectively with endothelial cells, although some investigators have tried other alternatives, such as linking heparin, warfarin, activated charcoal, and even artificial membrane on the grafts so as to enhance the prosthetic antithrombogenicity.[14–16]

After Herring successfully seeded ECs on small arterial grafts, this approach has succeeded in improving patency of small arterial prostheses in vivo.[17] However, there has been little research on the improvement of venous graft patency by this technique.[6,7,18,19]

Haimovici emphasized that the period of 1 to 2 weeks following graft implantation was the critical period in realizing a long-term patency of venous grafts.[15] It was reported that the time necessary for ECs to cover 80% of the graft surface when the EC seeding technique was used was 12 weeks for the IVC.[7] Preventing venous graft occlusion during that period is

of paramount importance. We used the high density seeding method, the sodding technique, to promote the endothelialization process. Our results showed that the sodded ECs could cover 100% of the luminal surface of grafts in 1 to 2 weeks in both of our IVC experimental groups. Furthermore, the patency of sodded venous grafts was significantly improved at 100 days and 60 days in GI and GII respectively. This demonstrated that endothelialization can be accomplished much earlier than 12 weeks, and the critical postimplantation period described by Haimovici can be shortened and long-term patency reached by our method. Therefore, it is obvious that early endothelialization may markedly contribute to long-term patency of venous grafts.

The morphological and functional characteristics of endothelialization of the sodded graft in our study were not completely identical between GI and GII. The different shear stress exerted on different grafts of both groups might explain the variation. Ives and Eskin studied the results of shear stress on ECs in vitro and found that the stress was strongly related to the morphology of ECs.[20,21] Our results from in vivo studies match theirs. The sodded ECs on the venous grafts appear to have adaptively morphologic variations under the action of different shear stress in the period of time after sodding. Changes of ECs in GI were more distinct in cellular morphology, confluence, and orientation than those in GII. In GI grafts, the augmented blood flow by distal AVF might contribute to the flattening, maturation, and alignment in the direction of blood flow of the sodded ECs. We found that some platelets aggregated on the ECs at 7 days only in GII, while scarcely any platelets were deposited by that time in GI grafts. This finding might be connected to an insufficiently antithrombotic function of the sodded ECs in GII grafts.

Light microscopy revealed that the difference in the thickness of neointima between the sodded venous groups was significant. The neointima of GII was much thicker than that of GI. We considered that the greater hyperplastic intimal response to the prosthetic grafts of GII was related to greater platelet aggregation and lesser maturity of ECs. Platelets produce platelet-derived growth factor (PDGF) through which the growth of ECs, especially the smooth muscle cells, can be accelerated.[22] The irregularly growing endothelial cells seen at 7 days in the GII grafts were much less able to inhibit proliferation of smooth muscle cells when compared to the confluent endothelial cells. This finding was based on Castellot's in vitro study.[23] In addition, we now appreciate that the ECs themselves can secrete material that stimulates proliferation of both smooth muscle cells and fibroblasts.[24,25] It is therefore possible that the earlier the confluent monolayer of ECs is formed, the thinner the neointima of venous grafts will be. The neointima on the control grafts was also significantly thicker than that of sodded grafts in GI. It is then probable that spontaneous endothelialization of the control grafts formed later than the endothelialization derived from sodded ECs. We demonstrated that PGI_2 production of the luminal

surface of sodded grafts is significantly greater than that of the control grafts and vice versa for the TXB_2 at the 100th day following implantation. Although electron microscopic study showed different morphologic characteristics of ECs between experimental and control grafts, it is not known whether these differences reflect truly different cell origin or different neointimal environment in cellular composition and thickness of the subendothelial layer. Actually, our data showed that the amount of PGI_2 generated by ECs on the neointima of both sodded and control grafts was much lower than that of host veins. The different growth environment of the ECs may affect the production and degradation of PGI_2.

In our study, we expected to realize an early endothelialization, both on the venous and small arterial prosthetic grafts. However, in contrast to the results in the venous groups, the ECs were rarely found on the sodded small arterial grafts at the 7th day. Our results coincided with Herring's study, which also showed typical ECs were scarcely found in the seeded abdominal aortic Dacron grafts within 10 days of implantation.[26] This might be associated with high velocity arterial blood flow that denuded wall-adhered sodded ECs from the graft surface.[27]

We assume that the balance between the fibrin deposit and fibrinolysis on the graft surface is very likely to influence the sodded ECs to be attached and denuded in vivo after implantation. Considering this factor, we employed a clotting method to sod ECs that were only confined on the graft surface by fibrin mesh. After implantation, the balance tends to fibrin deposit that stabilizes the EC adhesion process while fibrinolysis, on the other hand, may cause a loss of ECs. However, as the EC function gradually recovers, the ECs not only obtain a certain ability to attach to the prosthesis wall, but they also promote dissolution of blood clots and plasminogen activator.[28] When the balance turns to the opposite, ECs then emerge to the prosthesis surface. Whereas the higher arterial blood pressure and flow rate favors inhibition of coagulation, the ECs are then more easily denuded by higher shear stress exerted on the arterial prosthesis inner surface.

The maturity process of sodded ECs is somewhat like the hypothesis of "seed and soil" in the concept of aplastic anemia. If the microenvironment composed of bony trabeculae is destroyed, the proliferation of stem cells will be limited. Evidently, the sodding technique applied to the venous prosthesis is much more likely to switch the microenvironment balance to the direction of ECs linking to the vascular surface than when the technique is applied to the arterial prosthesis.

Conclusions

1. Sodding technique with omentally derived MECs significantly improves the patency rate and the inhibition of neointimal thickening of the

venous prosthesis, presumably through a process of early endothelialization.
2. Early endothelialization can be accelerated in a proper hemodynamic condition by creation of an AVF distal to the sodded implanted prosthesis for one week.
3. The amount of PGI_2 yielded from cells of sodded graft is much greater than that from spontaneous cells.
4. Sodding technique is more favorably applied in venous prostheses than in arterial ones.
5. The MEC sodding technique improves the performance of venous vascular prostheses.

Acknowledgment. We thank Meadox Medical Inc., Oakland, N.J. for providing us with the knitted double-velour Microvel vascular prosthesis used for this study.

References

1. Kempczinski RF, et al. Endothelial cell seeding of a new PTFE vascular prosthesis. J Vasc Surg. 1985, 2:424–429.
2. Stanley JO, et al. Enhance patency of small-diameter, externally supported Dacron iliofemoral grafts seeded with endothelial cells. Surg. 1985, 92:994–1005.
3. Allen BT, et al. Influence of endothelial cell seeding on platelet deposition and patency in small-diameter Dacron arterial grafts. J Vasc Surg. 1984, 1:224–233.
4. Graham LM, et al. Improved patency of endothelial cells, long, knitted Dacron and ePTFE vascular prostheses. Asaio J. 1985, 8:65–73.
5. Shindo SD, et al. Improved patency of collagen-impregnated grafts after in vitro autogenous endothelial cell seeding. J Vasc Surg. 1987, 6:325–332.
6. Herring M, et al. Patency in canine inferior vena cava grafting: Effects of graft material, size and endothelial seeding. J Vasc Surg. 1984, 1:887.
7. Koveker GB, et al. Endothelial cell seeding of ePTFE vena cava conduits: Effects on luminal production of prostacyclin, platelet adherence and fibrinogen accumulation. J Vasc Surg. 1988, 7:600–605.
8. Jarrel BE, et al. Use of isolated capillary endothelial cells for the immediate establishment of a monolayer on a vascular graft at surgery. Surg. 1986, 100:392–399.
9. Rupnick MA, et al. Endothelialization of vascular prosthetic surfaces after seeding or sodding with human microvascular endothelial cells. J Vasc Surg. 1989, 9:788–795.
10. Wang Z-G, et al. Rapid cellular luminal coverage of Dacron inferior vena cava prostheses in dogs by immediate seeding of autogenous endothelial cells derived from omental tissue. J Vasc Surg. 1990, 12:168–79.
11. Yates SG, et al. The preclotting of porous arterial prostheses. Ann Surg. 1978, 188:611–622.
12. Watkins MT, et al. Adult human saphenous vein endothelial cells: Assessment

of their reproductive capacity for use in endothelial seeding. J Surg Research. 1989, 36:588–596.
13. Hoover EL, et al. Cyclooxygenase activity in the thoracoabdominal aorta after 24 hours of intraaortic balloon counterpulsation: An assessment of the effects of localized mechanical trauma. Vasc Surg. 1989, 23:175–182.
14. Huffnagel CA, et al. Comparative studies of cardiac and vascular implants in relation to thrombosis. Surg. 1969, 661:11–16.
15. Haimovici H, et al. An experimental and clinical evaluation of grafts in the venous system. Surg Gynecol Obstet. 1970, 131:1173–1186.
16. Sharp WV, et al. Electrolous: A new vascular interface. Trans Am Soc Artif Intern Organs 1968, 14:73–77.
17. Herring M, et al. A single-staged technique for seeding vascular grafts with autogenous endothelium. Surg. 1978, 84:498–504.
18. Plate G, et al. Endothelial seeding of venous prostheses. Surg. 1984, 96:929–936.
19. Nghia MV, et al. Effect of endothelial seeding and graft material on the patency rate of venous grafts. Vasc Surg. 1989, 23:208–213.
20. Ives CJ, et al. The importance of cell organ and substrata in the kinetics of endothelial cell alignment in response to steady flow. Trans Am Soc Artif Intern Organs 1983, 29: 269–274.
21. Eskin SG, et al. Responses of culture endothelial cells to steady flow. Microvasc Res. 1984, 28:87–94.
22. Zacharias RK, et al. Growth factor production by polytetrafluoroethylene vascular grafts. J Vasc Surg. 1988, 7:606–610.
23. Castellot JJ Jr, et al. Cultured endothelial cells produce a heparin-like inhibitor of smooth muscle cell growth. J Cell Biol. 1981, 90:372–379.
24. Dicorleto PE, et al. Cultured endothelial cells produce a platelet derived growth factor-like protein. Proc Nat Acad Sci. 1983, 80:1919–1923.
25. Fox PL, et al. Regulation of production of a platelet-derived growth factor-like protein in cultured bovine aortic endothelial cells. J Cell Physiol. 1984, 21:298–308.
26. Herring M, et al. Endothelial seeding of Dacron and polytetrafluoroethylene grafts: The cellular events of healing. Surg. 1984, 96:745–754.
27. Rosenman LE, et al. Kinetics of endothelial cell seeding. J Vasc Surg. 1980, 1:877–887.
28. Levin EG, et al. Cultured bovine endothelial cells produce both urokinase and tissue-type plasminogen activators. J Cell Biol. 1982, 94:631–636.

17
Protective Effects of Intrathecal Lidocaine Administration on Ischemic Injury of the Spinal Cord

ERIYA OKUDA, HIROSHI YOSHIZU, NOBUO HATORI, YOSHIYUKI HAGA, YOZO URIUDA, MASAFUMI SHIMIZU, ATSUHIRO MITSUMARU, and SUSUMU TANAKA

Summary

The protective effects of intrathecal lidocaine administration on experimental ischemic spinal cord injury following aortic cross-clamping (AXC) were studied. Twelve mongrel dogs were divided into two groups, six animals in an intrathecally saline-administered group (control group) and the other six in an intrathecally lidocaine-administered group (lidocaine group). The dose of lidocaine administration was determined by the change of somatosensory-evoked potential traces generated by brachial nerve stimulation. Following administration of the agents, all dogs underwent cross-clamping of the thoracic aorta and the left subclavian artery for 60 minutes. Mean proximal aortic pressure during AXC remained in a similar range to baseline pressure until 40 minutes after AXC in the lidocaine group, while it increased significantly in the control group. The ratio of mean distal aortic pressure to mean proximal aortic pressure during AXC (D/P), which was calculated for estimation of collateral blood flow changes, gradually increased in the lidocaine group, but did not change in the control group. Four of six dogs had spastic paraplegia in the control group, but none in the lidocaine group ($p < 0.05$). Histological examination revealed that extensive infarction of the gray matter was noticed in the control group, but changes in the lidocaine group were only slight. From these results, it was concluded that intrathecal lidocaine administration controlled proximal hypertension effectively, increased D/P during AXC, and diminished neurological damages following AXC.

Introduction

Ischemic injury of the spinal cord resulting in paraplegia is a catastrophic complication following surgery on the descending thoracic and thoracoabdominal aorta. Although it has been reported[1–3] that spinal cord injury

increases when aortic cross-clamping (AXC) time exceeds 30 minutes, no precise mechanism has been identified to explain the generation of paraplegia after AXC. Therefore, no technique has yet been proposed as an absolute preventive method for this devastating complication.

Many anesthetics are known to decrease metabolism of the central nervous system.[4] Intravenous[5] and intrathecal[6] lidocaine administration have been demonstrated to have favorable effects on experimental spinal cord injury generated by inflation of the epidural balloon. Astrup and colleagues[7–10] reported that lidocaine has certain effects on cerebral metabolism similar to those induced by hypothermia. In addition to its antiarrhythmic effect, lidocaine may protect the myocardium from ischemic and reperfusion injury.[11–14] Lesnefsky and colleagues[15] proved that lidocaine reduces lipid peroxidation product (conjugated dienes) in the myocardium during early reperfusion in experimental models by its membrane-stabilizing effects. Reperfusion injury was considered to be an important factor in paraplegia following AXC.[16,17] Consequently, the incidence of paraplegia following AXC can be expected to decrease by these effects of lidocaine. Although the primary site of action of spinal anesthesia was believed to be the spinal roots, Cohen[18] proved that a considerable concentration of lidocaine is found in the gray matter after intrathecal administration.

This experimental study was designed to investigate the protective effects of intrathecal lidocaine administration on ischemic spinal cord injury.

Methods

Twelve mongrel dogs weighing 12 to 19 kg were divided into two groups. Normal saline was administered intrathecally to six animals (control group), and lidocaine was administered in the same way to the other six (lidocaine group).

The animals were anesthetized with an intravenous injection of pentobarbital sodium (25 mg/kg), following an intramuscular injection of Ketamine HCl (5 mg/kg), and were intubated and placed on a Harvard ventilator using room air with 5 cm H_2O of positive end-expiratory pressure. Muscle relaxation was achieved using pancuronium bromide (0.1 mg/kg) at appropriate intervals to control ventilation and muscular artifacts in the somatosensory-evoked potential (SEP) traces.

Approximately 1000 ml of nondextrose lactate Ringer solution was infused intravenously during the procedure.

Pressure-monitoring catheters were inserted into the right brachial artery and the femoral artery for monitoring proximal and distal aortic pressures, respectively.

After a small laminectomy at L-1, a 22-gauge catheter was inserted into

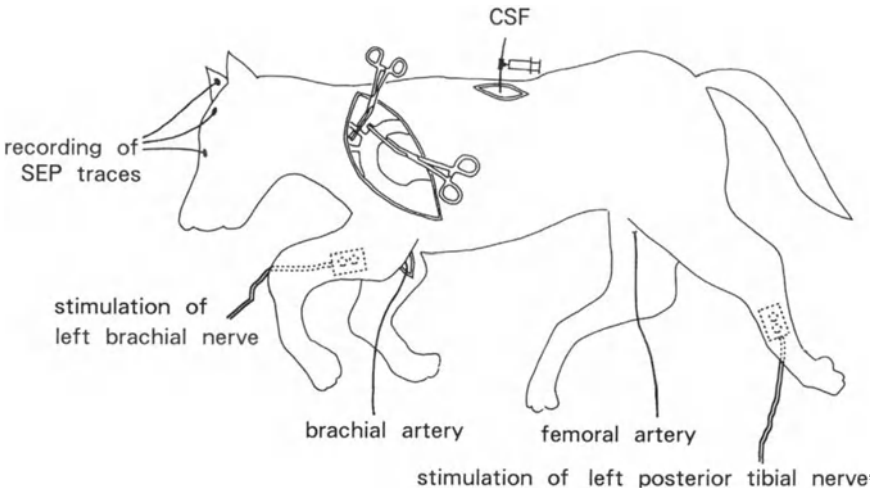


FIGURE 17.1. Schematic representation of the experimental model.

the subarachnoid space under direct vision by puncturing the dura for pressure monitoring and administration of normal saline or lidocaine.

The descending thoracic aorta and the left subclavian artery were exposed through a left thoracotomy in the fourth intercostal space.

In the control group, 0.9% normal saline (3.5 ml) at 37°C was administered intrathecally. At 15 minutes after saline administration, the descending thoracic aorta at the origin of the left subclavian artery and the left subclavian artery were cross-clamped (Fig. 17.1). In the lidocaine group, 3% lidocaine HCl (3.5 ml) at 37°C was administered intrathecally, and SEP traces generated by stimulation of the brachial nerve were monitored to evaluate the diffusion of spinal anesthesia. If loss of SEP was noted within 15 minutes, AXC was performed 15 minutes after lidocaine administration. If SEP traces showed no change, another dose of lidocaine HCl (3.5 ml) was administered 15 minutes after the initial injection, and AXC was performed 5 minutes later.

All animals were subjected to a 60-minute period of AXC.

Physiological studies were as follows: The electrocardiogram (ECG) was continuously monitored. Mean proximal aortic pressure and cerebrospinal fluid pressure (CSFP) were measured immediately prior to the intrathecal injection (baseline pressure); 5 and 10 minutes after intrathecal injection; immediately prior to AXC (preocclusion pressure); 5, 10, 20, 30, 40, 50, and 60 minutes after AXC; and 5, 10, 20, and 30 minutes following reperfusion. Spinal cord perfusion pressure (SCPP) was defined as the difference between arterial pressure and CSFP and was calculated by subtracting CSFP from the mean distal aortic pressure. The mean distal aortic pressure and SCPP were assessed during AXC.

Distal aortic pressure during AXC is considered to be regulated essentially by proximal aortic pressure. Therefore, the ratio of mean distal aortic pressure to mean proximal aortic pressure (D/P) was calculated for estimation of collateral blood flow change during AXC.

SEP traces were generated with an evoked potential system (Neuropack II plus, Nihon Kohden, Japan) by electrical stimulation of the left posterior tibial nerve in all animals. In addition, the left brachial nerve was stimulated in the lidocaine group. Responses were recorded from midline scalp electrodes at the nasion and at a point 55% of the distance from the nasion to the inion. A grounding electrode was placed in the right ear. Repetitive stimuli ($n = 256$) using a 200 V square-wave stimulus of 0.2 msec duration at a frequency of 2 Hz with a 2 Hz LoPass and a 1 kHz HiPass filter evoked improved SEP traces.

Blood was collected from the femoral artery prior to AXC, immediately prior to reperfusion, and 60 minutes after reperfusion. Then arterial blood gases, electrolytes (Na, K, Ca), lactic acid, and pyruvic acid were measured.

Rectal temperature during AXC was recorded.

Neurological assessment of hind limb paralysis was performed 48 hours after the operation.

Animals were sacrificed for histological examinations from 2 to 20 days after the operation. Then the spinal cord was removed and divided into three segments. The nonischemic part of the upper thoracic segment was evaluated as a control to determine ischemic damage to the lower thoracic and midlumbar segments.

All experimental animals received humane care and treatment in compliance with the "Guide for the Care and Use of Laboratory Animals," published by the National Institute of Health (NIH Publication No. 85-23, 1985).

All values are reported as mean ± the standard deviation. The data were analyzed with repeated-measures analysis of variance with a Student-Newman-Keuls multiple comparison test and, where appropriate, Student's t test. Fisher's exact test was used to compare the rates of paraplegia. Probability values of 0.05 or less were required for assumption of statistical significance.

Results

Neurological Findings

At 48 hours, four of six dogs in the control group had spastic paraplegia of the hind limbs. The remaining two dogs could stand and walk with no impairment. In contrast no dogs in the lidocaine group had spastic paraple-

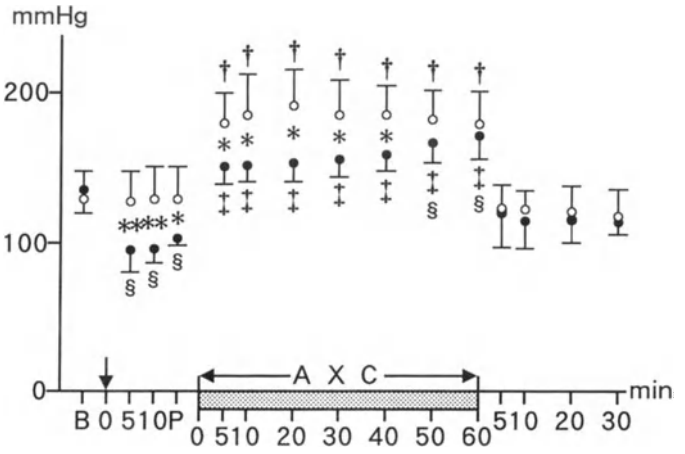
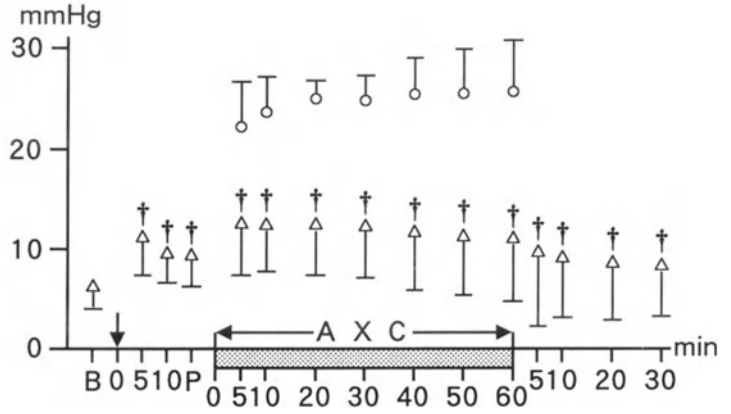


FIGURE 17.2. Plots of mean proximal aortic pressure during the experimental protocol. Mean proximal aortic pressure during aortic cross-clamping (AXC) in the lidocaine group was as high as the baseline (B) pressure until 40 min after AXC because of the initial drop induced by intrathecal lidocaine administration. (○, control group; ●, lidocaine group; ↓, intrathecal administration; *, $p < 0.05$, **, $p < 0.01$ control group vs. lidocaine group; †, $p < 0.05$ vs. B and preocculusion (P) pressure in control group; §, $p < 0.05$ vs. B pressure in lidocaine group; ‡, $p < 0.05$ vs P pressure in lidocaine group)

gia. One of six dogs showed good movement of the joints of the hind limbs but was unable to stand. The other five dogs could walk and run. Animals in the lidocaine group showed a significant difference in neurological outcome. Thus, intrathecal lidocaine administration was regarded to cause a significant reduction in the incidence of spastic paraplegia at 48 hours after the operation ($p < 0.05$, Fisher's exact test).

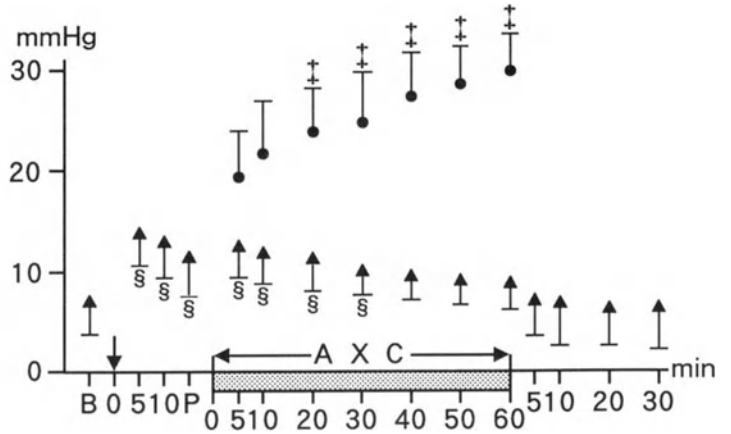
Mean Proximal Aortic Pressure

In the control group, mean proximal aortic pressure (Fig. 17.2) was on the same level with baseline pressure after saline administration. Therefore, mean proximal aortic pressure during AXC increased significantly over baseline pressure and preocclusion pressure. Following lidocaine administration, mean proximal aortic pressure dropped significantly from 136 ± 13 mmHg to 92 ± 12 mmHg in the lidocaine group. Therefore, mean proximal aortic pressure during AXC was as high as baseline pressure until 40 minutes after AXC, while it increased significantly over preocclusion pressure (102 ± 4 mmHg) during AXC. The comparison of mean proximal aortic pressure between the two groups revealed significant differences in the duration from intrathecal administration to 40 minutes after AXC.



A

FIGURE 17.3A. Plots of cerebrospinal fluid pressure (CSFP) and mean distal aortic pressures in the control group. CSFP increased significantly above the B pressure after intrathecal saline administration. The change of mean distal aortic pressures during AXC was not statistically significant. (○, mean distal aortic pressure; △, cerebrospinal fluid pressure; †, $p < 0.05$ vs. B pressure)



B

FIGURE 17.3B. Plots of cerebrospinal fluid and mean distal aortic pressure in the lidocaine group. CSFP increased significantly above the B pressure after intrathecal lidocaine administration, but it decreased gradually during AXC. Mean distal aortic pressure at times after 20 min were significantly higher than that after 5 min. (●, mean distal aortic pressure; ▲, cerebrospinal fluid pressure; §, $p < 0.05$ vs. B pressure; ‡, $p < 0.05$ vs. 5 min of AXC)

Mean Distal Aortic Pressure During AXC

Mean distal aortic pressure was on the same level during AXC (Fig. 17.3A,17.3B) in the control group. It increased gradually in the lidocaine group, and the pressure at times after 20 minutes was significantly higher than that after 5 minutes. However, no difference was noticed in the comparison of the two groups.

Ratio of Mean Distal Aortic Pressure to Mean Proximal Aortic Pressure During AXC (D/P)

The D/P (Fig. 17.4) at 5 minutes after AXC was on the same level between the two groups. As time passed, the D/P increased significantly in the lidocaine group but did not change in the control group. The difference between the two groups was not of statistical significance. The relationship between the time and the mean of D/P appeared nonlinear. Thus the logarithmic transformation was performed to achieve linearity (Fig. 17.5). Then after linear regression analysis, a significant difference was noticed in the comparison of the two regression parameters (0.05 ± 0.02 versus 0.13 ± 0.02).

CSFP

CSFP (Figs. 17.3A, 3B) increased significantly over baseline pressure after the intrathecal injection in each group. Therefore, rises after AXC and

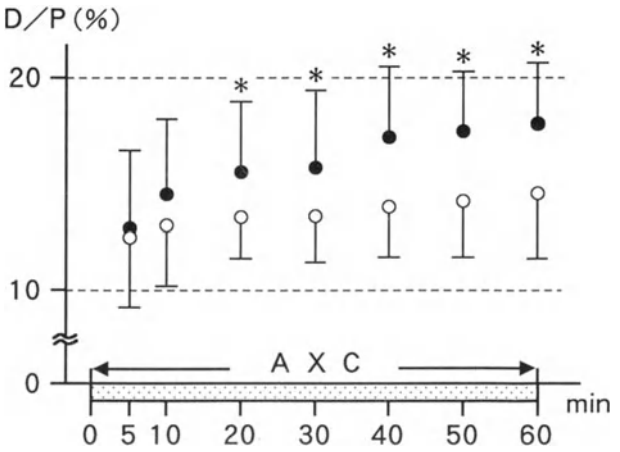


FIGURE 17.4. Plots of the ratio of mean distal aortic pressure to mean proximal aortic pressure (D/P) during AXC. The change of the D/P in the lidocaine group was significant, but not in the control group. (○, control group; ●, lidocaine group; *, $p < 0.05$ vs. 5 min of AXC in lidocaine group)

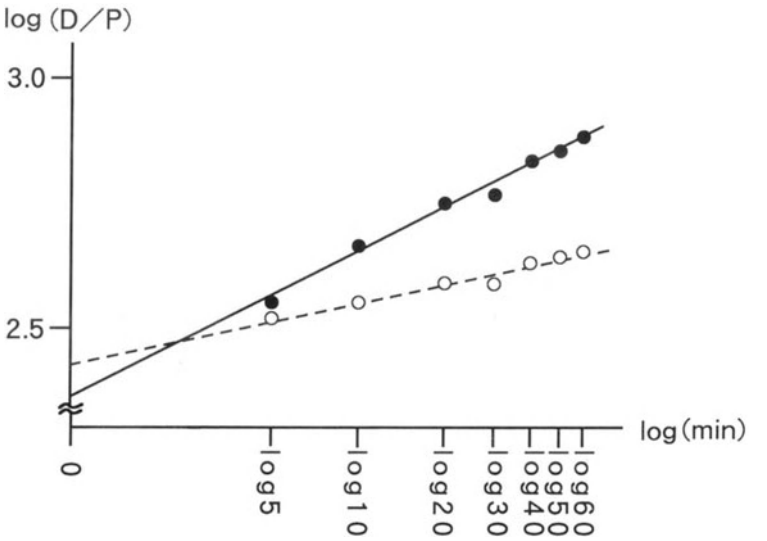


FIGURE 17.5. Plots of linear regression analysis after logarithmic transformation. The regression line in the control group is expressed as a broken line.: Log(D/P) = 0.05 Log(min) + 2.43; r = 0.97; $p < 0.01$. The regression line in the lidocaine group is expressed as a solid line.: Log(D/P) = 0.13 Log(min) + 2.36; r = 0.99; $p < 0.01$. A significant difference was noticed in the comparison of the two regression parameters ($p < 0.05$). (○, control group; ●, lidocaine group)

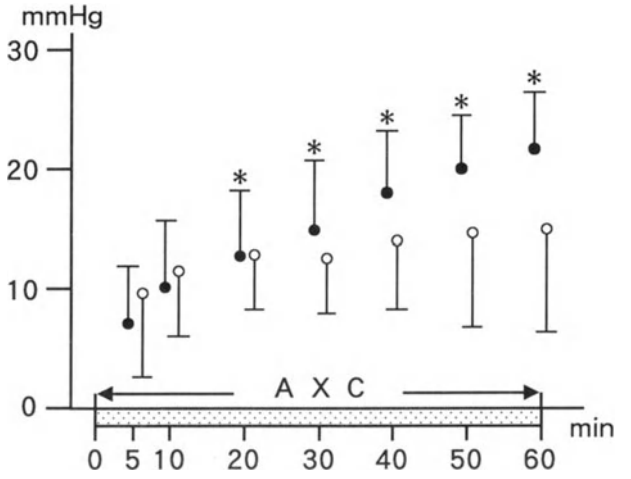


FIGURE 17.6. Plots of spinal cord perfusion pressure (SCPP) during AXC. SCPP at times after 20 min were significantly higher than that after 5 min in the lidocaine group. (○, control group; ●, lidocaine group; * $p < 0.05$ vs. 5 min of AXC in lidocaine group)

drops after reperfusion were not statistically significant. No significant difference was detected in the comparison of the two groups.

SCPP During AXC

SCPP during AXC (Fig. 17.6) increased gradually in the lidocaine group but not in the control group. The difference between the two groups was not of statistical significance.

ECG

All animals displayed normal sinus rhythm before AXC. No arrhythmia was observed after AXC in the lidocaine group. In contrast, ventricular arrhythmias were seen in five of six dogs following AXC in the control group. Three dogs had multifocal ventricular ectopies, and two had bigeminy. These ventricular ectopic beats appeared 10 or 15 minutes after AXC, then disappeared in the late phases of AXC.

SEP

In three of six dogs in the lidocaine group, SEP traces generated by brachial nerve stimulation disappeared within 15 minutes after lidocaine administration. In the remaining three dogs, loss of SEP was noted after the additional lidocaine administration. The amplitude in SEP traces recovered during AXC in all animals. SEP traces generated by stimulation of the posterior tibial nerve disappeared quickly after the initial lidocaine administration and did not recover during the operation in the lidocaine group. In the control group, SEP did not change after saline administration. In five of six dogs in the control group, SEP traces disappeared after AXC, but in one of these five dogs SEP traces recovered after reperfusion. Another dog in the control group showed no change of SEP traces during the procedure.

Administration Volume of Normal Saline and Lidocaine

In the control group, 3.5 ml of saline was administered intrathecally to all animals. In the lidocaine group, 3.5 ml of 3% lidocaine was administered initially to all animals, and the same dose was injected additionally in three of six dogs. Therefore, the average dose of 3% lidocaine was 5.25 ml, that is, 158 mg. This was equivalent to 10.3 mg per kg of weight.

Blood

No significant changes were observed in blood gases (PH, PCO_2, PO_2, HCO_3, B.E., and O_2SAT) and electrolytes (Na, K, and Ca) (Table 17.1,

TABLE 17.1. Blood gases and electrolytes.

	Prior to AXC		Prior to reperfusion		60 min after reperfusion	
	Control	Lidocaine	Control	Lidocaine	Control	Lidocaine
PH	7.44 ± 0.08	7.43 ± 0.06	7.40 ± 0.08	7.44 ± 0.04	7.45 ± 0.09	7.42 ± 0.05
PCO_2 (mmHg)	29.6 ± 5.2	29.9 ± 6.2	25.8 ± 2.5	25.1 ± 4.7	26.8 ± 3.0	28.1 ± 5.9
PO_2 (mmHg)	114.9 ± 24.7	110.2 ± 17.9	112.1 ± 27.2	120.9 ± 19.6	125.9 ± 11.8	129.5 ± 18.6
HCO_3^- (mmol/l)	19.9 ± 2.1	19.3 ± 2.5	16.0 ± 3.5	16.7 ± 2.9	18.8 ± 3.3	17.9 ± 3.2
B.E. (mmol/l)	−3.6 ± 2.7	−4.4 ± 2.3	−7.8 ± 2.1	−6.7 ± 2.8	−4.6 ± 4.1	−5.9 ± 3.0
O_2SAT (%)	97.9 ± 1.6	97.9 ± 1.2	97.4 ± 2.1	98.5 ± 0.6	98.7 ± 0.5	98.7 ± 0.6
Na (mmol/l)	142.9 ± 4.7	146.2 ± 1.8	142.2 ± 4.1	144.8 ± 1.0	142.3 ± 4.3	145.8 ± 1.0
K (mmol/l)	4.4 ± 0.4	4.0 ± 0.2	4.8 ± 0.7	4.3 ± 0.3	4.8 ± 0.7	3.9 ± 0.4
Ca (mmol/l)	0.98 ± 0.10	1.03 ± 0.08	0.96 ± 0.12	0.96 ± 0.14	0.99 ± 0.13	1.04 ± 0.11

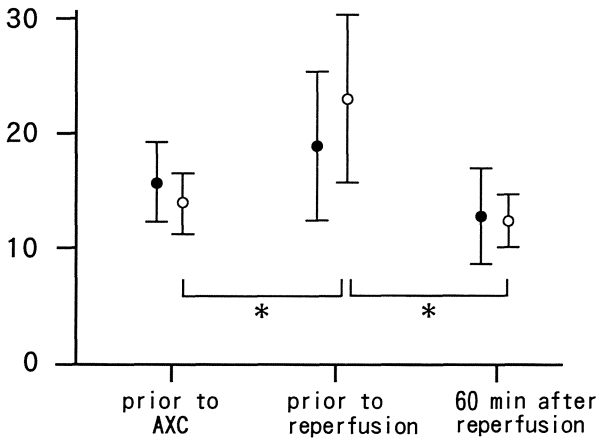


FIGURE 17.7. Plots of the ratio of lactic acid to pyruvic acid (L/P). The L/P prior to reperfusion was significantly higher than that prior to AXC and that 60 min after reperfusion in control group. (○, control group; ●, lidocaine group)

and Fig. 17.7). The changes of the ratio of lactic acid to pyruvic acid (L/P) were not significant between the two groups or within the lidocaine group. In the control group, however, L/P prior to reperfusion was significantly higher than that prior to AXC and that 60 minutes after reperfusion.

Rectal Temperature

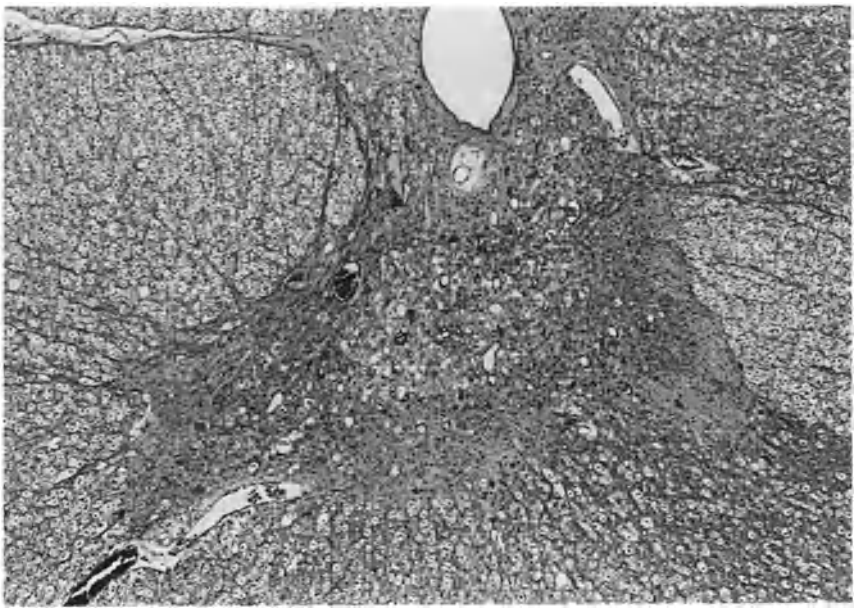
The lowest temperature in the rectum during AXC was 35.2 ± 0.5°C in the control group and 34.9 ± 0.7°C in the lidocaine group, where no significant difference was seen.

Histological Findings

The upper thoracic segments for nonischemic parts were normal in all animals. The lower thoracic or midlumbar segment in the control group demonstrated extensive infarction in the gray matter. In contrast, the corresponding segment in the lidocaine group showed limited neutrophil infiltration and edema of axons and nerve cells. Therefore, the histological changes (Figs. 17.8A, 17.8B) in the lidocaine group were considered to be reversible ischemic injuries.

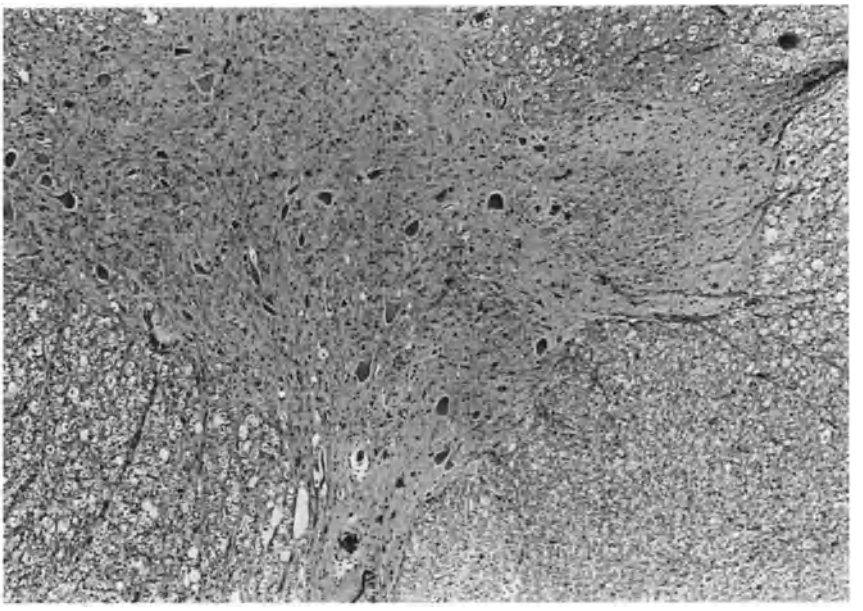
Discussion

Symbas and colleagues[19] reported that plasma epinephrine and norepinephrine markedly increased in an animal group with simple AXC. Prox-



A

FIGURE 17.8A. Microscopic section of midlumbar segment of the spinal cord taken from a paraplegic dog in the control group at 4 days after the operation. The extensive infarction in the gray matter was demonstrated.



B

FIGURE 17.8B. Corresponding region in the lidocaine group. Changes in the gray matter were only slight in contrast to those in the control group.

imal aortic pressure in the control group of this study elevated gradually during the early phase of AXC. The rise of proximal aortic pressure stopped about 20 minutes after AXC. The ventricular ectopic beats appeared correspondingly in the control group. These phenomena may be caused by the release of catecholamines. However, the plasma level of catecholamines in patients with high spinal anesthesia was reported to be lower than that with inhalation anesthesia during operations and recovery.[20] In addition, degree of suppression of plasma epinephrine excretion correlated with the blocked level of adrenal medulla innervation under spinal anesthesia. High spinal anesthesia reduces plasma catecholamines because it interrupts the conduction of afferent nerves from surgical stresses and of efferent nerves from the upper central nervous system. As vasoconstrictive action of catecholamine is obviously harmful to ischemic organs, high spinal anesthesia is beneficial for ischemic organs during AXC.

According to a retrospective analysis of 596 traumatic ruptures of the thoracic aorta, mortality rates associated with bypass procedure, temporary shunting and simple cross-clamping were 16.7%, 11.4% and 5.8%, respectively, while incidence of paraplegia was 2.2%, 2.3% and 5.8%, respectively.[3] In addition, some expert surgeons prefer simple AXC and rapid reanastomosis in elective operations on the thoracic and thoracoabdominal aorta.[1,21–24] A simple, safe, and universal surgical procedure in this field has not yet been created.

Proximal hypertension following AXC without adjuncts is deleterious for the left ventricle with depressed function and may cause cerebrovascular accidents as well. Therefore, vasodilators have been used for reduction of proximal aortic pressure and for improvement of collateral circulation to the distal organs. In spite of its beneficial effect on proximal hypertension, Marini and colleagues[25] reported that intravenous administration of sodium nitroprusside (SNP) increased the risk of neurological injury by reducing spinal cord perfusion pressure. They showed that mean distal aortic pressure decreased gradually during AXC while mean proximal aortic pressure was maintained on the same level with baseline pressure with infusion of SNP. In other words, the D/P decreased gradually. They proved by means of radiolabeled microspheres that a blood flow to the lower spinal cord segment decreased compared with controls during AXC in animals receiving SNP. The same tendency was recognized in the report by Gelman and colleagues.[26] Although the difference did not reach statistical significance probably because of the high values before AXC, plasma epinephrine was reported to increase during AXC in dogs given intravenous SNP.[19] Thus the failure of improvement in collateral circulation by SNP may result from releasing catecholamines.

In contrast, the D/P increased gradually with statistical significance following intrathecal lidocaine administration in our experiments. In linear regression analysis after logarithmic transformation, the regression parameter in the lidocaine group was significantly larger than that in the control

group, which means the collateral blood flow to the distal organs during AXC increased more in the lidocaine group than in the control group. Thus, intrathecal lidocaine administration is considered to have a beneficial effect on reducing proximal hypertension and increasing collateral blood flow during AXC. The change of the L/P in blood may be affected by this effect.

The pattern of changes in CSFP resembles that of proximal mean aortic pressure. Therefore, proper control of proximal hypertension in the lidocaine group may suppress CSFP rise and be favorable. In the early phase during AXC, CSFP in the lidocaine group was higher than that in the control group because the administered dose was larger in the lidocaine group than in the control group. In addition, mean distal aortic pressure in the lidocaine group was lower than that in the control group in proportion to mean proximal aortic pressure. Therefore, SCPP in the lidocaine group was considered to be lower than that in the control group. In the late phase of AXC, however, mean distal aortic pressure increased gradually in the lidocaine group, and SCPP was higher than that in the control group. But SCPP in the lidocaine group could not reach an adequate pressure level to protect the spinal cord and was as high as that in the control group in the experiment by McCullough and (McCullough) colleagues[27] in which eight of ten dogs had spastic paraplegia.

As Astrup and colleagues[7–10] have described, lidocaine may have a possibility to provide hypothermia-like protection on cerebral metabolism while intrathecal lidocaine administration may directly decrease spinal cord metabolism. On the other hand, Marcus and colleagues[28] reported that stimulation (30 V, 5 Hz) of the intact left sciatic and femoral nerves resulted in a 50% increase in blood flow to the left gray matter of the lumbosacral cord. In addition, it was demonstrated that stimulation of the femoral and the sciatic nerves increased the rate of glucose metabolism in spinal cord gray matter on the stimulated side.[29] In other words, blood flow to spinal cord gray matter increased selectively in response to metabolic stimuli. However, metabolic stimuli are blocked by spinal anesthesia. Then the increase in blood flow at the gray matter is not forced by metabolic stimuli under spinal anesthesia. Consequently, a silent environment for the spinal cord, produced by spinal anesthesia, is considered to be advantageous for the ischemic spinal cord.

Cohen[18] proved that the highest concentrations of intrathecally-administered 2.5% lidocaine (1 ml) are found in the posterior and lateral columns, but lidocaine concentration in the anterior horn was about three quarters of the minimum effective concentration.[30] In this study, we used 3% lidocaine. In addition, drainage of lidocaine to the vein may decrease because of the interruption of circulation following AXC. Consequently, the optimal concentration can still be expected in the anterior horn.

In the lidocaine group in this experiment, one of six dogs did not recover completely, and minor changes were seen in histological examination.

Therefore, the spinal cord during AXC was not free from the effects of ischemia even if intrathecal lidocaine administration was performed. However, intrathecal lidocaine administration was considered to diminish spinal cord injury generated during and after AXC.

In conclusion, intrathecal lidocaine administration under the conditions of this experiment effectively controlled proximal hypertension, increased D/P during AXC, and diminished neurological damages. These results suggest that intrathecal lidocaine administration is useful in clinical settings requiring AXC during surgery on the descending thoracic and thoracoabdominal aorta.

Acknowledgments. We gratefully acknowledge Hiroshi Tamaki and Yoshimasa Kusama for technical assistance in this study; and Fumiko Tamada, M.D., and Professor Minoru Suzuki, Department of Pathology I, National Defense Medical College, for assistance with the histological analysis.

References

1. Livesay JJ, Cooley DA, Ventemiglia RA, Montero CG, Warrian RK, Brown DM, Duncan JM. Surgical experience in descending thoracic aneurysmectomy with and without adjuncts to avoid ischemia. Ann Thorac Surg. 39(1);1985:37–46.
2. Katz NM, Blackstone EH, Kirklin JW, Karp RB. Incremental risk factors for spinal cord injury following operation for acute traumatic aortic transection. J Thorac Cardiovasc Surg. 81(5);1981:669–674.
3. Svensson LG, Antunes MJ, Kinsley RH. Traumatic rupture of the thoracic aorta. A report of 14 cases and a review of the literature. S Afr Med J. 67(21);1985:853–857.
4. Siesjö BK. *Brain Energy Metabolism.* p 233–265. Chichester, John Wiley and Sons, 1978.
5. Kobrine AI, Evans DE, LeGrys DC, Yaffe LJ, Bradley ME. Effect of intravenous lidocaine on experimental spinal cord injury. J Neurosurg. 60(3);1984:595–601.
6. Cole DJ, Shapiro HM, Drummond JC, Zivin JA. Halothane, fentanyl/nitrous oxide, and spinal lidocaine protect against spinal cord injury in the rat. Anesthesiology 70(6);1989:967–972.
7. Astrup J, Skovsted P, Gjerris F, Sørensen HR. Increase in extracellular potassium in the brain during circulatory arrest: effects of hypothermia, lidocaine, and thiopental. Anesthesiology, 55 (3);1981:256–262.
8. Astrup J, Sørenson PM, Sørenson HR. Inhibition of cerebral oxygen and glucose consumption in the dog by hypothermia, pentobarbital, and lidocaine. Anesthesiology 55(3);1981:263–268.
9. Astrup J. Energy-requiring cell functions in the ischemic brain. J Neurosurg. 56(4);1982:482–497.
10. Astrup J, Sørensen PM, Sørensen HR. Oxygen and glucose consumption re-

lated to Na^+–K^+ transport in canine brain. Stroke 12(6);1981:726–730.
11. Nasser FN, Walls JT, Edwards WD, Harrison CE. Lidocaine-induced reduction in size of experimental myocardial infarction. Am J Cardiol. 46(6);1980:967–975.
12. Kyo S, Laraia PJ, Magrassi P, Levine FH, Tsai CH, Austen WG, Buckley MJ. Myocardial protection by lidocaine during cardioplegia. J Surg Res. 34(6);1983:533–542.
13. Schaub RG, Stewart G, Strong M, Ruotolo R, Lemole G. Reduction of ischemic myocardial damage in the dog by lidocaine infusion. Am J Pathol. 87(2);1977:399–405.
14. Boudoulas H, Karayannacos PE, Lewis RP, Kakos GS, Kilman JW, Vasko JS. Potential effect of lidocaine on ischemic myocardial injury: experimental and clinical observations. J Surg Res. 24(6);1978:469–476.
15. Lesnefsky EJ, VanBenthuysen KM, McMurtry IF, Shikes RH, Johnston RB, Horwitz LD. Lidocaine reduces canine infarct size and decreases release of a lipid peroxidation product. J Cardiovasc Pharmacol. 13(6);1989:895–901.
16. Coles JC, Ahmed SN, Mehta HU, Kaufmann JCE. Role of free radical scavenger in protection of spinal cord during ischemia. Ann Thorac Surg. 41(5); 1986:551–556.
17. Lim KH, Connolly M, Rose D, Jacobowitz I, Cunningham JN. Prevention of reperfusion injury of the ischemic spinal cord: use of recombinant superoxide dismutase. Ann Thorac Surg. 42(3);1986:282–286.
18. Cohen EN. Distribution of local anesthetic agents in the neuraxis of the dog. Anesthesiology. 29(5);1968:1002–1005.
19. Symbas PN, Pfaender BS, Drucker MH, Lester JL, Gravanis MB, Zacharopoulos L. Cross-clamping of the descending aorta: hemodynamic and neurohumoral effects. J Thorac Cardiovasc Surg. 85(2);1983:300–305.
20. Pflug AE, Halter JB. Effect of spinal anesthesia on adrenergic tone and the neuroendocrine responses to surgical stress in humans. Anesthesiology 55(2);1981:120–126.
21. Crawford ES, Rubio PA. Reappraisal of adjuncts to avoid ischemia in the treatment of aneurysms of descending thoracic aorta. J Thorac Cardiovasc Surg. 66(5);1973:693–704.
22. DeBakey ME, McCollum CH, Graham JM. Surgical treatment of aneurysms of the descending thoracic aorta: long term results in five hundred patients. J Cardiovasc Surg. (*Torino*) 19(6);1978:571–1576.
23. Hollier LH. Protecting the brain and spinal cord. J Vasc Surg. 5(3);1987:524–528.
24. Najafi H, Javid H, Hunter J, Serry C, Monson D. Descending aortic aneurysmectomy without adjuncts to avoid ischemia. Ann Thorac Surg. 30(4); 1980:326–335.
25. Marini CP, Grubbs PE, Toporoff B, Woloszyn TT, Coons MS, Acinapura AJ, Cunningham JN. Effect of sodium nitroprusside on spinal cord perfusion and paraplegia during aortic cross-clamping. Ann Thorac Surg. 47(3);1989:379–383.
26. Gelman S, Reves JG, Fowler K, Samuelson PN, Lell WA, Smith LR. Regional blood flow during cross-clamping of the thoracic aorta and infusion of sodium nitroprusside. J Thorac Cardiovasc Surg. 85(2);1983:287–291.

27. McCullough JL, Hollier LH, Nugent M. Paraplegia after thoracic aortic occlusion: Influence of cerebrospinal fluid drainage. J Vasc Surg. 7(1);1988:153–60.
28. Marcus ML, Heistad DD, Ehrhardt JC, Abboud FM. Regulation of total and regional spinal cord blood flow. Circ Res. 41(1);1977:128–134.
29. Kennedy C, Des Rosiers MH, Jehle JW, Reivich M, Sharpe F, Sokoloff L. Mapping of functional neural pathways by autoradiographic survey of local metabolic rate with [^{14}C] deoxyglucose. Science 187(4179);1975:850–853.
30. Bromage PR. *Epidural Analgesia*. p 121–130. Philadelphia, WB Saunders, 1978.

18
Surgical Treatment of Lesions of the Thoracic Aorta in Patients with Multiorgan System Trauma

AUREL C. CERNAIANU, ANTHONY J. DELROSSI, JONATHAN H. CILLEY, JR., RICHARD K. SPENCE, and RUDOLPH C. CAMISHION

Abstract

Thirty-six patients admitted at our Level I Trauma Center with multiorgan system injuries and acute lesions of the thoracic aorta have been studied. Mean Injury Severity Score (ISS) was 27 ± 4. Four patients underwent Emergency Department (ED) resuscitative thoracotomy. One survived and fully recovered. The remaining patients underwent concomitant surgical repair of the aortic lesions and treatment of other multiorgan systems. The overall mortality rate was 17%. ISS for survivors was significantly lower than for nonsurvivors (23 ± 4 vs 35 ± 5, $p < 0.05$). The survivors arrived faster from the accident scene than nonsurvivors (45 ± 15 min vs 75 ± 10 min, $p < 0.05$). The time for survivors and nonsurvivors to arrive in the operating room for repair of the aortic injury was not different (13 ± 6 hrs. vs 14 ± 7, $p > 0.05$). All deaths occurred in the emergent or semiurgent groups. Four patients (two of whom presented with multiple lesions of the thoracic aorta) developed ischemia in distal organs. Two patients developed paralysis and two had lower limb spasticity. All discharged survivors were alive at 12 months follow-up. Sixty-four percent of the patients underwent aortic repair with "clamp/sew" technique. The rest were operated with either heparinized shunts or cardiopulmonary bypass (CPB). The type of surgical repair had no influence on the outcome of patients with single, uncomplicated aortic lesions, however, "clamp/sew" technique did not provide adequate protection when used for repair of multiple aortic tears.

Our study demonstrated that the outcome of surgical treatment of the traumatic aortic lesions of patients with multiorgan injury depends upon the time elapsed from the accident to the arrival to the ED, the severity of multiorgan system injury, the extent of thoracic aortic injury and appropriate timing of surgical repair of the aortic lesions and the surgical treatment of other injured organs.

Introduction

Traumatic lesions of the thoracic aorta produced by blunt thoracic trauma still carries a high mortality rate.[1] Approximately 98% of the survivors will die in

three to four months if the lesions are left untreated.[2] Since the highest mortality rate has been seen in patients with an increased number of injured systems, particularly in those with combined thoracoabdominal injury, the strategy for diagnosis and treatment is very challenging.[3] Despite attempts to standardize the treatment,[4] the management of the traumatic disruptions of the thoracic aorta remains controversial and the outcome unpredictable.[5] This chapter presents an update of our experience related to the surgical treatment of acute lesions of the thoracic aorta encountered in patients sustaining multiorgan system trauma.

Clinical Material and Methods

Of 12,180 patients admitted to our Level I Trauma Center between June 1985 and May 1992 with multiorgan system injuries, 36 patients have been diagnosed with an acute disruption of the thoracic aorta. There were 30 males and 6 females. The mean Injury Severity Score (ISS) was 22 ± 4. The demographic data and the mechanism of injury is presented in Table 18.1. An accelerating mechanism was present in 28% of the cases, a decelerating mechanism in 67%, and 5% were falls from heights. The majority of patients (64%) had been admitted to the ED directly from the accident scene. The remaining were transferred from other hospitals. All patients suffered from blunt chest trauma.

Five patients (14%) arrived in the ED from the accident scene in hemodynamic shock without vital signs. Although cardiopulmonary resuscitation was begun in all cases, two patients died during ED resuscitative thoracotomy. The other three patients underwent surgical repair of the thoracic aortic injury. Two patients survived and fully recovered; another underwent successful thoracotomy and repair of the thoracic disruption, however, died later from exsanguinating intra-abdominal injuries during laparotomy. The clinical status upon arrival in the ED is shown in Table 18.2. Eighty-six percent of the patients were hemodynamically stable on admission. Nineteen patients (53%) presented with chest pain, 10 (29%) presented with different degrees of respiratory distress, 3 (8%) had hemoptysis and 2(6%) hoarseness. Six patients (17%) presented without signs of

TABLE 18.1. Patient's characterisitcs on admission.

Variables	No. of patients (%)
Gender	
Male	30 (83%)
Female	6 (17%)
Type of Accident	
Vehicular	
Accelerating	10 (28%)
Decelerating	24 (67%)
Falls	2 (5%)
Admission	
From accident scene	23 (64%)
Transfer from other hospital	13 (36%)

TABLE 18.2. Clinical status upon arrival in the ED.

Variables	No. of patients (%)
Hemodynamic Status	
Hypovolemic shock	5 (14%)
Stable	31 (86%)
Clinical Manifestation[a]	
Chest pain	19 (53%)
Respiratory distress	10 (29%)
Hemoptysis	3 (8%)
Hoarseness	2 (6%)
Asymptomatic[b]	6 (17%)
Pseudocoarctation	0 (0%)

[a]Many patients presented with a combination of findings.
[b]In relation to the diagnosis of thoracic aortic injury.

intrathoracic injury. Most of the patients had a combination of findings. The classic findings of "pseudocoarctation syndrome" was not seen in our series. All hemodynamically stable patients underwent routine laboratory tests, and chest roentgenogram on admission. Supine chest roentgenogram was obtained in the majority of cases (86%). In only a few patients was an upright chest film possible (14%). The overall initial radiographic findings are shown in Table 18.3. Eighty percent of the patients had a widened mediastinum, 7% presented with massive hemothorax, 9% had visible pleural capping, and 14% presented with loss of the aortic knob. Three patients (8%) had a negative chest roentgenogram on admission. Twenty-nine patients (80%) demonstrated single or combination of chest radiograph findings defined as "high probability signs" for thoracic aortic lesion. Nine patients (25%) underwent emergent aortogram to define the extent of intrathoracic vascular injury within the first 10 hours from the injury. Eighteen patients (50%) underwent aortography within 24 to 48 hours post injury. Nine patients (25%) most of whom have been transferred from other community hospitals to our Level I Trauma Center had the diagnosis of thoracic aortic injury established after 48 hours post injury. The extent of multiorgan trauma is outlined in Table 18.4. The majority of patients presented with injuries to more than one organ system.

TABLE 18.3. Initial radiographic findings.*

Variables	No. of patients (%)
Widened mediastinum	29 (80%)
Massive hemothorax	6 (17%)
Pleural capping	3 (8%)
Loss of aortic knob	5 (14%)
No findings	3 (8%)

*Many patients presented with a combination of findings.

TABLE 18.4. Multiorgan system involvement.*

Variables	No. of patients (%)
CNS	5 (14%)
Cardiac/lung contusions	5 (14%)
Intraabdominal injuries	
Spleen	6 (17%)
Mesentery	3 (8%)
Liver	2 (5%)
Skeletal fractures	
Rib	17 (47%)
Pelvis/spine	6 (17%)
Extremities	16 (44%)

*Most of the patients presented with a combination of lesions.

Surgical Approach

Five patients arriving without vital signs at the Trauma Center underwent resuscitative thoracotomy. Two patients had been resuscitated and survived long term. Two patients died from massive intrathoracic exsanguination. One other patient underwent successful resuscitative thoracotomy and repair of the aortic lesion, however, continued to bleed profusely from intra-abdominal lesions. He died during laparotomy.

The timing of thoracotomy in relation to other associated surgical procedures is shown in Table 18.5. The extent of multiorgan system involvement dictated the indication and timing of surgical approach. Laparotomy was performed prior to thoracotomy if peritoneal lavage was positive in patients with thoracoabdominal trauma. Thoracotomy and repair of the contained aortic lesion took place after controlling the intraperitoneal bleeding. Orthopedic injuries were handled after thoracotomy and aortic repair in all patients. The specific surgical techniques used for repair of the thoracic aortic lesion and their outcome are summarized in Table 18.6.

A posterolateral thoracotomy was used in all patients as the surgical approach to the injured thoracic aorta. The surgical technique was selected independently by each surgeon depending upon his/her own clinical judgment, surgical expertise and intraoperative findings. Thirty-one patients (86%) had single lesions located "classically" below the origin of the subclavian artery. Three patients presented with multiple lesions of the thoracic aorta extending to the aortic arch. The clinical course of these patients and the anatomic findings have been described elsewhere.[5] The majority of cases were operated using the "clamp/sew" technique. The average aortic cross-clamp (AXC) time was 28 minutes. A heparinized Gott shunt placed between the aortic arch and either the distal aorta or the femoral artery was used in 5 patients, with a mean AXC time of 38 minutes. The use of shunts resulted in no complications. Cardiopulmonary bypass with total body heparinization was employed in one patient with multiple tears of

TABLE 18.5. Operative procedure, timing and outcome.

Procedure	Type of operation			
	Emergent	Semiurgent	Elective	Total
Thoractomy alone*	7 (2)	2	1	10 (2)
Thoracotomy/orthopedic repair	6	7 (1)	5	16 (1)
Thoracotomy/laparotomy	3 (1)	1 (1)	0	3 (2)
Thoracotomy/thoracotomy	2 (1)	3	0	7 (1)

Parentheses represent deaths. *Include those undergoing ED resuscitative thoracotomy.

the thoracic aorta in whom the "clamp/sew" technique has been initially considered. The AXC was 43 minutes. There were no heparin-induced complications.

Statistical analysis was performed with SPSS Plus (version 3.0). Paired *t*-test were used as appropriate. A p value less than 0.05 was considered statistical significant. Tables present data as mean ± standard deviation where appropriate.

Results

Of the 36 patients arriving in the ED, 6 patients (17%) died during their hospitalization (Tables 18.5 and 18.6). Of those, 2(5%) were ED deaths resulted from extensive intrathoracic exsanguination combined with multiorgan trauma. One patient underwent resuscitative thoracotomy and interposition of a Dacron graft. He later underwent surgical orthopedic repair and survived. Another patient who sustained a thoracoabdominal injury underwent successful repair of the thoracic aortic lesion, however, he died later during laparotomy for laceration of the spleen and liver (Table 18.6). Low cardiac output, complications of polytrauma and multiorgan failure resulted in early postoperative death in 3 other patients. Thirty patients (83%) fully recovered and were alive at 12 months follow-up. The determinants of outcome for the survivors from nonsurvivors are presented in

TABLE 18.6. Surgical technique for aortic repair and outcome.

Technique	No. of patients		Complications	
	Alive	Dead	Spinal cord injury	Renal failure
Resuscitative thoracotomy	2	3[a,b]	0	0
Clamp/sew	22	3	4[a]	1
Heparin shunt	5	0	0	0
CPB	1	0	0	0

[a]No aortic repair.

[b]Includes one patient who survived ED resuscitative thoracotomy but died during secondary laparotomy.

[c]Includes two patients with multiple tears of the thoracic aorta. CPB = Cardiopulmonary bypass.

TABLE 18.7. Determinants of outcome.

Variable	No. of patients (%) Alive (n = 30)	Dead (n = 6)	Significance
Time from accident to ED (min)	45 ± 15	75 ± 10	$p < 0.05$
Injury severity score	23 ± 4	35 ± 5	$p < 0.05$
No. of injured systems	2.6 ± 0.3	3.6 ± 0.6	$p < 0.05$
Time to make diagnosis (hrs)	13 ± 6	14 ± 7	NS
Time from diagnosis to OR (hrs)	7 ± 3	8 ± 2	NS

ED = Emergency Department, NS = not significant ($p < 0.05$)

Table 18.7. Survivors had significantly lower ISS scores than nonsurvivors (23 ± 4 vs 35 ± 5, $p < 0.05$) and fewer number of injured systems (2.4 ± 0.3 vs 3.6 ± 0.6, $p < 0.05$). The time to arrive in the ED from the accident scene was also shorter for survivors than for nonsurvivors (45 ± 15 min vs 75 ± 10 min, $p < 0.05$). In both groups the same amount of time was spent in making the definitive diagnosis of thoracic injury. Once this was obtained, the survivors arrived in the operating room in the same time as the nonsurvivors. Four patients with intact neurological status on admission presented at discharge with the sequelae of ischemia to the spinal cord. One patient also developed acute renal failure. All four patients underwent repair of the aortic lesion with the "clamp/sew" method (Table 18.6). The specific AXC time were 43 minutes, 55 minutes, 61 minutes, and 67 minutes, respectively. The AXC time of the last two patients was inadvertently prolonged due to the presence of multiple aortic tears. At follow-up, two patients had permanent paraplegia; two others remained with only minor spasticity of the lower limbs. All deaths had been encountered when the surgical repair of the traumatic lesion of the thoracic aorta took place as an emergent (four cases) or semiurgent operation (two cases).

Discussion

The diagnosis of intrathoracic vascular injury in patients with multisystem organ trauma remains a difficult task. Pate[6] and Spence et al[7] have emphasized that a high degree of individualization is necessary for each particular case. The sequence of establishing the diagnosis of aortic disruption and the therapeutic interventions in a series of patients with multiorgan system injuries and high probability signs for aortic disruption manifested by widened mediastinum has been investigated by Richardson et al.[8] The authors emphasized the importance of combining the accurate and expeditious diagnosis of such entities with the appropriate management of this type of patient.

The outcome of patients undergoing surgical treatment of traumatic lesions of the thoracic aorta has been related to the technique used for repair.

Clamp/sew,[9,10,11,12] primary repair,[13–15] heparin-bonded shunts[16–19] or the use of CPB,[6,20,21] have all been advocated as the method of choice for surgical management of such cases. Some techniques have been considered very controversial particularly when they appear to increase the morbidity. "Clamp/sew" technique is considered by some investigators as potentially adding a high degree of risk for ischemic sequalae when there are longer clamp times and no shunting procedures.[6,22] The use of heparin for anticoagulation during CPB may increase the risks of bleeding, particularly in polytraumatized patients[23–27] and the use of heparinized shunts is controversial in terms of it's protective effect to the distal organs and in terms of necessary surgical expertise in it's application.[28,29] Pharmacological manipulation for improvement of the spinal cord ischemic tolerance during surgical repair of the thoracic aorta has been investigated in clinical and/or experimental trials. The use of hypothermia,[30] sympathetic blockade,[31–33] experimental 21-aminosteroids,[34] and perfluorocarbons[35] have been shown to improve the ischemic tolerance and diminish the progression of pathologic changes seen at the spinal cord level during AXC. Somatosensory evoked potentials (SEP) have been used to monitor the changes induced in neuroconduction at the spinal cord level by AXC.[36] The application of SEP monitoring, however, is not always possible in the trauma setting. The results of our study demonstrated, however, that there are a series of factors that may influence the outcome of the surgical treatment of the lesions of the thoracic aorta in patients with multiorgan system trauma.

Other studies[4] have also demonstrated the difficulty of assessing polytraumatized with concomitant traumatic disruption of the thoracic aorta. The delay in making the diagnosis of disrupted thoracic aorta is generally the result of either the presence of multiorgan injuries, the application of resuscitative and stabilization procedures, or related to the transfer of the patients from other institutions. The outcome of our series are similar to those published by Pate in 1985.[6]

As seen in our series, patients with thoracic aortic injury may present with any hemodynamic state ranging from complete stability to cardiovascular collapse. Hemodynamic status on arrival in the ED is an important factor for survival. As mentioned previously, two patients of four undergoing ED resuscitative thoracotomy have survived and fully recovered. Patients who arrived faster at the ED from the accident scene had a better chance for survival. Patients sustaining extensive trauma may undergo more resuscitation at the injury scene resulting in delay in transportation to the Trauma Center. Even when intensive resuscitation measures are applied in the field, these patients may be transported to the ED with a compromised cardiorespiratory status which may impact on the short-term and long-term morbidity and mortality.

Our survivors confirmed the fact that the extent of multiorgan involvement may influence the survival rate of the patients with traumatic aortic injury.[4,6] The mortality rate increased when surgical repair of the aortic lesion was undertaken on an emergent or semi-urgent basis probably due to the impact of early extensive cardiorespiratory compromise. In our series, survivors and nonsurvivors spent the same amount of time in the establishment of the diagnosis of thoracic

aortic injuries, and arrived in the operating room in the same time frame. One may assume that, once the diagnosis of acute disruption of the thoracic aorta has been established, all measures are taken for rapid surgical intervention.

The presence of ischemic lesions produced by extensive AXC was influenced by the intrathoracic findings. Although the preoperative aortogram correctly diagnosed the existance of aortic injuries, it did not demonstrate their full extent, particularly when multiple aortic tears were present. There were no specific aortographic findings which might suggest the presence of multiple aortic tears. Because of the emergent character of establishing the diagnosis we could not use in our series any of the adjunctive techniques (computed tomograms, magnetic resonance imaging and ultrasonography) advocated by some investigators to complement the diagnostic arsenal for injury of the thoracic aorta.[37,38] Consequently, the real extent of the aortic injury had to be determined intraoperatively and the surgeon had to adjust the intraoperative management accordingly. Two of the three patients with multiple lesions in whom no heparinized shunts or CPB were used, needed prolonged AXC time for repair resulting in an increased morbidity.

Our series did not favor any of the surgical techniques for repair as the method of choice. At our center, we more frequently used the "clamp/sew" technique because it offered simplicity and rapidity necessary in the case of surgical intervention of the polytraumatized patient. It is, however, important to emphasize that "clamp/sew" technique may be inappropriate in the case where complicated intrathoracic findings are present. In this case, the availability of cardiovascular perfusion technology and personnel and/or the expertise to use shunts are important factors which may change the outcome.

References

1. Hartford JM, Fayer RL, Shaver TE: Transection of the thoracic aorta: Assessment of a trauma system. Am J Surg. 1986;151:224–29.
2. Avery JE, Hall DP, Adams JE, Headrick JR, Nipp RE: Traumatic rupture of the thoracic aorta. South Med J. 1979;72:1238–40.
3. Ross SE, Cernaianu AC: Epidemiology of thoracic injuries: Mechanism of injury and pathophysiology. Top Emerg Med. 1990;12(1):1–6.
4. Hanschen S, Snow NJ, Richardson JD: Thoracicaortic rupture in patients with multisystem injuries. South Med J. 1985;75(6):653–56.
5. DelRossi AJ, Cernaianu AC, Cilley JH Jr, Madden L, Spence RK: Multiple traumatic disruptions of the thoracic aorta. Chest 1990;97:1207–9.
6. Pate JW: Traumatic rupture of the aorta: Emergency operation. Ann Thorac Surg. 1985;39:531–37.
7. Spence RK, Alexander JB, Camishion RC: Blunt rupture of the thoracic great vessels. Trauma Q. 1990;6(6):27–41.
8. Richardson DJ, Wilson ME, Miller FB. The widened mediastinum: diagnostic and therapeutic priorities. Ann Surg. 1990;6:731–37.
9. Crawford ES, Walker HSJ, Saleh DA, Normann NA: Graft replacement of aneurysm in descending thoracic aorta: Results without bypass or shunting. Surgery 1981; 89:73–85.

10. Najafi H, Javid H, Hunter J, Serry C. Monson D: Descending aortic aneurysmectomy without adjuncts to avoid ischemia. Ann Thorac Surg. 1980;30:326–35.
11. Mattox KL, Holzman M, Pickard LR, Beall AC Jr, DeBakey MD: Clamp/repair: A safe technique for treatment of blunt injury to the descending thoracic aorta. Ann Thorac Surg. 1985;40:456–63.
12. Marvasti MA, Meyer JA, Ford BE, Parker FB Jr: Spinal cord ischemia following operation for traumatic aortic transection. Ann Thorac Surg. 1986;42:425–8.
13. Orringer MB, Krisch MM: Primary repair of acute traumatic aortic disruption. Ann Thorac Surg. 1983;35:672–75.
14. McBride LR, Tidik S, Stothert JC, Barner HB, Kaiser GC, Willman VL, et al: Primary repair of traumatic aortic disruption. Ann Thorac Surg. 1987;43:65–7.
15. Antunes JM: Acute traumatic rupture of the aorta: Repair by simple aortic cross-clamping. Ann Thorac Surg. 1987;44:257–59.
16. Donahoo JS, Brawley RK, Gott VL: The heparin-coated vascular shunt for thoracic aortic and great vessel procedures: A ten year experience. Ann Thorac Surg. 1977;23:507–13.
17. Gott VL: Heparinized shunts for thoracic vascular operations. Ann Thorac Surg. 1972;14:219–20.
18. Murray GF, Young WG: Thoracic aneurysmectomy utilizing direct left ventriculofemoral shunt (TDMAC-heparin) bypass. Ann Thorac Surg. 1976;21:26–29.
19. Wolfe WG, Kleinman LH, Wechsler AS, Sabiston DC Jr: Heparin-coated shunts for lesions of the descending thoracic aorta. Arch Surg. 1977;112:1481–87.
20. Cooley DA, DeBakey ME, Morris GC: Controlled extracorporeal circulation in surgical treatment of aortic aneurysm. Ann Surg. 1957;146:473–86.
21. Oliver HF Jr, Maher TD, Liebler GA, Park SB, Burkholder JA, Magovern GJ: Use of the BioMedicus centrifugal pump in traumatic tears of the thoracic aorta. Ann Thorac Surg. 1984;38:586–91.
22. Katz NM, Blackstone EH, Kirkland JW, Karp RB: Incremental risk factors for spinal cord injury following operation for acute traumatic aortic transection. J Thorac Cardiovasc Surg. 1981;81:669–74.
23. Rent R, Ertel N, Eisenstein R, Gewurz H: Complement activation by interaction of polyanions and polycations: Heparin-protamine induced consumption of complement. J Immunol. 1975;114:120–4.
24. Carreras LO: Thrombosis and thrombocytopenia induced by heparin. Scan J Haematol. 1980;36:64–80.
25. Chong BH, Pitney WAR, Castaldi PA: Heparin-induced thrombocytopenia: Association of thrombotic complications with heparin dependent IgG antibody that induces thromboxane synthesis and platelet aggregation. Lancet 1982;2:1246–8.
26. Olinger GN, Hussey CV, Olive JA, Malik MI: Cardiopulmonary bypass for patients with previously documented heparin-induced platelet aggregation. J Thorac Cardiovasc Surg. 1984;87:673–77.
27. Prater RVM, Oka Y, Hong Y, Tsubo T, Loubser PG, Masone R: Protamine induced circulatory changes. J Thorac Cardiovasc Surg. 1984;87:687–92.
28. Lawrence GH, Hessel EA, Sauvage LR, Krause AH: Results of the use of TDMAC-heparin shunt in the surgery of aneurysms of the descending thoracic aorta. J Thorac Cardiovasc Surg. 1977;73:393–98.
29. Crawford ES, Waler HS, Salch SA, Normann NA: Graft replacement of aneurysm in descending thoracic aorta: Results without bypass or shunting. Surgery 1981; 89:73–85.

30. White RJ, Albin MS, Harris LS, Yashon D: Spinal cord injury: Sequential morphology and hypothermic stailization. Surg Forum 1969;20:432–34.
31. Osterholm JL, Mathews GJ: Altered norepinephrine metabolism following experimental spinal cord injury. Part 2. Protection against traumatic spinal cord hemorrhagic necrosis by norepinephrine synthesis blockade with alpha methyl tyrosin. J Neurosurg. 1972;36:395–401.
32. Osterholm JL, Mathews GJ, Irvin JD, Calesnick B: Antinorepinephrine therapy against traumatic hemorrhagic necrosis of the spinal cord. Preliminary report. Clin Neurosurg. 1973;20:382–99.
33. Naftchi NE: Functional restoration of the traumatically injured spinal cord in cats by clonidine. Science 1982;217:1042–44.
34. Hall ED: Effects of the 21-aminosteroid U74006F on posttraumatic spinal cord ischemia in cats. J Neurosurg. 1988;68:462–65.
35. DelRossi AJ, Cernaianu AC, Cilley JH Jr, et al.: Preventive effect of Fluosol-DA for paraplegia encountered after surgical treatment of thoracicaorta. J Thorac Cardiovasc Surg. 1990;99:(4):665–69.
36. Cunningham JN Jr, Laschinger JC, Merkin HA, Nathan IM, Colvin S, Ransohoff J, et al: Measurement of spinal cord ischemia during operations on the thoracic aorta. Ann Surg. 1982;196:285–96.
37. Godwin JD, Korobkin M: Acute disease of the aorta: Diagnosis by computed tomography and ultrasonography. Radiol Clin N Am. 1983;21:551–54.
38. Boldman AP, Kotler MN, Scanlon MH, Ostrum B, Parameswara R, Parry WR: The complementary role of magnetic resonance imagining, Doppler echocardiography, and computed tomography in the diagnosis of dissecting thoracic aneurysms. Am Heart J. 1986;111:970–81.

19
Current Experience with Angioplasty and Stents in the Iliac Artery

MICHAEL C. DALSING, KAREN O. EHRMAN, DOLORES F. CIKRIT, STEPHEN G. LALKA, and ALAN P. SAWCHUK

Introduction

Percutaneous transluminal angioplasty (PTA) has become a part of our approach to treatment of patients with lower extremity vascular occlusive disease. Advances in balloon technology, the materials of construction, and the methods of delivery have all improved overall safety and efficacy of this technique.[1] Due to these technical improvements of PTA, little effort was directed to stents, another possible way of improving PTA results, originally suggested by Dotter in the 1960s.[2] Our task in this chapter is to review some of the salient events in the advancement of intravascular stents and to report on our early clinical experience with one such device.

Is There a Need?

The need for an internal support or endoskeleton to improve the results we hoped to achieve from PTA was recognized in two general areas. The first was to improve early technical results of PTA and the other to correct or prevent problems with restenosis.

Our initial experience with PTA at Indiana University demonstrated to us that improved results were a possibility. In our early experience with PTA, technical failures were observed in 20 of 132 (15.15%) PTA attempts and were evenly divided between the iliac/common femoral artery location (13.7%) and the superficial femoral/popliteal location (15.0%).[3] All were observed in cases of severe stenosis (8) or total occlusion (12). Four vessels were not accessible through a groin approach. In 11 cases the guidewire could not be passed. In five (25%) cases, dilation, although possible, was not successful. These were patients who might be helped by improvements over a simple PTA technique. Spencer and his coworkers have given us sufficient information to suggest that technical failures are a concern and that new technology may impact these results.[4] He reported that 31 of 282 (10.99%) attempted PTA interventions failed and that in at least eight of

these (25.8%), the lesion could be dilated but resulted in early failure. Our involvement in VA Cooperative Study 199 was also enlightening. In this report there were 20 (15.5% overall) early treatment failures. Ten of these lesions could not be crossed with a guidewire and therefore could not be helped by a supporting endoskeleton, but the remainder might have benefited by a device which could maintain a widely open channel.[5] These and similar experiences in other parts of the valvular system have defined early technical failures as an area which might be impacted by intraluminal support. The underlying cause of these failures may include acute thrombosis, dissection, elastic recoil, spasm or any combination of these events.[1,3,4,6]

Even when initially successful, recurrent symptoms from restenosis of PTA does occur. Our early experience with balloon angioplasty firmly implanted this concept into our minds.[3] Of the 23 patients followed for longer than 6 months and treated with angioplasty alone, 16 were initially successful but only eight (50%) maintained this level of improvement at 9.4 ± 4.5 months' follow-up. Twenty-five percent (four patients) had completely reverted to predilation hemodynamic measurements. Although several factors impact the results of PTA, the overall reported experience is a rapid initial decline in the success of PTA concentrated in the first 6 months to 1 year of follow-up with a more gradual decline thereafter.[3–5,7–9] Recurrent disease at the site of previous dilatation has been documented by several authors.[3,4,7] The reason for restenosis may be fibromuscular hyperplasia or typical atherosclerosis.[10] When the disease process progresses this rapidly, it may be logical to consider adjuvants to improve the result of PTA at the second attempt. This is where the intraluminal stent may come into play.

Initial Considerations

Before becoming involved in a new clinical endeavor, an investigation of the new technique is critical. Several intraluminal stent designs were under investigation in the early 1980s. Thermal expansion stents made of nitonol were constructed as a coil at high temperatures, then straightened at room temperature for ease of delivery. Despite antiplatelet medications, neointimal thickening tended to narrow the stented lumen at 8 weeks in a canine model.[11] Self-expanding stents of various designs were also being considered. Maass et al. reported on several steel alloy spiral springs.[12] A canine and calf animal experiment demonstrated its feasibility with no reported case of migration or acute thrombosis. The delivery system, however, was very cumbersome and the expansion factor only 1.2:1 to 5:1. Wright and his associates in 1985 reported on a percutaneously placed self-expanding stent constructed in a zigzag configuration of surgical stainless steel wire.[13] A canine experiment including 30 stents demonstrated only one incident of migration in the vena cava and no cases of acute thrombosis

or vascular erosion. The design of the stent did not allow complete contact with the arterial wall. Therefore, delayed or incomplete endothelialization of the stent in the dog model was reported.

In 1985 Julio Palmaz first reported a canine experiment using a balloon-expandable stent.[14] This was a continuous, woven, stainless steel wire design with cross-points silver-soldered to resist radial collapse. The wire thickness was 150–200 um with solder points not exceeding 450 um. It was expanded to its desired diameter of 6–10 mm by an angioplasty balloon to which the stent had been mounted. No anticoagulation was used. One stent thrombosis was reported as well as two partially clotted stents. Eight grafts did well for 1 to 8.5 weeks. A long-term study of this same stent was not any more promising.[15] Of 18 stents placed, two demonstrated acute thromboses and two occluded within 4 weeks. Six other stents demonstrated 30% or more luminal stenosis over a 34-week observation period. Finally in 1986, this same group reported their experience with a streamlined model.[16] It was constructed of continuous steel arranged in eight rows of staggered, offset slots that were 3.5 mm long. The stent was 1.67 mm in diameter and 15 mm in length when not in the expanded state. The

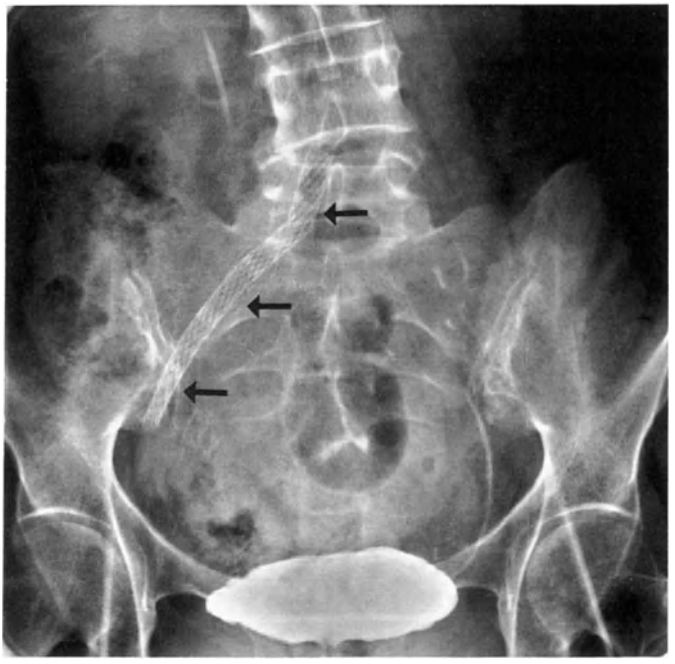


FIGURE 19.1. Plain radiograph of multiple overlapping stents (arrow) in the right common and external iliac artery. Note the radiologic clarity (visibility) which aids in proper placement of the stents.

results were impressive even without anticoagulation or antiplatelet drugs as adjuvant treatment. In 20 rabbits, atherosclerotic lesions were made by feeding the animals a hypercholesterolemic diet and by causing trauma to the intima with the use of an expanded balloon catheter. The cholesterol diet was maintained throughout the experiment trial. All stents remained patent up to 6 months after placement. No subintimal growth of plaque was observed in the stented area but was noted in portions of the artery proximal and distal to the stent. No angiographic or histologic narrowing of the plaque into the vessel lumen was noted at the area of stent placement. The stent was covered with neointimal 1 week after implantation. There was a continuous growth of this neointima from 36 ± 28 um at 1 week to 98 ± 36 um at 24 weeks, but the majority of the thickening was noted at 8 weeks with a much more gradual growth thereafter.

This stent design was also advantageous since it met several of the characteristics thought desirable for such a device. It was delivered and deployed by a standard PTA technique, and therefore, no difficult technical lessons would have to be learned. Furthermore, the device was visible by standard angiographic technique, and proper placement would be facilitated (Fig. 19.1). The stent design was versatile, such that the device could be expanded by the balloon to accommodate a vessel 8 to 12 mm in diameter. Biocompatibility and reliable expansion without migration were addressed in the experimental studies with excellent results.[16]

Materials and Methods

Technique

The clinical Palmaz stent is a slotted, seamless, stainless steel tube 3.2 mm in diameter and 3 cm long in the nonexpanded state (Fig. 19.2) (Johnson and Johnson, Intraventional Systems, Warren, NJ). The stent wall thickness is 0.12 mm. The stent can be optimally expanded to a diameter of 8 to 12 mm during deployment.

Figure 19.3 shows all the equipment required for stent placement. A standard Seldinger approach allows access to the common femoral artery, and a standard balloon angioplasty procedure is performed. This dilatation allows easier insertion of the introducer sheath if required at a later stage, confirms the ability of the lesion to accept a dilatation, and prevents unnecessary stent placement. The guidewire is always maintained in a position across the lesion to provide continued access to the target area. When a stent is required, the stent is mounted on an 8×30 mm angioplasty balloon catheter (PE Plus II, USCI, Billerica, MA) and further secured by a crimping tool after protecting the stent with a clear plastic crimping tube. A secure placement of the stent is confirmed by lack of movement on manual testing. It is now called the stent-balloon assembly. A 10F, 30-cm-

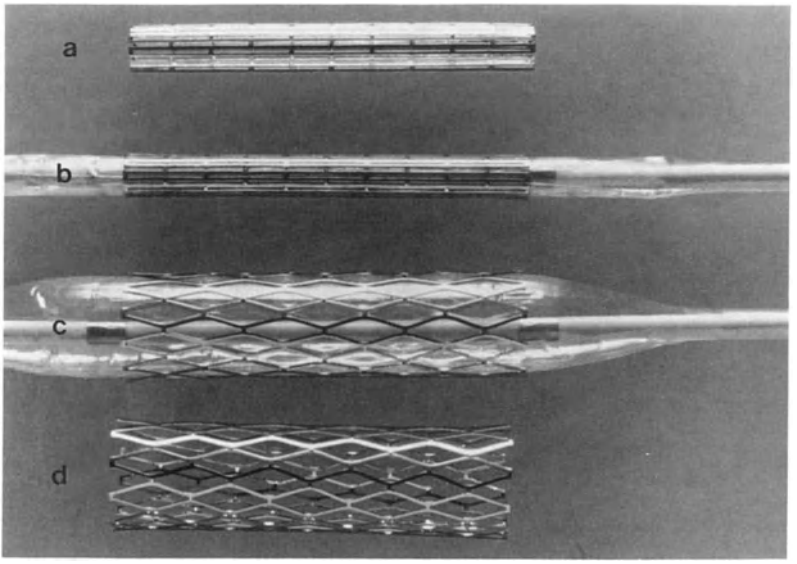


FIGURE 19.2. Photograph of the stent in an unexpanded (a) and then expanded state (d). Note the parallel stent design at rest which becomes a rectangular shape on expansion. A balloon angioplasty catheter lies in the unexpanded state (b), and with its dilatation (c) converts the stent to its open state.

long introducer and sheath with hemostatic valve (Cook, Bloomington, Indiana) is inserted over the guidewire into the vessel and advanced to a point proximal to the iliac occlusive target lesion. A stainless steel 5-cm-long open lumen introducer tube is inserted over the stent-balloon assembly to protect the stent while placing it into the hemostatic valve of the sheath. Once the stent part of the assembly is in the sheath, the introducer tube is pushed to the end of the balloon catheter to decrease blood loss. The stent-balloon assembly is advanced under fluoroscopic control to the target, and the sheath is retracted until the stent is fully exposed to the vessel lumen. This allows deployment of the stent while preventing entrapment in the sheath. The balloon is expanded by inflation pressures of 8 to 12 atmospheres. If a larger lumen is required, a balloon catheter change is made to allow stent dilation with a larger diameter balloon. After proper stent deployment, the balloon is deflated and gently rotated to detach it fully from the stent, and then is removed through the sheath.

The protocol for this study was approved by the Food and Drug Administration and our institutional review board. Informed consent was obtained from each patient after the experimental nature of the procedure was fully explained. Patient characteristics, such as age, medical illnesses, smoking status, as well as a noninvasive hemodynamic evaluation, were obtained. All patients were premedicated with 325 mg of aspirin the day

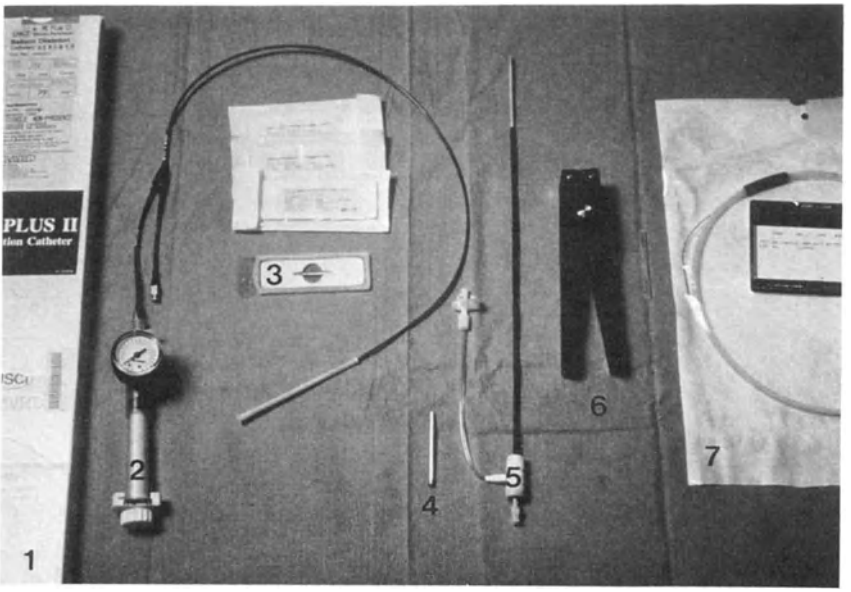


FIGURE 19.3. A picture of the equipment needed for a stent placement. (1) Bard PE Plus II 8 mm balloon catheter, (2) inflation device—any type, (3) Palmaz Balloon expandable stent in packaging with clear plastic crimping tube, (4) metal introducer tube, (5) Cook 10F 30-mm-sheath and introducer, (6) crimping tool (Johnson and Johnson), (7) 0.035 cm guidewire.

before intervention. Angiography confirmed iliac disease, and the intravascular pressure gradient was measured across the lesion. The patient received a 5000 unit bolus of heparin prior to any PTA intervention. Pressure measurements and angiography confirmed the result of the initial PTA intervention. Indications for stent use were: 1) an inadequate PTA result described as a >30% residual diameter stenosis, a pressure gradient >10 mmHg at rest or > a 15% change in the femoral/brachial systolic pressure index, or 2) for recurrent stenosis following a previous PTA. Contraindication to stent placement included: 1) extravasion of contrast material during initial PTA, 2) a markedly tortuous iliac artery, 3) extensive arterial calcification precluding initial PTA success, and 4) aneurysmal disease at the site of the proposed angioplasty. Relative contraindications were stenosis of the common femoral artery (< 5 mm in diameter) that would complicate the sheath placement, severe hypertension, impaired pain sensation, and poor distal arterial outflow. These relative contraindications could be managed by operative sheath placement, preintervention antihypertensive medications, cautious deployment, and anticoagulation or distal intervention to change the runoff status. If more than one stent was to be placed, the most cephalad was generally placed first, but this was not mandatory

(Fig. 19.1). Following stent placement, any residual gradient across the stent was measured by a standard angiographic catheter, which replaced the balloon angioplasty catheter. The sheath was removed and the puncture site controlled with manual compression. Patients were maintained on 325 mg aspirin for at least 3 months following the procedure.

Clinical follow-up consisted of a history, lower extremity vascular physical examination, and noninvasive Doppler examination that included at least an ankle/brachial index (ABI) at 2 weeks, and 1, 3, 6, 9, and 12 months following intervention. Thereafter, evaluation was performed on a twice-yearly basis. A plain pelvic radiograph as well as a repeat angiogram was scheduled for 3 to 6 months after stent placement.

Patients

From May 1987 through February 1990, 18 male and six female patients underwent iliac artery stent placement at the Indiana University Medical Center in Indianapolis. This period of review allows the opportunity for a significant follow-up even in the last patient studied and is an extension of an earlier experience reported by our group.[17]

Patients ranged in age from 44 to 76 years, with an average age of 61.8 ± 8.4 years. All were (10) or are currently (14) cigarette smokers. Other associated diseases included 15 patients with cardiovascular disease (six of whom required previous coronary artery intervention), six with cerebral vascular disease, five with chronic pulmonary disease, and four with chronic renal disease (serum creatinine >1.5). Sixteen patients were hypertensive and there were three diabetic patients only one of whom required insulin for control. Several patients had undergone a previous vascular surgical intervention. Nine patients had prior iliac PTA (three bilateral). There were three patients with a femorofemoral bypass graft, three patients with a femoropopliteal or femorotibial bypass graft, and one of these patients had both a femorofemoral and femoropopliteal bypass graft. Each of these grafts were patent and were in jeopardy due to inflow disease. One other patient had undergone a previous aortoiliac endarterectomy and a superficial femoral artery atherectomy.

There were 27 limbs requiring a stent procedure in this group of patients. The symptoms per limb were worsening or disabling claudication in 18, rest pain in seven, and ulceration or gangrene in two limbs. The preintervention average ABI was 0.63 ± 0.27. Seventeen stents were deployed in the right iliac artery and 10 in the left side. The location of stent placement was the common iliac artery in five, the external iliac artery in 10, and both the common and external iliac artery in 12. Thirteen vessels demonstrated calcification significant enough to be seen on plain radiographs but which did not preclude balloon angioplasty. The average percent stenosis of the target iliac lesion was $86.4 \pm 12.8\%$ (range 50% to 100%) with only one total occlusion. The mean diameter of the most stenotic area was

1.72 ± 1.28 mm as measured from the angiogram. The length of the vessel narrowing averaged 4.06 ± 3.92 centimeters (range 1 to 15 cm). The mean pressure gradient across the stenotic vessel was 31.3 ± 17.8 mmHg. Runoff status was generally normal and classified as per the Adhoc Committee on Reporting Standards[18] such that the resistance value was less than four for 22 limbs and $\geq$ four in five limbs. The indication for using a stent was restenosis following prior PTA in eight limbs, elastic recoil at the time of PTA in four, residual stenosis or a pressure gradient in 10, or severe dissection with resulting stenosis in five limbs. More than one stent (Fig. 19.1) was required for the desired effect in 18 cases for a total of 69 stents deployed (range 1–7, average; 2.6 ± 1.6).

Results

Following stent placement, the mean pressure gradient decreased to 1.07 ± 2.26 ($n = 15$) mmHg, and the immediate postintervention ABI increased to 0.88 ± 0.25 (Fig. 19.4). Two patients (8.3%), accounting for three limbs, died during the first month of follow-up. One patient died from a myocardial infarction 3 weeks after discharge, and the other patient died from a severe allergic reaction to the injected radiologic contrast material resulting in multi-system failure within 6 days. This latter patient required a thrombectomy of one limb because of hypotension and clotting of the stented area. The change in clinical status of the patients was evaluated according to criteria suggested by the Adhoc Committee on Reporting Standards[18] (Table 19.1). One of the patients in category zero experienced an unsuccessful stent procedure requiring occlusion of the iliac artery secondary to perforation and, shortly thereafter, a femorofemoral bypass graft for relief of symptoms. One patient required operative placement of the sixth stent under local anesthetic to resolve a dissection problem. I considered this patient successful at least in terms of the stenting procedure but a complication since the surgical exposure was not scheduled. Other short-term complications were two groin hematomas, one requiring a two-unit transfusion, but neither requiring an operative intervention. One of these patients also had the discovery of an arteriovenous fistula after the stenting procedure. There were, therefore, six local complications (22.2%) from the stenting procedure. All but two required operative correction. One patient required a planned operative exposure of the common femoral artery for stent placement and experienced no difficulties.

Within 1 month of stenting, three limbs in three patients underwent reconstruction of infrainguinal occlusive disease by profundoplasty in one and by in situ bypass grafting in the other two cases. These interventions were taken into account when reporting the resistance values for runoff since this is the runoff effecting the stent shortly after placement. There would be three more patients with a resistance valve ≥ 4 if we had not

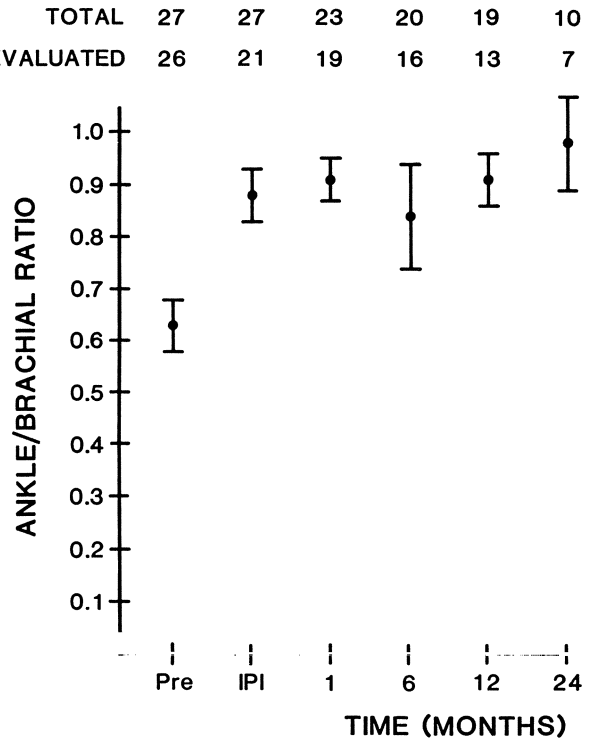


FIGURE 19.4. Average ankle/brachial index with standard error of the mean for preintervention (Pre), immediately postintervention (IPI) and at 1, 6, 12, and 24 months after stent placement. Total = total number of patients at risk, evaluated = those patients actually available for study at any given time. For the monthly follow-up values the numbers were obtained ± 3 months from the time indicated.

corrected for this fact. The average 1-month ABI with standard deviation is 0.91 ± 0.19. Seventeen patients had a 3 to 6-month postintervention angiogram. In no case was there migration of a stent demonstrated by pelvic radiograph. One patient demonstrated an angiographic stenosis of 62.5% in the area of stent placement which was successfully redilated. This patient was considered a failure. Two patients had a stenosis of <50%, one above and one below the stented area. One other patient experienced a >75% diameter stenosis directly below the stented area, refused further interventional angioplasty, and underwent a successful aortobifemoral bypass graft 6 months following the original stent placement. One other patient died of complications of pneumonia 3 months after the stenting procedure.

At 6 months after stent placement, 20 limbs in 17 patients were being followed. Sixteen limbs were available for study and demonstrated con-

TABLE 19.1. Change in overall clinical status per limb following stent placement.

Category	Description	# Limbs			
		0–1 Month	6 Months	12 Months	24 Months
+3	asymptomatic & ABI > 0.90	13	13	8	6
+2	1+ category & ABI > 0.10	5	2	3	
+1	1+ category or ABI > 0.10	2	1	1	1
0	no category & ABI ≤ 0.10	4		1	
Procedural mortality		3 (2 patients)			
Others at rick		0	4	6	3

tinued clinical and hemodynamic improvement in the majority of patients (Table 19.1, Fig. 19.4). One patient died at 8 months after stenting from a massive posterior circulation stroke. At 1 year, similar results to that seen at 6 months were noted. Between the first year and the next, one patient required a second stent placed below the previously placed stents and also a superficial femoral artery atherectomy to relieve his symptoms completely. One other patient was found to have a common iliac artery aneurysm above the stented segment. At 2 years' follow-up, only 10 limbs were at risk. Seven limbs were available for evaluation and showed maintenance of good clinical and hemodynamic results (Table 19.1, Fig. 19.4).

The total number of stent failures were three (11.1%). There were two early deaths (three limbs) less than 30 days after the procedure for a procedural mortality of 8.3%. Table 19.2 tabulates the specifics of each of these cases for clarification of risk factors. There were six local procedural complications for a complication rate of 22.2%, and four of these required surgical correction (14.8%). Interventions remote from the area of stent placement were required to maintain the desired clinical result in five instances.

The stenting procedure was successful in all four cases of elastic recoil, with a follow-up ranging from 8 to 24 months. Likewise, stent placement for dissection was routinely successful, with a follow-up ranging from 2 to 35 months. However, in one case originally scheduled for stent placement based on restenosis, there was also a significant dissection and this case failed. Eighty percent of the stent procedures performed because of an unacceptable hemodynamic PTA result and 87.5% of those performed for restenosis were successful as analyzed in this study. The observation period ranged from 3 to 28 months and 3 to 18 months respectively for these last two indications.

TABLE 19.2. Characteristics of patients with stent failure or perioperative mortality.

	Patient				
	13	14		19	24
Characteristics		R	L		
Male	x			x	x
Diabetics					x
HBP	x				x
Renal					
Indication*	CL	CL	CL	CL	CL
Location**	E	C&E	C&E	E	C&E
Length (cm)	2	6	6	6	4
% stenosis	80%	90%	90%	100%	85%
Side	L	R	L	R	R
Run-off***	O	O	O	4	O
Why stent****	GR	GR	GR	GR	RS
# stents	1	2	2	1	4
Complication	MI, open stent	contrast reaction (death) L clotted		perforation	RS**** at stent
Time	<4 weeks	6 days		immediate	3 months

*CL = claudication, RP = rest pain.
**E = External iliac artery, C = common iliac artery.
***See text.
****GR = gradient/residual stenosis, RS = restenosis.

Discussion

Iliac artery balloon angioplasty has become an accepted method of treating localized occlusive disease. However, the reported success rates at 1 year ranged between 50% and 93%.[4,5,7–8,19,20] This may be the result of immediately unsuccessful attempts at PTA (6–15%),[4,5,8,9,19] or of a recurrent disease process.[3–5,7–9] The introduction of an endoskeleton (stent) to mechanically maintain the opening made by balloon angioplasty seems logical in those patients otherwise destined for failure.

Elastic recoil was observed in four of our patients. The inherent elasticity of the arterial wall causes recoil after balloon angioplasty.[21] Eccentric lesions, where one wall is relatively devoid of plaque, may be prone to this problem because the balloon has essentially no supporting plaque present to help keep the lumen open after balloon deflation. The placement of a stent eliminates this problem because the internal support prevents radial collapse after the balloon is deflated. In each of our stented cases, the immediate result was a normal angiographic picture, removal of the pres-

sure gradient, improvement in the ABI, and clinical status that has been maintained for 8 to 24 months.

Angioplasty-induced dissection with residual stenosis may actually lead to occlusion of the lumen (Fig. 19.5). Significant dissection was the primary cause of stent placement in five of our cases, although it was the reason for the need of multiple stents in three other cases. These patients experienced diminished blood flow through the vessel and would probably have required surgery if stents were not available. Whenever dissection played a role in the need for stent placement, two or more stents were required for an adequate angiographic result and resolution of the pressure gradient. It is imperative, therefore, that the guidewire be maintained across the lesion or vascular access may be lost forever. All patients were treated successfully and were clinically improved for 2 to 35 months. One patient who had been initially scheduled for stent placement due to restenosis following prior PTA also experienced a long dissection and ultimately failed at 3 months. He is considered under restenosis problems. Stents may be especially successful in alleviating problems associated with PTA-induced dissection.[22] Intimal dissection is reported in approximately 2% of cases of iliac angioplasty.[8,23] On the basis of a recent stent experience, this complication may be more frequent than previously reported and possibly approaches 5%.[22]

Certainly, iliac lesions that fail PTA based upon hemodynamic measurements would not result in the desired clinical patient benefit. Ten of our patients are in this category as a reason for stent placement. A significant number of patients ($\approx$25%) treated with PTA alone still have a postprocedural gradient $\geq$10 mmHg at rest.[19,20] Elimination of the resting pressure gradient appears to be clinically beneficial.[19,24,25] Since each of the 10 patients in which stents were required for resolution of residual disease would have been considered PTA failures, an 80% success rate seems reasonable. The failures were one case in which occlusion occurred after a hypotensive event (patient died), and one case of total occlusion with perforation.

The final reason for stent placement was to prevent a second stenosis after a previous PTA failure. Restenosis at the site of prior iliac PTA is reported in 3.3% to at least 13.8% of cases.[5,9] The cause may be progressive atherosclerotic disease or fibromuscular proliferation.[10] The majority of restenosis cases occur in the first 6 months to 1 year after PTA.[3–5,7–9] Eight of our patients were treated for this reason and one failed (12.5%). The one failure was a true restenosis in the area of stenting. Certainly, these lesions treated by stenting are prone to restenosis after PTA since they have already demonstrated this propensity. An 87.5% success rate may be acceptable in this group of patients. It is postulated that the stent may decrease problems with restenosis by forming a relatively smooth surface that prevents the proliferative injury repair.[25] However, the build-up of hyperplastic tissue noted in an initial experimental study[16] would sug-

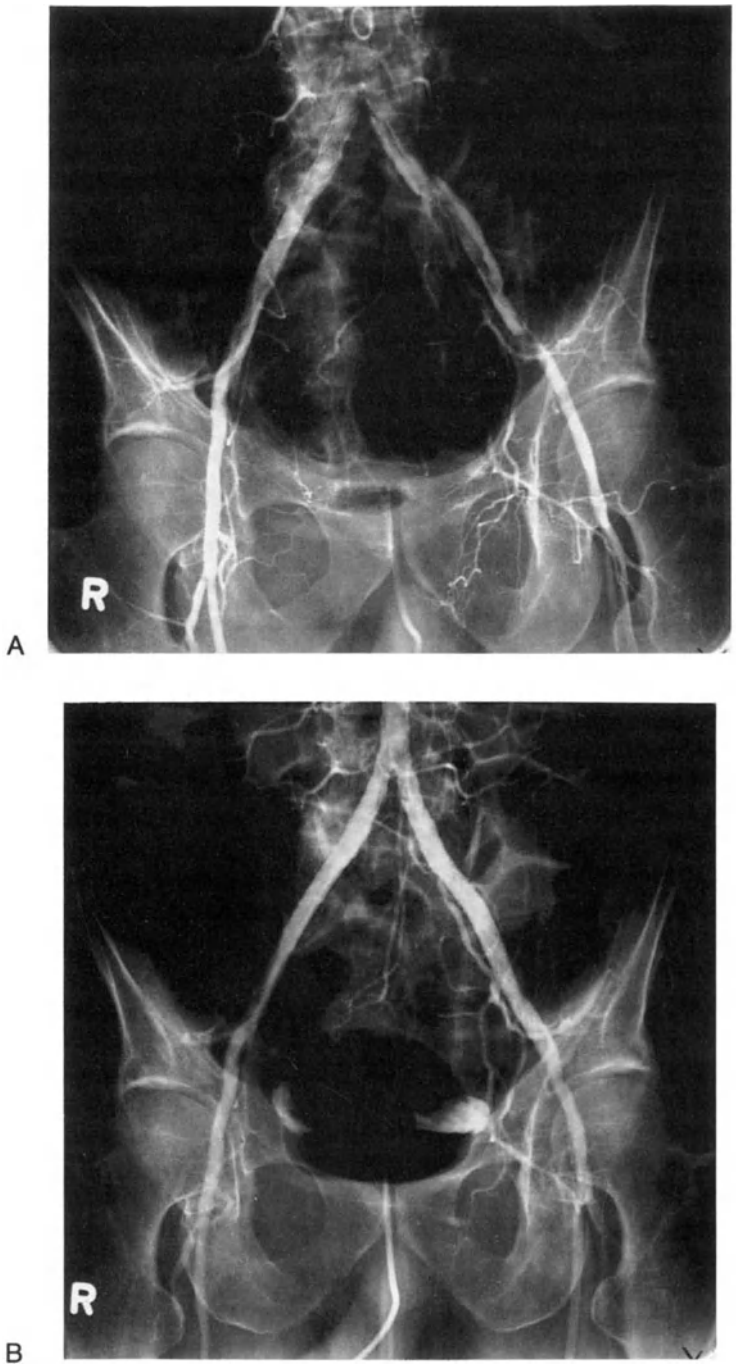


FIGURE 19.5. (A) Angiogram of a left iliac artery dissection after attempted PTA. Note almost total occlusion of the vessel and the obvious spiral dissection. (B) Resolution of the dissection after the placement of four stents.

gest the possibility of stent area restenosis and was actually noted in our patient.

Total iliac occlusions have a less successful patency rate after PTA than stenotic iliac artery lesions.[7] Colapinto described finding the true lumen of the totally occluded artery as the most difficult of all angioplasty procedures, with a 22% technical failure rate, but was able to report a 78% four-year patency rate after an initially successful PTA.[26] Two investigators have found the early results of stenting to be very favorable in the totally occluded artery.[27,28] Our one case of perforation is the only completely occluded iliac artery treated by us with a stent and cannot shed light on this subject.

On review of our three stent failures (Table 19.2), no factor, such as a particular preoperative risk factor, the indication for the procedure, or the specifics of the stenting procedure itself (length of lesion or number of stents required) gives a clear picture as to who would be likely to fail. More patients would have to be evaluated to clarify those cases in which stents may be more detrimental than beneficial. The four factors found by Johnston and coworkers to be predictive of PTA failures (indication, location, runoff, and stenosis versus occlusion)[7] were not obvious predictive factors when stents were used in our small series. In a larger series evaluating the Palmaz stent, it was also commented that the influence of risk factors, runoff, and the nature of the lesion would be evaluated only after sufficient data became available.[29]

Complications occurred in six (22.2%) of our patients. Four required aggressive intervention to correct the problem (14.8%). If all complications are included, as it is in this study, 10% to 31% of standard iliac PTA interventions will experience a complication.[5,19,23] In our study 1.8% to 6% required treatment of a more aggressive nature than merely observation to alleviate the problem.[19,23] Our rate of complications does not seem dramatically different from routine PTA interventions. With the larger sheath required and the need for multiple balloon exchanges, the requirement for operative intervention to correct complications may actually be greater with stent placement than in a routine PTA series. It is too early to make a definite statement in this regard. One patient experienced two complications—the arteriovenous fistula and the hematoma requiring a two-unit transfusion. This patient is a small female with a common femoral artery approximately 5 mm in diameter and similar small iliac vessels (approximately 7 mm in diameter), which might partially explain why she experienced these problems. Surgical interventions in the aortoiliac system also report operative complications in the 4% to 30% range.[30–33] Major complications were reported in 9 to 13% of cases.[31,32] The early results of stenting are not prohibitive based on this comparison but many suggest a higher rate of major complications. However, in a larger series of stent patients, the overall complication rate has decreased to 11.7%.[29]

The two procedural deaths reflect both the systemic nature of our

patients' vascular disease, resulting in a myocardial infarction, and the risks of any arterial contrast injection, an allergic reaction. Obviously, no vascular intervention is without risk. The patient suffering a myocardial infarction did not have symptoms prior to intervention. The patient with the contrast reaction had no previous history of problems with allergic reactions to radiologic contrast agents. Obviously, the actual stent placement was not the cause of mortality in either case, but the decision to do the procedure in these patients does warrant the classification of a procedural mortality. Further experience will be required to determine if this 8.3% risk is real or an overestimation of the procedural risk. Certainly, the ultimate procedural risk should be less than the reported 3% surgical risk and <1% PTA risk for the correction of aortoiliac occlusive disease.[30–33,4,5,7,19]

The overall results demonstrate a definite clinical benefit (advancement to category 2 or 3) in patients at risk of 75%, 58%, and 60% at 6, 12, and 24 months after stent placement. These results in patients essentially failing PTA prior to stent placement are quite comparable to the 83%, 78%, and 74% success reported in a recent experience with iliac PTA.[5] It is also similar to the results of Johnstons' larger experience with PTA.[7] It may approach the results of some methods of surgical intervention, but the aortobifemoral graft still appears to be the "gold standard" for long-term patency.[31,32]

Conclusion

This early experience with the Palmaz expandable intraluminal stent suggests that it can be valuable in salvaging PTA cases that might otherwise be initial failures. PTA-induced dissections, elastic recoil, and technically unsuccessful PTA attempts can be corrected with acceptable results. PTA restenosis may also be favorably addressed by the use of stents, but longer follow-up is required to fully clarify this issue. There is significant morbidity and mortality reported in this initial learning experience with stents. These problems may improve with experience and technological advances, but presently these factors must be considered in the overall risk/benefit decision for any given patient. In the final analysis only a larger experience and more extensive follow-up will solidify the role of the Palmaz stent in the treatment of iliac artery occlusive disease.

References

1. Becker GJ, Katzen BT, Dake MD. Noncoronary Angioplasty. Radiology 1989; 170:921–940.
2. Dotter CT. Transluminally-placed Coilspring Endarterial Tube Grafts. Invest Radiology 1969;4:329–332.

3. Glover JL, Bendick PJ, Dilley RS, et al. Efficacy of balloon catheter dilatation for lower extremity atherosclerosis. Surgery 1982;91:560–565.
4. Spence RK, Freiman DB, Gatenby R, et al. Long-Term Results of Transluminal Angioplasty of the Iliac and Femoral Arteries. Arch Surg. 1981;116:1377–1386.
5. Wilson SE, Wolf GL, Cross AP. Percutaneous transluminal angioplasty versus operation for peripheral arteriosclerosis. J Vasc Surg. 1989;9:1–9.
6. Lu C, Zarins CK, Yang C, et al. Percutaneous Transluminal Angioplasty for Limb Salvage. Radiology 1982;142:337–341.
7. Johnston KW, Rae M, Hogg-Johnston SA, et al. 5-Year Results of a Prospective Study of Percutaneous Transluminal Angioplasty. Ann Surg. 1987;4:403–413.
8. Stokes KR, Strunk HM, Campbell DR, et al. Five-Year Results of Iliac and Femoropopliteal Angioplasty in Diabetic Patients. Radiology 1990;174:977–982.
9. Cambria RP, Faust G, Gusberg R, et al. Percutaneous Angioplasty for Peripheral Arterial Occlusive Disease. Arch Surg. 1987;122:238–287.
10. Simpsom JB, Selmon MR, Robertson GC, et al. Transluminal Atherectomy for Occlusive Peripheral Vascular Disease. Am J Cardiol. 1988;61:96G–101G.
11. Cragg AH, Lund G, Rysavy JA, et al. Percutaneous Arterial Grafting. Radiology 1984;150:45–49.
12. Maass D, Zollikofer CL, Largiader F, et al. Radiological Follow-up of Transluminally Inserted Vascular Endoprostheses: An Experimental Study Using Expanding Spirals. Radiology 1984;152:659–663.
13. Wright KC, Wallace S, Charnasangavej C, et al. Percutaneous Endovascular Stents: An Experimental Evaluation. Radiology 1985;156:69–72.
14. Palmaz JC, Sibbitt RR, Reuter SR, et al. Expandable Intraluminal Graft: A Preliminary Study. Radiology 1985;156:73–77.
15. Palmaz JC, Sibbitt RR, Tio FO, et al. Expandable Intraluminal Vascular Graft: A Feasibility Study. Surgery 1986;99:199–205.
16. Palmaz JC, Windeler SA, Garcia F, et al. Atherosclerotic Rabbit Aortas: Expandable Intraluminal Grafting. Radiology 1986;160:723–726.
17. Cikrit DF, Becker GJ, Dalsing MC, et al. Early Experience with the Palmaz Expandable Intraluminal Stent in Iliac Artery Stenosis. Ann Vasc Surg. 1991;5:150–155.
18. Rutherford RB, Flanigan DP, Gupta SK, et al. Suggested Standards for Reports Dealing with Lower Extremity Ischemia. J Vasc Surg. 1986;4:80–94.
19. Kadir S, White RI, Kaufman SL, et al. Long-Term Results of Aortoiliac Angioplasty. Surgery 1983;94:10–14.
20. Walden R, Siegel Y, Rubinstein ZJ, et al. Percutaneous Transluminal Angioplasty. J Vasc Surg. 1986;3:583–590.
21. LeVeen RF, Wolf GL, Turco MA. Morphometric Changes in Normal Arteries and Those Undergoing Transluminal Angioplasty. Invest Radiol. 1983;18:63–67.
22. Becker GJ, Palmaz JC, Rees CR, et al. Angioplasty-Induced Dissections in Human Iliac Arteries: Management with Palmaz Balloon-Expandable Intraluminal Stents. Radiology 1990;176:31–38.
23. Gardiner GA, Meyerovitz MF, Stokes KR, et al. Complications of Transluminal Angioplasty. Radiology 1986;159:201–208.
24. Flanigan DP, Ryan TJ, Williams LR, et al. Aortofemoral or Femoropopliteal

Revascularization? A Prospective Evaluation of the Papaverine Test. J Vasc Surg. 1984;1:215–223.
25. Palmaz JC, Richter GM, Noeldge G, et al. Intraluminal Stents in Atherosclerotic Iliac Artery Stenosis: Preliminary Report of a Multicenter Study. Radiology 1988;168:727–731.
26. Colapinto RF, Stronell RD, Johnston WK. Transluminal Angioplasty of Complete Iliac Obstructions. AJR 1986;146:859–862.
27. Gunther RW, Vorwerk D, Bohndorf K, et al. Iliac and Femoral Artery Stenoses and Occlusions: Treatment with Intravascular Stents. Radiology 1989;172:725–730.
28. Rees CR, Palmaz JC, Garcia O, et al. Angioplasty and Stenting of Completely Occluded Iliac Arteries. Radiology 1989;172:953–959.
29. Palmaz JC, Garcia OJ, Schatz RA, et al. Placement of Balloon-Expandable Intraluminal Stents in Iliac Arteries: First 171 Procedures. Radiology 1990;174:969–975.
30. Piotrowski JJ, Pearce WH, Jones DN, et al. Aortobifemoral Bypass: The Operation of Choice for Unilateral Iliac Occlusion? J Vasc Surg. 1988;8:211–218.
31. Szilagyi DE, Elliott JP, Smith RF, et al. A Thirty-Year Survey of the Reconstructive Surgical Treatment of Aortoiliac Occlusive Disease. J Vasc Surg. 1986;3:421–436.
32. Bunt TJ. Aortic Reconstruction vs Extra-Anatomic Bypass and Angioplasty. Arch Surg. 1986;121:1166–1171.
33. Couch NP, Clowes AW, Whittemore AD, et al. The Iliac-Origin Arterial Graft: A Useful Alternative for Iliac Occlusive Disease. Surgery 1985;97:83–87.

20
Percutaneous Endovascular Therapy in a Surgeon's Practice

FRANK J. CRIADO

Percutaneous endovascular therapy will soon enter its third decade of clinical practice. Technological breakthroughs in catheter design and growing expertise and experience with endovascular manipulations have resulted in the advent of a new frontier in vascular therapy. Balloon angioplasty and, more recently, atherectomy, stenting and thrombolysis offer exciting alternatives to conventional reconstructive surgery in the treatment of occlusive vascular lesions. Acknowledged advantages include, mainly: a) percutaneous approach, b) use of local anesthesia, c) short hospital stay and lower cost, d) little if any postprocedure disability, and e) easy repeatability. These characteristics no doubt have contributed much to the rapid acceptance and growth of interventional practices. On the other hand, it is paramount to realize that important limitations and dilemmas continue to exist, principally: 1) uncertain durability for some procedures and devices as a result of the lack of prolonged follow-up studies and properly designed trials; 2) the problem of rethrombosis and restenosis which occur frequently, especially during the first 6 months following percutaneous recanalization, and 3) uncertain clinical indications for the performance of these procedures.

Interventional percutaneous procedures have been said to have the capability of either replacing or improving vascular reconstructive surgery in the treatment of arterial blockage. This is, in our judgement, a very common and significant misconception. By and large, catheter-based technology is best at recanalizing or dilating mild discrete arterial plaques for which surgery would seldom be considered. When dealing with more extensive occlusive lesions, the results both midterm and long-term are so poor that its application may not be warranted outside a trial setting. One additional consideration in this context relates to clinical indications for intervention: the mild/favorable lesions mentioned above are only infrequently symptomatic enough to warrant invasive therapy.

Surgeons' attitudes regarding interventional therapy have varied from complete condemnation and obstruction to healthy skepticism and critique. The introduction of laser technology, especially the hot-tip device,

promoted other reactions and insecurities among some vascular surgeons. They feared that nonsurgical interventionists would impinge on their vascular practices as a growing number of cases would be recanalized percutaneously without surgery. Some of them took it one step further by acquiring a vascular laser and calling themselves interventionists. They had no clear understanding of the subspecialty and its requirements. Their fate and success rates were no different from those of the device itself.

We are now in a better position to understand this whole scenario of endovascular therapy. While some of the procedures and devices may be unproven and marginally useful, it is clear the dedicated vascular surgeon of the future will not be able to afford ignoring current developments. Aside from the procedures designed to treat arterial stenoses and occlusion, there are many other disorders and methods about to be greatly influenced (or "overtaken") by catheter-based technology. Most prominently: aortic aneurysms, aortic dissections, intraluminal bypasses, and percutaneous clot management in acute and chronic situations, to name but a few. Ongoing experimentation and early applications promise to bring them into the clinical interventional arena over the next few years. The now-widespread acceptance of percutaneous insertion for IVC filters is another example of the continuing shift from the operating room to the angio suite and catheterization laboratory.

Should vascular surgeons be concerned? Definitely! Unless we no longer feel excited about progress in vascular therapeutics, the inescapable conclusion is that we must follow these developments very closely and seriously consider whether catheter technology should not be part of our armamentarium. When vascular surgeons ponder the wisdom and feasibility of becoming interventionalists, they should focus on two major obstacles in this pursuit:

1. The lack of catheter skills and training. This is a difficult art which cannot be learned in a 2-day tutorial. It involves much observation and practice. The ideal solution would be the incorporation of catheter skills training into residency and fellowship programs. This will obviously take many years to become reality.
2. The lack of adequate x-ray/angiographic capabilities in the O.R. Surgeons are taking a long time to see that present-day vascular therapy and surgery require the kind of finesse and procedural imaging capabilities our nonsurgical colleagues have enjoyed for years. Our own solution was the installation of a unique dedicated system consisting of a ceiling-mounted C-arm and special carbon-fiber operating table (International Surgical Systems, Phoenix, Arizona). The creation of a cohesive team (which should include an experienced angioradiologic technician) is the final significant component leading to maximal efficiency and capability.

Percutaneous endovascular therapy constitutes approximately 18% of our current practice with lower extremity occlusive disease. Superficial

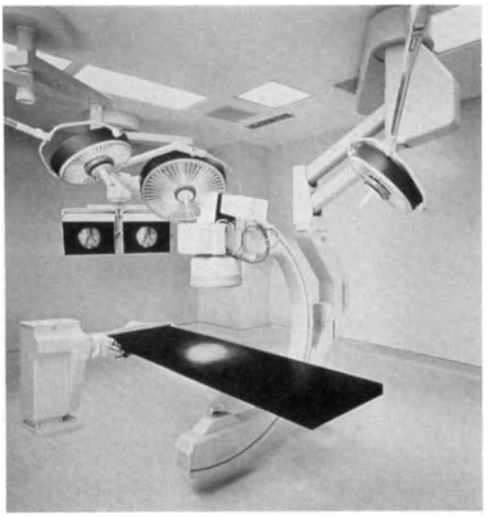


FIGURE 20.1. Endovascular Suite (by International Surgical Systems, Inc., Phoenix, Arizona).

femoral/popliteal artery procedures are 65% of the total. Eight percent are tibial/peroneal, and this is the subset which continues to grow the most. Sixty-five percent of the lesions are stenotic. Initial success rate is over 90%, but our incidence of restenosis/reocclusion overall is 32% at 6 months. Complications are mostly access-related, with groin hematoma leading the group at 20%; only 1% of these require surgical evacuation, though. These two figures of initial success and complication rates are critical parameters to gauge one's performance with percutaneous therapy. Balloon angioplasty, with or without stent, and directional (Simpson) atherectomy are employed in the majority of instances. Catheter thrombolysis with urokinase as primary therapy, adjunctive to other methods or to treat complications, is an increasingly utilized method.

Catheter interventional capabilities have also been found to be important in the areas of percutaneous IVC filter insertions and venous access for hemodialysis and other purposes. Furthermore, the availability of high-quality angiography in the vascular surgical suite has been extremely advantageous for the performance of exacting and rapid completion angiography in cases of distal vein bypasses.

In conclusion, percutaneous endovascular therapy and the required catheter and imaging capabilities are likely to be prominent in the armamentarium of the vascular surgeon of the future. Proper training, dedication, team approach, and adequate caseload are paramount in attaining success in this subspecialty. The vascular O.R. of tomorrow will resemble a catheterization laboratory as vascular surgeons and angiologists become more sophisticated and skilled.

21
Strategies and Techniques for Peripheral Laser Angioplasty: 1991

EDWARD B. DIETHRICH

Introduction

As we enter the fifth year of clinically applied peripheral laser angioplasty, we can no longer deny the impact on vascular surgery generated by development of this intraluminal technique and its analogs. Indeed, endovascular technology has grown at such an astounding pace in recent years that vascular surgeons now have access to an array of new percutaneous and/or intraoperative devices that can facilitate treatment of vascular occlusive disease.

As this interventional armamentarium was developing in the late '80s, it became incumbent upon vascular surgeons to acquire the requisite ancillary skills (e.g., percutaneous access techniques, intraoperative arteriography) for effective use of this instrumentation.[1–6] Now, with a variety of interventional devices in clinical use and our increased knowledge of plaque morphology, lesion characterization, individual device efficacy, procedural assessment, and adverse sequelae, the decision process for treatment selection has become far more complex than it was just 18 months ago.

Today, the vascular surgeon can evaluate atherosclerotic disease with greatly enhanced accuracy, deriving far more specific information about an obstruction than was ever required for classical vascular reconstruction. Options for treating this disease have expanded from the old progressive triad of exercise, medication, and bypass surgery, to include primary therapy using balloon dilation, laser angioplasty, thrombolytic therapy, atherectomy, and stents, most with several devices from which to select.

All these options and a more complex diagnostic database require coordination if these new resources are to be used to their fullest advantage for maximum revascularization. *Strategies* for selecting endovascular interventions are coming to the fore as a vital component of vascular disease treatment.

As a contribution to this new phase in endovascular surgery's evolution, this chapter will not only review in detail current techniques for peripheral

laser-assisted angioplasty, but also provide strategies for synchronous use of other marketed and emerging interventional therapies, basing selection on lesion pathology and anticipated outcome.

Vascular Laser Therapy in 1991: An Overview

When intraluminal recanalization was brought into prominence in the late '70s by Gruentzig's coronary balloon catheter,[7] it seemed only logical to many investigators that laser energy delivered through a similar intraluminal apparatus could become the endovascular "scalpel" of the future, elegantly and selectively removing unwanted tissue rather than merely compressing it. Beguiled by the appeal of this "magic wand" concept, researchers raced to overcome the complications plaguing direct laser irradiation. Their impatience led to the premature promotion of "hot-tip" technology as a viable and safe recanalization technique.

Unfortunately, this initial enthusiasm for laser angioplasty waned as it became obvious that thermal technology could not meet clinical expectations.[8,9] Although the procedural success rate for the hot-tip probe in the peripheral system was acceptable (about 79% in most series), suspicions regarding thermal injury potential and disappointing mid- and long-term recurrence rates reopened the original developmental course toward direct laser application and the goal of precise plaque ablation.

Today, our peripheral laser angioplasty program bears little resemblance to the one we inaugurated four years ago.[10–12] At that time we were concerned with establishing clinical efficacy, so all symptomatic patients with documented significant atherosclerotic lesions in the lower extremities were offered laser angioplasty as initial therapy.

Now, we still provide our patients with the advantages of less-invasive endovascular technology, but each lesion is better assessed than ever before with duplex scanning, intravascular ultrasound (IVUS) and/or angioscopy. By more accurately defining the pathology, we can select from an array of intraluminal techniques and devices to use as sole endovascular therapy or as an adjunct to classical revascularization techniques, a distinct advantage for the vascular surgeon.

At present, there are many instances in which lasers are recognized to be of little benefit and, in some circumstances, even deleterious. The infrapopliteal arteries, for example, showed such a high rate of recurrence after laser-assisted angioplasty with the thermal system that for some time lasing of these smaller arteries was abandoned. A new protocol has been established utilizing small over-the-wire catheters powered either by excimer or holmium lasers, and our early results have been encouraging.

Stenoses in general do not benefit greatly from laser application with the current technology. In large arteries particularly, the small catheters (greatest diameter now available is 3 mm) produce little ablative effect

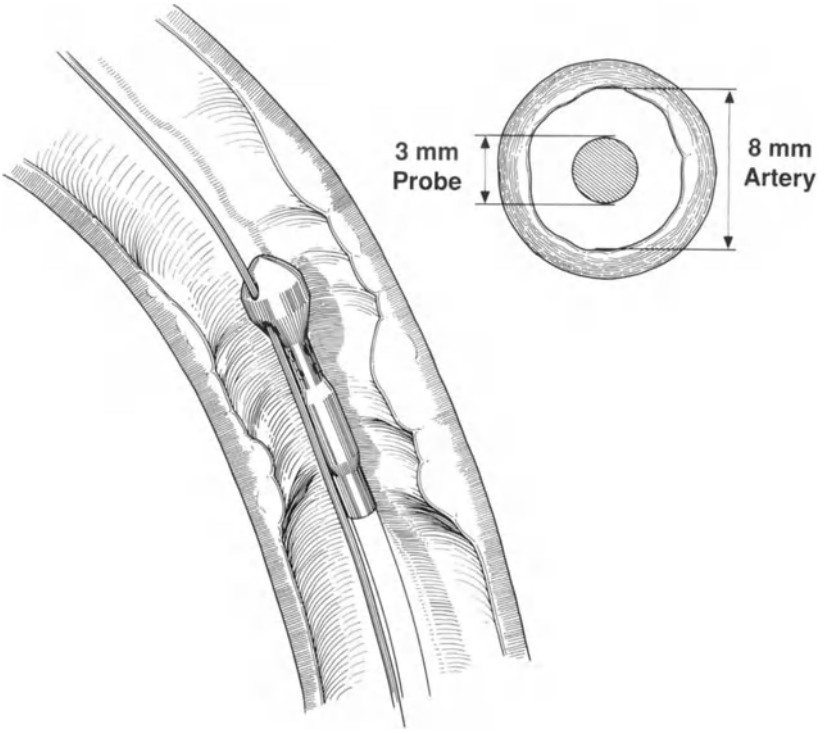


FIGURE 21.1. In large-bore arteries, even the largest 3.0 mm laser catheter passed coaxially cannot ablate a significant volume of atheromatous material.

(Fig. 21.1). In the presence of dense calcium, debulking is further inhibited and the procedural failure rate is extremely high. Currently, there is no laser under clinical investigation that is effective in all calcified lesions, to my knowledge.

Lesion contour is also a significant confounding factor in laser therapy. If a stenosis is eccentric along most of its course, the probe will quite often slide over the plaque, going up and down like a roller coaster within the artery, reducing its contact with the plaque and its ablative efficiency (Fig. 21.2).

Even in concentric lesions, there is a certain amount of Dottering evident with all laser catheters, which contributes to the "angioplasty effect" and reduces ablation. This failure to remove significant portions of atherosclerotic material continues as one of the main drawbacks to laser angioplasty, despite the introduction of new and more powerful laser sources.

It is in the occluded arteries that today's laser technology is most effective, recanalizing wire-resistant lesions and reducing the volume of atheromatous material to some degree. However, suboptimal ablation is still a

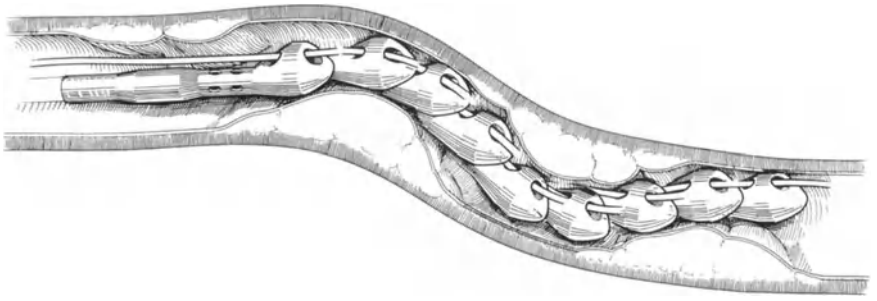


FIGURE 21.2. Lesion contour can affect laser efficiency. If a lesion is eccentric along most of its course, a probe will quite often slide over the lesion, going up and down like a roller coaster within the artery, reducing contact with the plaque and ablative efficiency.

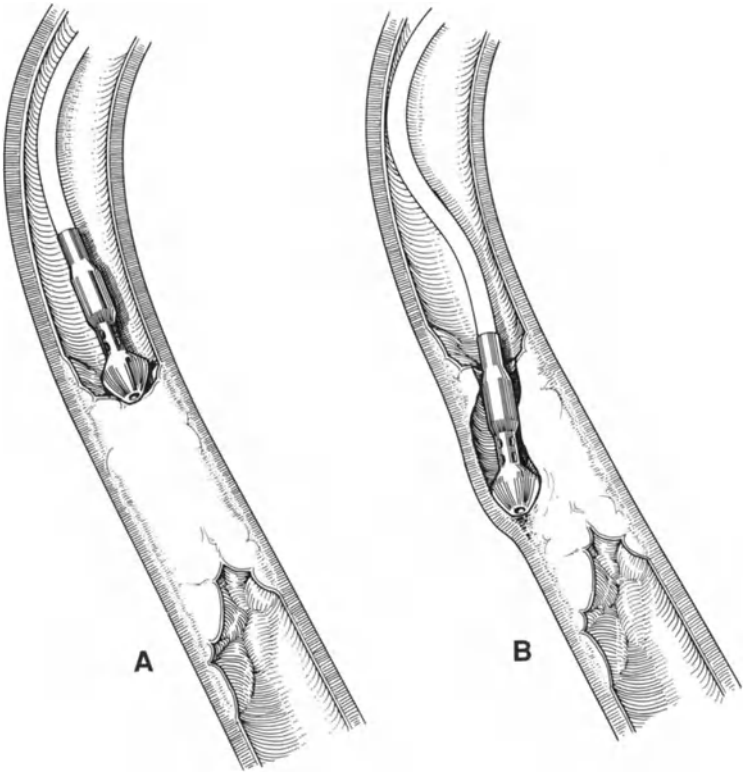


FIGURE 21.3. When a probe encounters an occlusion, it seeks the path of least resistance, which is usually between the intima and media (**A**), leaving the atherosclerotic plaque untouched outside the dissection plane (**B**).

problem. The initial mechanistic impression of ablation in this pathology was the laser probe meeting the plaque perpendicularly to maintain a coaxial alignment through the lesion. In reality, as soon as most probes encounter plaque, they seek the path of least resistance, deviating into a newly formed dissection plane. Most frequently, this aberrant path lies between the intima and media, leaving the atherosclerotic plaque untouched outside the dissection plane (Fig. 21.3).

This deviation does not necessarily portend a negative procedural result, inasmuch as luminal reentry may be achieved distal to the lesion. However, it is obvious that if the majority of atherosclerotic material remains intact along a dissection plane, then the opportunity for reocclusion is significant.

Although lasers at their current level of development no longer dominate our endovascular surgery program, their use and integration with other recanalization techniques can now be delineated more precisely in a treatment strategy that derives from plaque morphology.

Despite this new emphasis on selection strategies, however, we cannot forget the importance of proper technique and superior radiographic and ancillary equipment. Without these foundations, endovascular surgery cannot reach its full potential.

The Endovascular Treatment Suite

While there are many factors that can adversely impact the technical quality of vascular recanalization with any of today's intraluminal devices, poor quality or inadequate intraprocedural imaging is probably the most significant.

I have made this statement time and again, but the message is vital. Visualization of the pathology in a completely unobstructed manner is critical in the selection of appropriate interventions, their safe performance, and their assessment. This sophisticated monitoring can be achieved only with the high resolution radiographic equipment available in some newer radiologic suites and cardiac catheterization laboratories, although even many of these do not lend themselves readily to our latest interventions.

Surgical theaters are usually devoid of this equipment, and it is imperative that they be updated if endovascular therapies are to be used. The Arizona Heart Institute's endovascular operating suites at Humana Hospital-Phoenix have gone through an evolutionary developmental phase, being specially designed and constructed to accommodate today's state-of-the-art vascular intervention and imaging instrumentation. These theaters now represent some of the world's most advanced technology.

The primary components of the vascular laser operating theater center around intraoperative arteriography. The ceiling-mounted surgical C-arm roentgenographic unit (ISS-2000 Plus Intraoperative Imaging System, In-

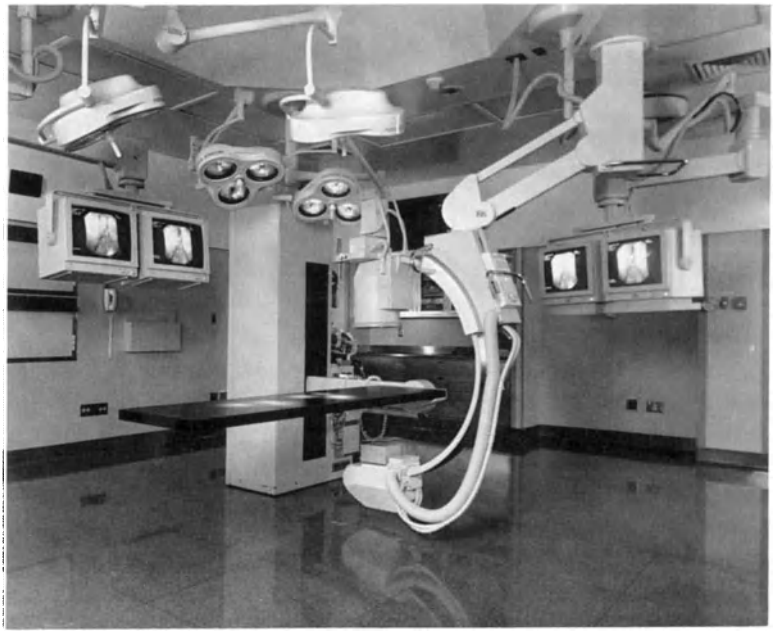


FIGURE 21.4. A ceiling-mounted surgical C-arm roentgenographic unit with image enhancer and a nonmetallic, carbon fiber surgical table are vital components of today's vascular intervention surgical suite. The table, supported by a telescoping pedestal at one end, provides complete clearance beneath for the mobile C-arm and allows vertical travel from 28 to 48 inches, 20° side-to-side roll, and 20° Trendelenburg tilt (standard and reverse). Monitor screens are conveniently mounted for easy visibility.

ternational Surgical Systems, Phoenix, AZ) with image enhancer is integrated with a 3/4″ videotape recorder and monitor for contrast injection visualization. A second monitor with an Eigen disk is also used to provide still images of selected arteriographic segments, facilitating "roadmapping," or real-time subtraction, a tool essential to complex angioplasty procedures. For hard-copy documentation, Polaroid film packs with adaptors may be integrated with the radiographic equipment.

To optimize the usefulness of radiographic equipment in vascular laser procedures, a new nonmetallic, carbon fiber surgical table (International Surgical Systems) has been developed especially for these techniques. This thin but highly stable table is supported at only one end to provide complete clearance beneath for a panning x-ray system (Fig. 21.4). Its telescoping pedestal allows vertical travel from 28 to 48 inches, 20° side-to-side roll, and 20° Trendelenburg tilt (standard and reverse). The table itself can be removed from the pedestal for exchange with other types of table tops.

A metric ruler (USA XRAY) is used to quantify and designate lesions. Placed on the table beneath the patient's pelvis, it is calibrated from the level of the umbilicus to provide these reference measurements.

As an important aside, I cannot stress enough how crucial are the ceiling-mounted C-arm and the metal-free operating table. In my visits to OR suites around the world, I have observed procedures being performed with portable fluoroscopic units and regular OR tables whose metal content interferes with imaging quality. Optimum results simply cannot be obtained using inferior equipment no matter how talented the surgeon or interventionist.

Standard monitoring equipment must also be available for electrocardiography and systemic arterial pressure measurements and differentials. In addition, it is now mandatory to have both percutaneous and intraoperative angioscopy available for direct visualization of lesion pathology and procedural progress.[13] Disposable angioscopes in sizes ranging from 1.5 mm to 3.0 mm can be connected to high-quality medical, video color monitors with hardcopy documentation available through video recorders.

A variety of irrigating systems are available to provide computer-assisted, pulsed irrigation and image storage for maximum visibility with minimal flush volume. In most systems, heparinized saline is delivered under pressure at preset pulse durations on demand via a foot pedal. Synchronization of the flushing with image capture for the digitized output allows both freeze-frame and real-time imaging.

To assist in obtaining a clear field of observation, blood flow must be inhibited as much as possible. Vascular clamps control proximal flow during intraoperative angioscopy. In percutaneous cases, the sheath is sized as near as possible to the artery's diameter to provide adequate proximal control, but manual compression of the common femoral or external iliac artery is sometimes necessary (Fig. 21.5). Retrograde flow in either case is reduced by a pressure cuff placed below the knee and inflated during periods of observation.

The importance of ultrasound imaging cannot be underestimated in today's vascular procedures. Preoperatively, duplex scanning provides good quantification of a lesion, its characteristics, and the impact on flow. These ultrasound studies of plaque morphology are becoming ever more important in the selection of an appropriate interventional device. For example, if dense calcium is present, the lesion may not be amenable to laser therapy at all.

Intraprocedurally, intravascular ultrasound scanning (IVUS) is invaluable for both preprocedural evaluation and post-interventional assessment.[14,15] It can detect flaps and dissections missed by fluoroscopy and dictate the implantation of a stent or the need for atherectomy. Further, it provides far better poststenting evaluation than does arteriography.[16] Very soon, three-dimensional reconstructions of serial IVUS images will be a regular part of our ultrasound technology, further enhancing our assessment capabilities.

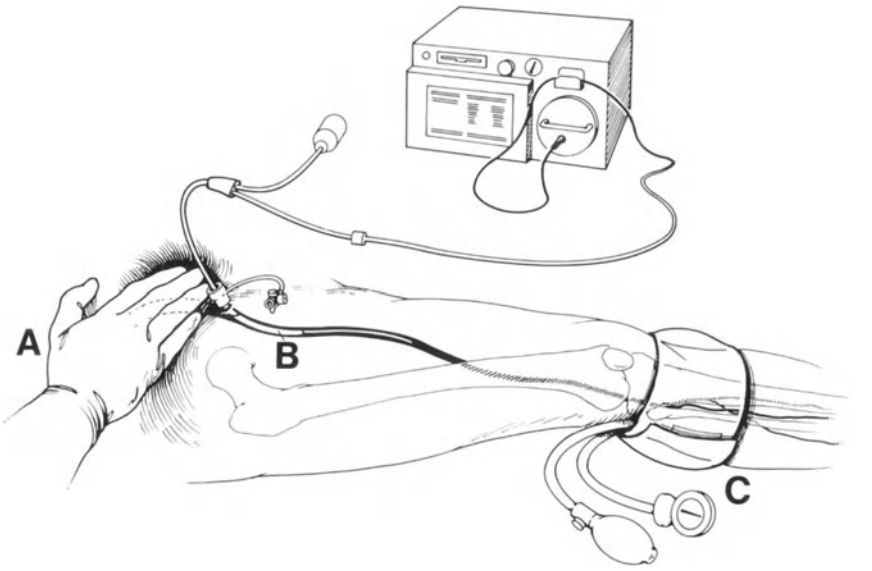


FIGURE 21.5. Clear viewing fields for angioscopy can be maintained by combining a pulsed infusion system with pressure control of proximal and distal collateral blood flow. For percutaneous angioscopy, manual compression on the common femoral or external iliac artery (**A**) helps control inflow with a sheath sized to approximate the vessel's diameter (**B**). A below-knee pressure cuff (**C**) is inflated during observation periods to inhibit retrograde flow.

At this time, our laser program uses the pulsed, near-infrared holmium:YAG system (Trimedyne, Inc., Tustin, CA) and the newly marketed hybrid probe design, the 2.5 mm Spectraprobe-50 (Trimedyne).

The Spectraprobe-50, with its conically-shaped metal tip fitted to a 600 micron fiber similar to earlier models, has a 0.9 mm aperture that emits approximately 50% of the laser energy so that it achieves a maximum temperature of only 85°C when operated within recommended limits (3.6 J/pulse at 10 Hz). With a maximum energy density of 4000 mJ/mm^2, the Spectraprobe is highly effective in crossing occlusions resistant to standard guidewire passage. Furthermore, the probe can be more coaxially aligned, partially owing to special fluoroscopic guidance systems and newer guiding catheter techniques, making it more advantageous than wire-dissected planes.

A new generation of hollow fiber catheter, the Halocath (Trimedyne), is being evaluated for use in larger arteries and stenoses. It features a fused quartz lens that distributes 100% of the holmium laser energy to create a path greater than the 3.0 mm diameter of the probe. While the design retains the central 0.035″ guidewire channel, the lens configuration reduces the number of fibers in the bundle, increasing flexibility.

We are also investigating a pulsed ultraviolet excimer (308 nm) system

(CVX-300, Spectranetics, Colorado Springs, CO) for the infrapopliteal arteries. The blunt-tipped excimer catheters are available in 1.4 mm (45×100 micron fiber bundle) and 1.7 mm (13×200 micron fibers) versions that track over a 0.014″ guidewire. Several modifications of this catheter design are expected within the year.

Patient Evaluation

A vascular work-up for patients with activity-inhibiting claudication is contained within a comprehensive cardiovascular examination to determine risk factors and disclose latent, concomitant occlusive disease.

A lower limb arterial Doppler examination with exercise (unless contraindicated) is used to determine the baseline ankle/brachial index (ABI) and identify the likely location of the vascular compromise through segmental pressure readings.

The hemodynamic impact of many lower limb lesions cannot be properly evaluated from these simple tests, however. New magnetic resonance (MR) blood flow scanning provides a rapid, noninvasive method for ascertaining blood flow impairment. Alternately, for lesions in the femoral or distal circulation, color flow Doppler or duplex scanning may also be used to estimate the hemodynamic significance of lesions and determine plaque morphology.

Arteriography continues to be the accepted standard for lesion characterization prior to treatment. However, a combination of all these lesion and flow assessment tests can provide sufficient diagnostic information to defer the requisite preprocedural contrast arteriogram until the time of interventional treatment in many cases.

Overview of Laser Applications and Alternate Therapies

Because the dynamic nature of laser technology persists, criteria for selection of an appropriate laser or ancillary intervention must remain flexible at this point to absorb enhancements and new devices. However, our experience has given us sufficient data on which to base strategies for disease treatment based on lesion location, plaque morphology, and device capabilities.

In the *iliac system*, today's laser technology works very well in *occlusions*, particularly those that cannot be traversed retrograde with a wire. Alternately, a transaortic approach, as described below, can facilitate wire passage and guide antegrade lasing in amenable occlusions. However, because the size of the largest marketed probe (2.5 mm) is significantly smaller than iliac artery diameter, only minimal tissue can be ablated to debulk the lesion. This "suboptimal ablation" is also the reason why

stenoses in the iliac system really do not warrant laser therapy with current technology unless they are concentric and very tight.

Furthermore, this inadequate ablation dictates that iliac lesions must always be dilated to achieve maximum luminal area, since atherectomy offers little additional ablative assistance, again due to the small size of the device. Once dilatation has been judged adequate by pressure gradient obliteration, contrast imaging, and IVUS scanning, stenting is highly recommended. In fact, we always use an intravascular stent in occluded iliac arteries (discussed in detail later). With the significant amount of atherosclerotic material remaining and the great likelihood of an irregular, debris-laden intima, iliac occlusions demonstrate a greater propensity toward reocclusion than do stenoses. Stenting is at present the only means to achieve long-term patency for this type of lesion, and it is strongly recommended for stenoses as well to ensure adequate proximal inflow.

In the *superficial femoral artery* (SFA), occlusions also enjoy a high rate of initial recanalization success with the 2.5 mm Spectraprobe, but inadequate ablative volume persists here, too, owing to the small 0.9 mm aperture. The 3.0 mm Halocath, with its larger effective lasing path, has proven more satisfactory in preliminary use, but longer follow-up must be accumulated before conclusions can be drawn.

Lengthy SFA occlusions (>7 cm) demonstrate a challenge for laser therapy. Calcium deposits may cause deviation of the probe, creating a neolumen that may at times coincide with the true lumen. This irregular channel leaves significant portions of plaque which the balloon must later deform.

Many SFA occlusions may respond well to some form of rotational atherectomy, either with the unguided Theratek (Kensey) catheters (Dow Corning Wright, Arlington, TN) or the wire-guided versions, such as the Transluminal Extraction Catheter (TEC, Interventional Technologies, San Diego, CA) and Rotablators (Heart Technology, Bellevue, WA). Data regarding long-term follow-up on these techniques is not yet available, however.

SFA stenoses, particularly eccentric ones, may suffer a similar fate because the wire cannot guide the effective lasing surface of the catheter into maximum contact with the lesion. For eccentric lesions, directional atherectomy with the Simpson Atherocath (Peripheral Systems Group, Mountain View, CA) may be able to ablate more tissue than current laser catheters. However, the amount of tissue excised appears insufficient to most surgeons accustomed to endarterectomy specimens.

In the distal vessels, lasers have traditionally performed satisfactorily only in the *popliteal artery*. The smaller trifurcation vessels require precise laser control to avoid arterial injury; the coronary-sized probes powered by the excimer laser (under investigation) seem to work well for stenoses. However, dilation remains the mainstay of treatment in the infrapopliteal area.

Under some circumstances, the use of a *thrombolytic agent* prior to laser

angioplasty can be helpful. If a thrombus superimposed on an atherosclerotic plaque is suggested by either clinical symptoms, angiographic findings, or angioscopy, then clot lysis can often facilitate guidewire passage across the lesion for laser and balloon angioplasty. This sequence can be used in the iliac, SFA, and popliteal/trifurcation arterial distributions.

In the iliac area, either transaortic or axillary antegrade infusion of the thrombolytic agent is effective. For SFA and popliteal lesions, lytic therapy can be initiated through contralateral access or antegrade femoral artery cannulation with a small 4–5 Fr catheter or sheath. Percutaneous angioscopy can be used to monitor progress of the treatment.

Our current protocol calls for the intraarterial infusion of 250,000 units of urokinase followed by drip infusion at 40,000–100,000 units/hour. The partial thromboplastin time is maintained in the 40–90 second range with a concomitant heparin drip (800–1,200 unit/hour after a 1,000–3,000 unit bolus). Infusions up to 48 hours have been used with this regimen followed by successful laser-assisted angioplasty.

Basic Laser Angioplasty Techniques

Patient Preparation

Patients selected for laser angioplasty are started on 325 mg/day of aspirin and 75 mg dipyridamole tid 24 hours before surgery. Inasmuch as the majority of these patients are scheduled for same-day admission, the standard preoperative work-up required for hospitalization is done in advance.

Epidural block is our preferred form of anesthesia for laser-assisted procedures regardless of the entry technique. This mode of anesthesia has several advantages over local or general methods, and it produces a profound sympathetic response that eliminates to a great extent the vasospasm observed with local anesthesia. If epidural block is contraindicated (patients with previous spinal operations or in whom a thrombolytic agent will be used), local or, more rarely, general anesthesia may be employed.

Approach Selection Criteria

The approach is determined by location of the lesions and necessity for exposure of the common femoral artery (CFA). In general, the open technique is required for approach to the iliac, common femoral, profunda femoris, and superficial femoral arteries in instances of: 1) complete occlusion of the common or external iliac artery (with absent femoral pulse) if percutaneous needle insertion is not possible; 2) extensive atherosclerotic disease preventing introduction of the sheath at the site of percutaneous entry; 3) common femoral or profunda femoris lesions in conjunction with SFA occlusion and stenoses; or 4) situations requiring femoral-femoral bypass grafting (i.e., one iliac artery can be successfully lased, but the

occluded contralateral artery is resistant to laser/balloon dilation). Popliteal lesions need open access only when the route through the proximal SFA is occluded. We prefer the percutaneous approach because of its simplicity and freedom from incision-related complications, but we have found the surgical route necessary in about 20% of our cases.

We have found that in treating bilateral SFA disease, staging these procedures is preferable, doing each limb on successive days with the epidural catheter left in place while the patient stays in the intensive care unit for circulatory observation.

Iliac Artery Access Techniques

For percutaneous access to suitable iliac lesions, the standard *retrograde approach* begins with an 18-gauge Potts-Cournard or Argon needle with obturator (USCI-Bard, Billerica, MA) inserted cephalad in the CFA. A #9 or #10 Fr sheath (Cordis, Miami, FL) is likewise inserted retrograde from the CFA into the external iliac artery. A pressure line is attached to the sheath's side port to monitor femoral-radial pressure differential (elimination of any pressure gradient is an important determinant of successful iliac recanalization). Once sheath placement is confirmed with contrast, 2500–5000 units of heparin (depending on patient size) are given intravenously.

A 0.035″ (150 or 240 cm length) hydrophilic wire (Glidewire, Medi-tech, Watertown, MA) is then passed into the aorta to guide laser catheter delivery (holmium-powered Spectraprobe or Halocath). If an iliac occlusion cannot be traversed retrograde with a wire, it can be very successfully recanalized with the Spectraprobe, often using a 10 Fr Superflow guiding catheter (Schneider, Minneapolis, MN) and roadmapping to enhance lasing success.

On the other hand, *transaortic wire passage* is an option that should not be overlooked in those situations where retrograde crossing of the iliac lesion is difficult. A contralateral wire can be directed across the aortic bifurcation using a standard internal mammary, artery-guiding catheter. Often the wire will cross into the contralateral external iliac artery, and it can be delivered into the percutaneous sheath either directly or with the aid of biopsy forceps. Once a wire is thus positioned, antegrade recanalization with the laser and balloon can be easily accomplished.

SFA and Distal Arterial Access Techniques

Percutaneous access to the superficial femoral, popliteal and trifurcation arteries using an *antegrade femoral approach* commences with the 18-gauge Potts-Cournard or Argon needle inserted antegrade into the CFA. This guides the 0.035″ Glidewire, which is passed as far distally as possible under fluoroscopic control. With the needle withdrawn, an appropriately

sized (#5–9 Fr) introducer with sheath is positioned antegrade in the CFA and proximal SFA. (Sheaths are used in cases of surgical access as well to control introduction of the laser probe, guidewire or balloon.) Once sheath placement is confirmed with contrast, 2500–5000 units of heparin (depending on patient size) are given intravenously. Over-the-wire treatment can then commence with the Spectraprobe.

Surgical Access

If lesion pathology as outlined above dictates an open approach, a vertical incision is made to expose the CFA from the inguinal ligament distally to its bifurcation into the origin of the profunda femoris artery and the proximal SFA. After heparin administration, the common femoral and profunda femoris arteries are clamped. In the open technique, there is often no need to clamp the SFA since it is generally occluded at its origin.

With the arteries crossclamped, an arteriotomy is made in the CFA for introduction of the sheath and laser probe. The incision is extended approximately 3 mm into the SFA. If a significant obstruction exists in either the profunda femoris or common femoral arteries, a classical endarterectomy is performed, intentionally terminating near the origin of the SFA.

If the SFA is obstructed at the incision site, the Spectraprobe-50 is introduced into the atherosclerotic plaque and passed distally to create a 2–4 cm channel in the SFA. The catheter sheath is then inserted through the arteriotomy; a small bolus of contrast material is injected through the sheath to confirm its location and identify any potential pathway for the probe through the distal arterial obstruction. With the entry site thus prepared, the procedure continues as described for the percutaneous approach.

When dealing with a flush occlusion of the SFA, it is important to determine if a flap of atherosclerotic plaque has been created by the probe at the proximal aspect of the artery. In accessing the severely diseased SFA, the probe has a tendency to deviate posteriorly from the coaxial plane, leaving residual plaque on the anterior wall at the site of the incision (Fig. 21.6). If this flap is left untreated, the reestablished blood flow will dissect this area causing the proximal artery to reocclude.

To prevent this problem, the flap must be tacked up using the *boat-dock technique* (Fig 21.7) prior to closing the arteriotomy. A piece of knitted Dacron graft material (Sauvage) is cut the length of the arteriotomy. The residual atherosclerotic plaque at the proximal SFA is secured anteriorly against the artery wall with 5-0 prolene suture. The Dacron patch is then sutured to the secured plaque as if a boat were being moored into dock. When completed, the atherosclerotic plaque, like a pennant on the bow, is fixed in position. Just prior to completing the suture line, the clamp in the SFA is relased and retrograde flow from the newly created posterior channel can be observed. Attention to these details is extremely important to assure success in treating disease at the origin of the SFA.

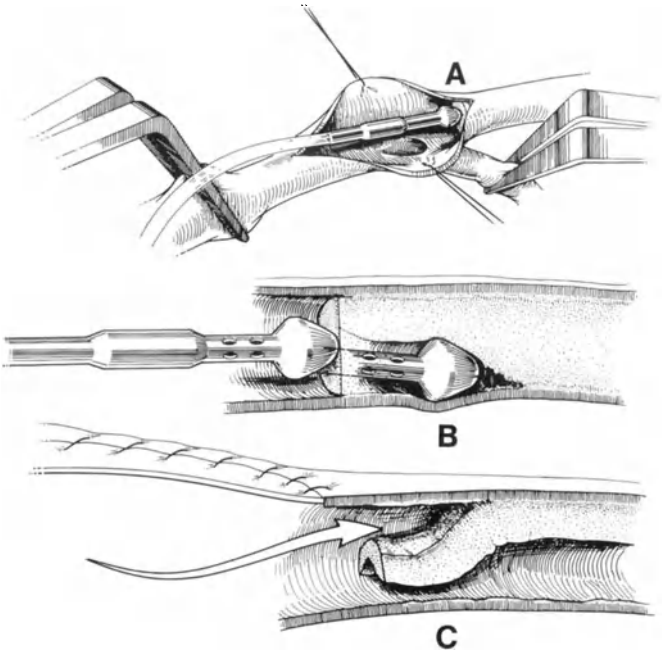


FIGURE 21.6. In the surgical approach to severe superficial femoral artery lesions, the probe encounters the plaque in the coaxial plane (**A**). However, posterior deviation (**B**) of the probe creates an anterior atherosclerotic flap that, if unrepaired, can compromise circulation and cause restenosis (**C**).

The open approach is completed without reversal of the heparinization, and a Jackson-Pratt drain is placed along the incision base.

Retrograde Approach

When the SFA origin is occluded but a long segment of popliteal artery is open above the knee, it is possible to use a retrograde popliteal approach to open the entire length of the occluded SFA.

The procedure, in which both the Glidewire and Spectraprobe work well, is begun with the patient in a supine position. A 5 Fr obturator is introduced percutaneously into the CFA in an antegrade fashion. The obturator glides into the profunda femoris artery because the SFA is blocked. Its position is confirmed with a bolus of contrast material. The obturator is secured to the skin with a suture, and a three-way stopcock and long venous tubing are connected to the obturator.

The patient is then rolled into the prone position, and the popliteal space is prepped and draped. The fluoroscopic unit is centered directly over the popliteal area. Contrast is injected through the proximal obturator, and as

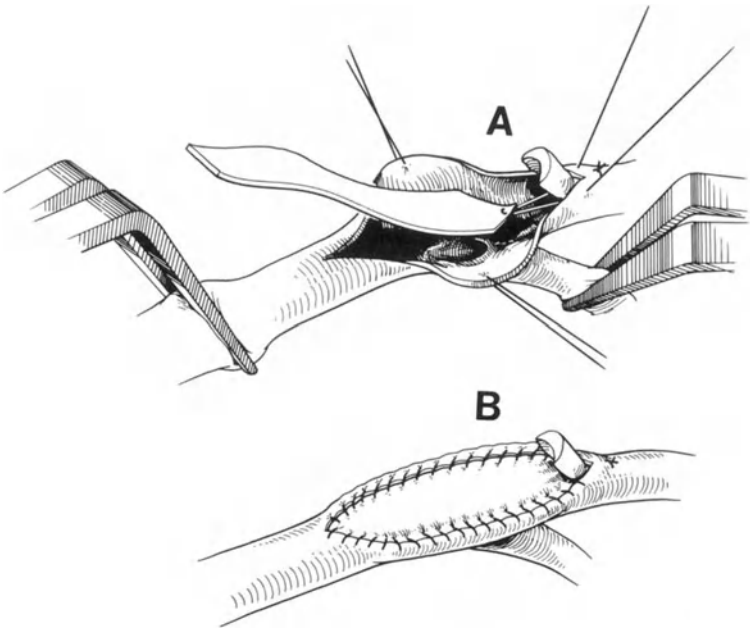


FIGURE 21.7. The "boat-dock" procedure for anterior flap repair begins (**A**) with a knitted Dacron patch sized to fit the arteriotomy. With the flap secured anteriorly against the artery wall with 5-0 prolene suture, the patch is moved into position over the arteriotomy as if a boat were being moored to dock. The graft is sutured in place (**B**), exteriorizing the flap material to leave it dangling like a pennant on the boat.

the open segment of the popliteal artery is visualized, the 18-gauge Potts-Cournard needle is inserted. Once entry is confirmed, a guidewire is passed into the artery for sheath insertion. Retrograde lasing and balloon dilation are performed as in the antegrade approach, with pressure gradients measured through the popliteal sheath. If a gradient persists, it indicates that a proximal flap is present, usually at the origin of the occluded SFA. The patient should then be turned over to perform the boat-dock procedure (see Fig. 21.7) at the proximal access site. Alternately, if an open repair as described above is undesirable, a proximal intraluminal stent may be inserted or atherectomy can be performed at the proximal flap area. We have no experience with these techniques at this time, however.

Dilation

For post-lasing dilation (still necessary except in the few instances in which larger probes may adequately open the artery), a variety of balloon catheters are employed, depending on the nature of the lesions. In superficial

femoral (SFA) and popliteal arteries, the most frequently used balloons range from 4–6 mm in diameter by 4–10 cm in length (Ultrathin and Blue-Max, Medi-tech, Inc.); iliac arteries require 8 or 10 mm by 8 cm models.

For infrapopliteal and distal branches, there is a new generation of Sub-4 small vessel balloons to facilitate dilation. These 3.8 Fr catheters are coated with the same hydrophilic material as the Glidewire to reduce friction.

Balloon expansion can be facilitated with either of two inflation devices, the Indeflator Plus 20 (ACS, Mountain View, CA) or the Intelliflator (Merit Medical Systems, Salt Lake City, UT). Dilation is performed in 30 to 60 second intervals at atmospheric pressures ranging from 6–17, depending on the balloon characteristics and nature of the lesion.

Procedural Assessment

I cannot emphasize enough the importance of pressure gradient monitoring and today's newer assessment tools as complements to the control arteriogram. Since angioplasty began, the completion arteriogram or fluoroscopy with contrast have served as the assessment gold standard. A cosmetically acceptable image has reassured the operator of a satisfactory recanalization.

However, our angioscopic evaluations after successful laser recanalization have given us direct evidence that postprocedure completion arteriograms significantly overrate success of the procedure.

This critical discrepancy between the angiographic picture and the actual condition of the artery represents what we call the "spoof factor"—a situation in which the operator has been deceived or fooled.[17] Once the spoof factor is acknowledged, it is much easier to understand the recurrence seen in longitudinal studies.

Clearly, we need to use more accurate endoscopic assessment methods that are being developed. Angioscopy allows direct visualization but lacks quantitative capabilities, with limited applicability at present in the larger iliac arteries. IVUS scans, on the other hand, offer a reproducible means of measuring luminal diameters in both large- and small-bore arteries. Additionally, intravascular ultrasound provides a great deal of information that augments standard imaging techniques, such as arterial wall composition, nature and configuration of plaques, identification of dissection planes, and integrity of stents and degree of tissue growth within the struts.

Intraluminal Stent Implantation[18]

Today's implantable prosthetic stent designs fall into three main categories: thermally expandable coils made of nitinol; spring-loaded, self-expanding tubular stents composed of either stainless steel or polyester; or metal coils, loops, and cylinders positioned and expanded by a balloon.

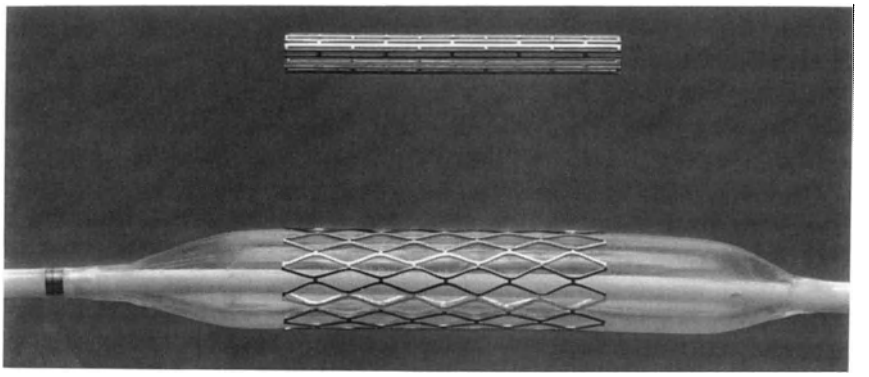


FIGURE 21.8. The Palmaz balloon-expandable tube stent, when expanded, demonstrates the diamond pattern assumed by the steel struts to maintain luminal integrity.

The balloon-expandable devices appear at present to be the most easily, reliably, and securely implanted.

The Palmaz balloon-expandable stent (Johnson & Johnson, Warren, NJ) for peripheral vessels is a stainless steel tube designed with multiple rows of staggered rectangular slots that assume a diamond shape when expanded, reducing to 10% the amount of metal in contact with the luminal surface (Fig. 21.8).

As used in the iliac system (currently the only FDA-approved site), the 3 mm by 3 cm stent is 0.015 mm thick and is expanded to a ratio of 6:1 by an 8 mm by 3 cm balloon (PE Plus II, Cordis) delivered through a 9.0 Fr Pinnacle sheath (Terumo) over an Amplatz 0.035″ super stiff wire (Medi-tech).

The Strecker stent (Medi-tech) is a tubular stent of knitted tantalum wire 0.1 mm in diameter. It is radially and longitudinally flexible and somewhat elastic. It becomes up to 3 cm long when deployed and comes in sizes ranging from 5–8 mm that are expanded by 5 Fr balloons delivered through 8 or 9 Fr sheaths. This stent's flexibility makes it particularly useful for delivery through curved arteries and in vessels subject to flexion from adjacent joints or structures, such as the common femoral and popliteal arteries. (Currently, this stent is undergoing clinical investigation in the U.S.)

Stents have become a prime component in the battle to overcome restenosis, which occurs at a rate of 30% or more in all angioplasties regardless of intervention method. We use them routinely in all iliac occlusions, and now in any iliac stenoses that demonstrate abnormal dilation characteristics (e.g., resistance, recoil) or dilation failure (e.g., persistent filling defect, dissection, thrombosis, or occlusion).

Indications for stenting are expanding to match progress in vascular intervention. In addition to coronary and renal arteries, stents are being investigated in the abdominal aorta, pulmonary artery, superficial femoral and popliteal arteries, and for treatment of aneurysms and stenosis in arteriovenous shunts. In due course, these additional applications may prove as successful in maintaining blood flow as the original iliac application.

IVUS imaging and 3-dimensional arterial reconstructions are particularly valuable tools both in selecting stents as a therapy and the assessment of implantation. Because intraoperative arteriography is notably deficient in certain areas of post-angioplasty evaluation, IVUS imaging as outlined above provides an intraarterial view with quantifiable cross-sectional parameters that can be used to verify adequate deployment and assess long-term patency in follow-up examinations.

Once the decision to use one or more stents has been made, a sheath with hemostatic valve is inserted and passed across the stenosis. The stent is then threaded through the sheath (Fig. 21.9A) and advanced to the lesion with the sheath withdrawn for final positioning. Freeze-frame contrast injection images and roadmapping assure exact stent placement.

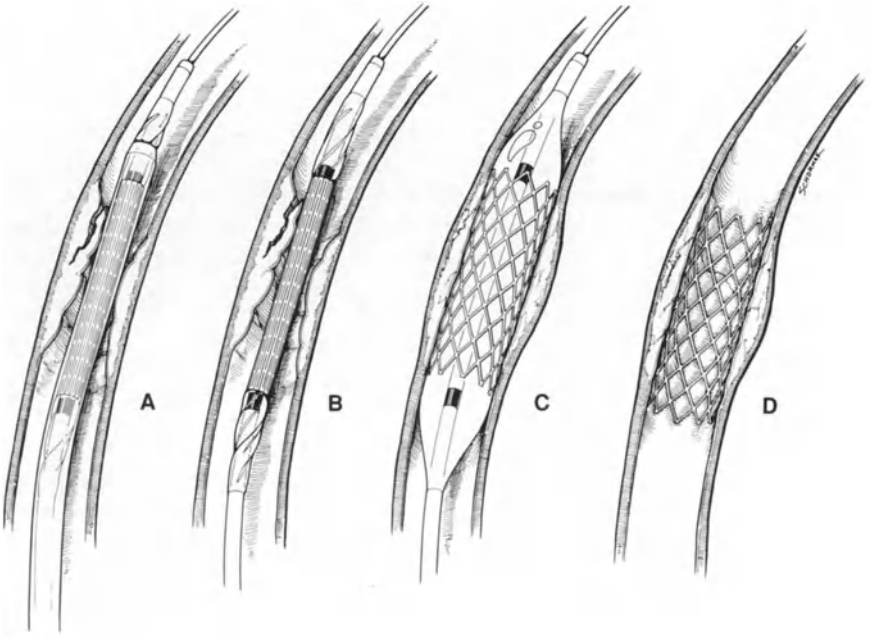


FIGURE 21.9. (**A**) The preloaded Palmaz stent is positioned at the target site. The sheath is withdrawn (**B**), and the balloon is inflated (**C**) to implant the stent permanently on the arterial wall (**D**).

The balloon is inflated at 8–12 atmospheres to expand the stent (Fig. 21.9B). Residual irregularity or stenosis on either side of the stent may be treated by inflating the same balloon or, if indicated, the next larger size. Multiple stents may be overlapped 5–8 mm along the lesion's entire length.

To complement the pressure gradient eradication that signals a successful hemodynamic result, either IVUS imaging or angioscopy should be used routinely following stent placement to ensure satisfactory expansion and integration with the arterial wall.

Postoperative Care

When a satisfactory laser-assisted angioplasty has been completed, the patient is transferred to the ICU without reversal of heparin. The activated coagulation time (ACT) is allowed to return to less than 150 seconds before the sheath is removed.

The benefit of postprocedural heparinization has become controversial of late. We now use a 1000 unit/hr heparin IV drip (to maintain the ACT above 200 seconds) for 24–48 hours only in cases of complex or long lesions. Patients also continue their aspirin/dipyridamole therapy in the ICU. Ambulation is begun 12 hours after the procedure, provided there are no contraindications and recovery from anesthesia is complete.

Following discharge, antiplatelet therapy (325 mg/day aspirin and 75 mg tid dipyridamole) is continued indefinitely. However, there have been some advantages postulated deriving from the use of warfarin during the healing process. To this end, we have added Coumadin to posttreatment drug regimen (to double the PTT) for six months in patients with complex lesions or reoperative cases.

Postoperative evaluation and exercise arterial Doppler examination are performed within 10 days of intervention. The patient's progress is followed thereafter at six-month intervals by ABI unless return of symptoms or failure to improve warrants closer investigation.

Complications[19]

As our experience has grown, our incidence of adverse sequelae has diminished. Today, groin hematoma is the most common complication of a percutaneous intervention. It can be minimized by careful attention to sheath removal technique that includes 30 minutes of pressure on the site after sheath extraction.

Arterial damage related to laser devices has also lessened due to wire guidance and more controlled energy emission, but wire dissections persist. Among the three categories of arterial damage we employ, the most usual type of injury is Class I dissection in which the catheter or wire deviates from the normal lumen. This neolumen may or may not rejoin the true

lumen, and the intimal flaps that are created may warrant stenting to prevent restenosis in the future.

Less common Class II injuries involve perforation of the adventitia in which the puncture site seals on its own without operative intervention. The more serious Class III perforations, in which arterial damage must be repaired surgically, has occurred only once in our experience.

Comments on the Future Direction of Laser Angioplasty

The investigation of new laser sources and ways in which to properly channel their energy for plaque ablation is progressing at an amazing pace. Although hot-tip technology was not satisfactory, the vascular laser industry is committed to producing systems that will work safely and effectively. It is now clear, however, that this developmental process, directed toward some of the areas outlined below, will be more extensive and prolonged than had been assumed initially.

Paramount on the list of problems to be addressed is increasing the atheroablative potential. The relatively significant acute failure rate (20%) of laser angioplasty is related primarily to the inability to cross a lesion and failure to reenter the proper distal channel, situations found mainly in calcified occlusions.

Defining the appropriate laser source and lasing parameters is one way in which to maximize plaque removal. Pulsing the laser emission allows the use of high-energy beams that can achieve the ablative threshold of calcium (about 1100 mJ/mm^2) without significant thermal accumulation.

Furthermore, atherosclerotic tissue absorption characteristics for the different lasers currently available indicates that the readily absorbed 308 nm excimer wavelength should be more efficient than the less easily absorbed Nd: YAG energy. In similar fashion, the smaller, less expensive solid-state, pulsed lasers like the midinfrared holmium:YAG may work as well as the excimer, though there are some lesions resistant to any laser technology currently under evaluation.

None of today's laser sources have been able to achieve sole-treatment status except in a few cases, so with the concomitant use of balloon dilation, it is not surprising that their rate of restenosis appears similar to that seen with standard PTA alone. If a tangible benefit for laser angioplasty is to be established, laser catheters must be properly designed for sole therapy so that any impact on long-term patency over PTA can be determined.

The current direction of catheter development is toward multiple fibers in either single or multiple circumferential rows. Theoretically, the more fibers available, the greater the potential for plaque removal. However, at present, one of the most significant limitations to successful ablation relates to the excessive "dead space" between and around fibers where no active light is present.

When an area of "dead space" encounters plaque, Dottering rather than ablation occurs. Although this combination of mechanical and ablative recanalization has been seen with most laser catheters, it is now apparent that incomplete ablation has major impact on long-term success. This has been confirmed with angioscopy after laser intervention. Even with the best catheter currently available, appreciable residual material remains, and the intraluminal surface is far from satisfactory. Ultimately, a catheter design that maximizes "active space" will be needed.

In the interim, however, there are now alternatives to ablation via laser vaporization. Either as sole therapy or as an ancillary procedure, atherectomy can augment ablation. Several of the devices can provide satisfactory acute results, although it is usually necessary to follow atherectomy with balloon dilation also. It will be interesting to see which of the atherectomy devices now under study proves most efficacious, particularly if a system can be designed to permit sole therapy without the need for dilation.

It is critical that we have improved guidance systems. Properly aligned penetration of an occlusion and reentry into the true lumen are, at the present time, a combination of skill and luck. Both direct vision angioscopy and new intravascular ultrasound imaging are being eagerly studied as potential guidance systems for the laser probe. Additionally, spectroscopic analysis and plaque-targeting identification techniques are in the early developmental stages.

As mentioned earlier, endovascular stenting must be used to help prevent restenosis, at least until we have a better means of combatting intimal hyperplasia. Stents have proven themselves in the iliac system, where the inability of current interventions to provide sufficient debulking encourages reocclusion. In the SFA, preliminary studies with Strecker and Palmaz stents have not been as rewarding, but the need will remain until we are better equipped to deal with the restenosis problem.

Finally, more accurate assessment of the angioplasty result must be realized. Since the initiation of angioplasty, the completion arteriogram or fluoroscopy with contrast have served as the assessment gold standards. A cosmetically acceptable image has reassured the operator of a satisfactory recanalization. However, our angioscopic evaluations have led us to the conclusion that completion arteriograms following angioplasty grossly overrate the success of the procedure. In fact, in most of our observations, residual atherosclerotic disease and injury from angioplasty are considerable.

IVUS scans offer a reproducible means of measuring luminal diameters, of particular value in both acute and long-term assessment. Additionally, IVUS scans provide a great deal of complementary information, such as arterial wall composition, nature and configuration of plaques, as well as identification of dissection planes. Further, the sonograms depict far better

than arteriography the stent configuration, its proper deployment, and degree of tissue growth within the struts.

Conclusions

It is evident that vascular laser technology continues in a state of flux. Hot-tip technology was a stopgap method heralded as a "magic wand." It has proven less than satisfactory because of the thermal component's inability to successfully ablate plaque and the potential for tissue damage. This has precipitated the rapid development of several new areas of investigation that ultimately should prove even more fruitful. All of this, though, will require time and patience on the part of both physician-consumers and researchers.

With this in mind, though, it is important to recognize that the currently marketed laser technology still has its applications. Despite repeated assertions by certain specialty groups that "every lesion can be traversed by a wire" (or any number of special wires), clinical experience with resistant occlusions proves otherwise.

Indeed, it is precisely this need for laser therapy and its potential for precision recanalization that will continue to fuel rapid development of enhancements necessary to reach this goal. Endovascular procedures will become increasingly more diverse and prominent in the future, and an important component of this armamentarium will be laser technology.

References

1. Diethrich EB. Use of lasers by the peripheral vascular surgeon. Texas Heart Inst J 1989;16:182–187.
2. Diethrich EB. Deficiencies of the modern vascular surgeon: a dilemma, a solution (editorial). Ann Vasc Surg 1991;5:99–100.
3. Frisch R. Endovascular techniques and the vascular surgeon. *Ann Vasc Surg* 1991;5:1–3.
4. Lawrence PF. The surgeon's role in laser angioplasty. Texas Heart Inst J 1989;16:179–81.
5. Rutherford RB. Endovascular surgery: the new challenge. J Vasc Surg 1989;10:208–210.
6. Wexler L, Levin DC, Dorros G, Ring EJ, Jatzen BT, Spittell JA, King SB. Training standards for physicians performing peripheral angioplasty: new developments. Radiology 1991;178:19–21.
7. Gruentzig ER, Senning A, Siegenthaler WE. Nonoperative dilatation of coronary artery stenosis. Percutaneous transluminal coronary angioplasty. N Engl J Med 1979;301:61–68.
8. Diethrich EB. Laser angioplasty: a critical review based on 1849 clinical procedures. Angiology 1990;41:757–767.

9. Diethrich EB, Timbadia E, Bahadir I. Late failure in laser-assisted angioplasty: analysis of cause. In: *Advances in Vascular Pathology*, Stano A, Novo S (eds.), Amsterdam, Elsevier, 1989, pp. 381–6.
10. Diethrich EB, Rozaci J, Timbadia E, Bahadir I, Coburn K, Zenzen S. Laser angioplasty—a surgical perspective. Medicamundi 1987;32:127–32.
11. Diethrich EB, Rozaci J, Timbadia E, Bahadir I, Coburn K, Zenzen S. Argon laser-assisted peripheral angioplasty. Vasc Surg 1988;22:77–87.
12. Diethrich EB. Intraoperative laser thermal angioplasty. In: *Lasers in Cardiovascular Disease*, 2nd ed., White RA, Grundfest W (eds.), Chicago, Year Book, 1989, pp. 82–106.
13. Diethrich EB, Yoffe B, Kiessling JJ, Santiago O, Bahadir I, Stern LA, Levine D. Percutaneous angioscopy in endovascular surgery: value in intervention selection and failure analysis. Angiology 1991;43:1–10.
14. Diethrich EB, Ivens Ferraz D, Hull K, Santiago O, Bahadir I, Kiessling JJ, Stern LA. Intravascular ultrasound imaging of iliac stenoses during endovascular surgery. J Cardiovasc Technol 1989;8:287–93.
15. Diethrich EB, Ivens Ferraz D, Bahadir I, Santiago O. Intravascular ultrasound imaging: use in peripheral laser-assisted angioplasty. J Intervent Cardiol 1990;3:145–155.
16. Diethrich EB, Ivens Ferraz D. Intravascular ultrasound evaluation of iliac stents one year following laser-assisted angioplasty. Echocardiography 1990; 7:547–550.
17. Diethrich EB. The spoof factor. (letter) J Am Coll Cardiol 1989;14:1125.
18. Diethrich EB. Endovascular stents in occlusive vascular disease. In: *Proceedings of the Second World Week of Professional Updating in Surgery of the University of Milan*, Montorsi W (ed.), Bologna, Italy, Monduzzi Editore, 1990, pp. 449–452.
19. Diethrich EB, Timbadia E, Bahadir I. Complications of laser-assisted angioplasty: recognition and classification of perforations. Texas Heart Inst J 1989;16:171–6.

22
Practical Application of Endovascular Techniques—Angioscopy, Balloons, Lasers, and Atherectomy

RODNEY A. WHITE

Introduction

Practical application of endovascular techniques relies on an understanding of the utility of current devices based on a knowledge of the development and distribution of atherosclerotic lesions. Technology is in an early evolutionary phase where recognition of potential as well as limitations leads to rapid development of newer methods.

At present, endovascular procedures are successful when used to treat well-defined areas of stenotic disease or short occlusions. The devices are usually positioned within lesions over guidewires inserted by tactile methods. With all current endovascular devices, there is only limited or no removal of lesions. Thus, immediate thrombosis or early recurrence from reorganization of residual material is a leading cause of short-term failure. Recent data has confirmed that even sophisticated arteriographic imaging significantly underestimates the degree of residual lesion, and that future observations and endpoints for treatment will be determined by accurate, controlled data accumulated by angioscopic and intraluminal ultrasound evaluations.

In this regard, our present approach to the use of endovascular techniques is to provide an alternative to surgical therapy, in patients where this is appropriate, as an adjunct to conventional therapy for treatment of tandem lesions, or for short-term benefit in patients who are poor risk candidates for definitive therapy. The latter group includes patients who can benefit from an interval patency before operation, and those who may heal wounds in threatened extremities when blood flow is restored for a limited period. Additional successful therapeutic applications of endovascular devices include angioscopic valvulotomy in in-situ vein bypass, angioscopy assisted thrombectomy and embolectomy, angioscopy and intraluminal ultrasound monitoring of angioplasty procedures, and the use of intravascular ultrasound for stent sizing and deployment.

Intravascular Access Techniques

Intravascular surgical procedures may be performed by either percutaneous or open incision surgical techniques. Percutaneous insertion of devices is used both in the radiology and cardiology suites and in the operating room. Outside the operating room, the percutaneous route is used in the majority of procedures, while in the operating room a higher percentage of interventions are done through an open surgical incision to accommodate introduction of larger devices or to combine an intravascular procedure with a conventional operation. Aside from balloon angioplasty catheters, many new intravascular devices are difficult to use percutaneously due to size limitations. As interventional devices developed, percutaneous adaption occurs following miniaturization of instruments.

Passage of devices along the vessel lumen theoretically enhances thrombogenicity of the vessel wall by endothelial denudation and can predispose to the long-term development of hyperplastic lesions caused by concomitent fracture of the internal elastic lamina of the artery.[1] During endovascular procedures, multiple insertions and withdrawals of devices compound the potential of this risk. Fluid overload is possible during procedures where repeated contrast dye examinations and anticoagulant fluid irrigations are used to avert thrombogenesis and to clear the field of view for angioscopic examinations. Contrast media volumes should be minimized during difficult procedures where repeated imaging is required. Blood loss must also be closely monitored in prolonged cases, particularly if an open operative approach is used or in cases where undetected bleeding can occur.

Introducer sheaths, which have a hemostatic valve at the instrument introduction port and additional ports for infusion of fluids and contrast dyes, are extremely useful in reducing trauma to vessel walls and in controlling blood loss (Fig. 22.1). Newer intraluminal access devices for use during both percutaneous and open incision approaches are being developed to decrease vessel trauma, to provide better hemostasis, and to facilitate removal of intravascular material. The Fogarty Expandable Access Device (Intimax™) illustrates a novel approach by providing an expandable sheath that is initially low profile for safe intraluminal introduction and then enlarges to conform to the arterial wall, limiting trauma and enhancing hemostasis. A large diameter (9 to 15 Fr), "iris-type" valve on the instrument introduction port accommodates large diameter devices and minimizes blood loss by enabling ease of introduction of devices and sealing characteristics (Fig. 22.2).

Percutaneous Procedures

Percutaneous procedures are limited to those performed using low profile catheters (less than 8 to 10 Fr diameter) and to procedures having a seg-

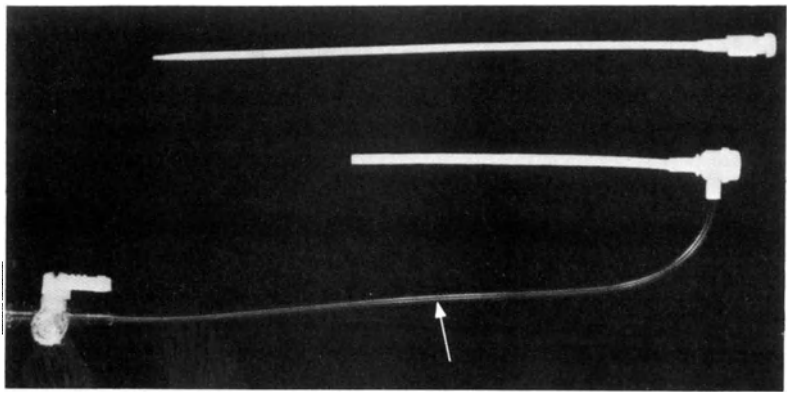


FIGURE 22.1. **A**, hemostatic 8 Fr introducer sheath with side port (arrow) for infusion of fluids or contrast media.

ment of patent vessel available proximal to the interventional site for introduction of instruments. Introduction of percutaneous devices is usually accomplished using the conventional Seldinger technique.[2] Percutaneous vascular access is performed by inserting a beveled needle through the arterial wall via a small skin incision to facilitate subsequent insertion of vessel dilators and instruments. The needle is slowly withdrawn until pulsatile arterial blood flow is achieved, signifying that the needle tip is in the arterial lumen. A guidewire is then introduced through the needle lumen and is positioned using fluoroscopy control. For many procedures an introducer sheath is threaded over the guidewire after passage of a vascular dilator. Sheath diameter is determined by the size of the device that will be passed through the sheath lumen into the artery.

Passage of needles, introducers and instruments through the arterial wall predisposes to dissection and formation of intimal flaps. Acute arterial occlusion may occur from dislodged or embolized intimal lesions. Vessel perforations occurring from use of smaller diameter interventional devices usually do not require repair, particularly if they occur in previously occluded or thrombosed vessels. In this case, there is usually little blood loss, and the perforation seals by rethrombosis of the segment. In addition, arteriovenous fistulas usually close spontaneously following reversal of anticoagulation. Rarely, perforations may continue to bleed, requiring surgical control. The most frequent site where this occurs is in intraperitoneal vessels or with punctures to the external iliac artery above the inguinal ligament to facilitate antegrade access to the femoral vessels. This approach is associated with a risk of hemorrhage into the retroperitoneum.

Intimal flaps usually require operative repair if they produce flow restriction. Microembolization of thrombus or atheromatous material is usually of no consequence if it is limited and there is no evidence of distal ische-

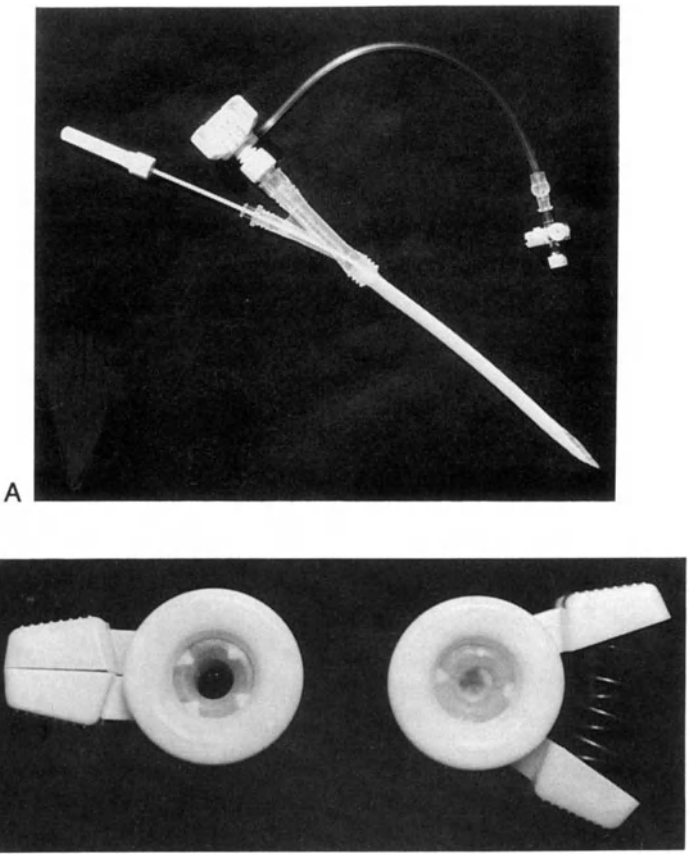


FIGURE 22.2. **A**, Intimax™ Access Device, and **B**, iris hemostatic valve. (Reprinted with permission from White RA: Complications associated with intravascular instrumentation: Endoscopy, atherectomy, lasers and dilatation devices. In: *Complications in Vascular Surgery*, 1991, 3rd ed., eds., Bernard VM, Towne JB, Grune & Stratton, Inc., New York, NY, pp. 420–432.)

mia. If larger artery occlusion develops, percutaneous aspiration thrombectomy or open surgical embolectomy is required to restore flow.[3] Intraarterial infusion of thrombolytic therapy may also be effective in dissolving fresh thrombus.[4] Massive embolization of small (20–200 um diameter) particles can cause diffuse necrosis of tissues producing devastating clinical consequences, such as a "trash limb."

The majority of complications of percutaneous procedures associated with long-term failures (in particular, recurrence of lesions) are common to all endovascular devices. False or pseudoaneurysms at the puncture site in the arterial wall are unique to percutaneous introduction sites, and

frequency increases with increasing size of cannulae and endovascular instruments. False aneurysms frequently become apparent at relatively short intervals following procedures and require surgical repair to prevent bleeding or embolization.

Intraoperative Applications of Endovascular Devices

Intraoperative application of endovascular devices permits use of a larger variety of instruments as either primary therapy or as an adjunct to another vascular procedure. A key factor in accomplishing successful angioplasty in the operating room is adequate radiologic imaging. High-resolution images, which significantly enhance precision of procedures, are a limiting factor in many institutions. Digital subtraction techniques have increased contrast imaging sensitivity, allowing detection of low levels of iodinated materials. Many digital units have freeze-frame and roadmapping features that permit a subtracted contrast image of a vessel to be superimposed on a live fluoroscopic visualization. The quality of equipment available for radiologic imaging of procedures varies from conventional C-arm fluoroscopes to sophisticated image-intensifiers and TV monitoring systems. Immediate image replay systems can improve information accuracy and enhance intervention safety. Recent advances in computerized image processing systems extend the advantages of digital imaging technology to C-arm fluoroscopy by enabling modular addition of contrast enhancement, image holding, and roadmapping during angiographic procedures using available equipment[5] (Fig. 22.3).

Intraoperative percutaneous use of devices is accomplished by methods described in the previous section. Many patients have significant vascular occlusive disease near a possible insertion site that precludes safe passage of a percutaneous introducer. In these cases, surgical incisions provide the best vessel access. Introduction of intravascular instruments through an open surgical incision has several advantages compared to percutaneous methods, as well as potential risks. Open incisions permit inspection of intravascular anatomy, such as the orifices of adjacent branch vessels, and helps decrease the incidence of vessel wall dissection during device introduction. Control of blood flow from collateral vessels by conventional operative methods also expedites angioscopic intraluminal visualization.

Once an arteriotomy has been fashioned, it is advantageous to work through a hemostatic introducer sheath with a sideport, similar to ones used for percutaneous procedures, to help prevent trauma to the blood vessel wall caused by repeated introduction and withdrawal of devices, to provide hemostasis at the introduction port, and to provide a port for injection of contrast dye. Introducer sheaths must be used very carefully as it is quite easy to injure the artery's luminal surface during placement of the sheath, particularly when it is introduced for some distance into an artery.

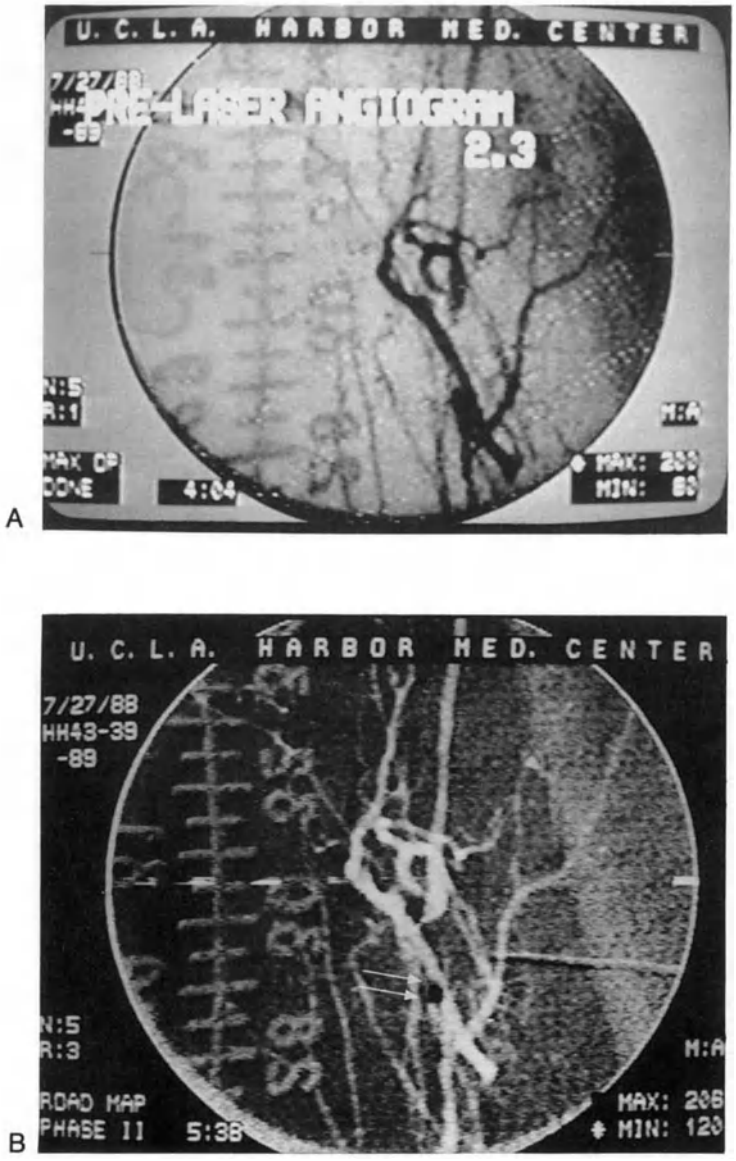


FIGURE 22.3. **A**, angiogram, and **B**, image-enhanced road map of an occlusion in the superficial femoral artery (arrows). A laser probe (double arrows) is positioned for advancement across the lesion. (Reprinted with permission from White RA, et al.: Perspectives for development of angioplasty guidance systems. In: *Lasers in Cardiovascular Disease: Clinical Applications, Alternative Angioplasty Devices and Guidance Systems*, 2nd ed., 1989, eds., White RA, Grundfest WS, Mosby Year Book Medical Publishers, Inc., pp 207–215.)

If a vessel is too diseased to accommodate an introducer sheath, hemostasis can be maintained using Roumel tourniquets with the endoluminal devices passed carefully through the controlled area.

Surgical incisions also permit continuation of anticoagulation, throughout the procedure and postoperatively, if the wound is drained to prevent hematoma formation. Continuation of anticoagulation has been shown to help prevent acute thrombosis of difficult recanalizations in comparison to percutaneous procedures where anticoagulation is reversed and pressure applied to the wound following removal of the intraluminal device to assure hemostatsis. An obvious liability of the open incision technique is the risk of wound infection, particularly if a patch material or prosthetic is used in the repair. The incidence of wound infection has been shown to be higher using the open technique during endovascular procedures.[6]

Angioscopy

Angioscopy, the endoscopic examination of the luminal anatomy of blood vessels, is being used to establish diagnosis and etiology of vascular diseases, to evaluate the technical accuracy of vascular reconstructions, and to visualize intraluminal instrumentation.[7] Attempts to perform cardiac endoscopy were reported in the early 1900s using rigid tubes passed into the cardiac chambers. Vascular endoscopy was initially attempted in the 1960s following development of modern flexible arthroscopes and choledochoscopes. At present, newly developed 0.8 to 3.3 mm diameter angioscopes feature improved fiberoptic imaging systems and light sources, enabling intraluminal inspection of smaller diameter vessels.

Angioscopy Equipment

For angioscopy of peripheral vessels larger than 3 mm diameter, multichannel endoscopic catheters, which incorporate a fluid channel for vessel lumen irrigation to keep the field of view and the lens free of blood, are optimal. Angioscopes that include a fluid channel are approximately 2.5 mm in diameter. This channel or additional channels in larger devices can be used for passage of guidewires, snares, laser fibers or other instruments. The larger angioscopes (2.5 to 3.3 mm diameter) are suited for most peripheral vascular procedures, while 0.8 to 1.7 mm diameter instruments are required for smaller vessels, such as the tibial or coronary arteries. The smaller designs sacrifice the fluid lumen to provide a narrower catheter diameter.

Angioscopic visualization is enhanced by coupling the scopes to a video camera. In this way the field of view can be projected on a TV monitor and is magnified 40–200x. Using the improved angioscopes and video display,

intravascular detail with greater than 0.2 mm spatial resolution at 5 mm is achieved, and the minimum focus distance ranges from 2.0 to 6.5 mm. A light source of at least 300 watts is required to provide sufficient illumination for obtaining adequate images through the fine fiberoptics. Most light sources available in hospitals for gastrointestinal and surgical endoscopy are usually about 100–150 watts power.

The angioscopes, video camera and cables are all gas sterilizable and are set up on a sterile field on a side-table prior to procedures. At all times, these instruments must be handled with care because of the fragility of the optical fibers. Immediately following use, blood is cleared from the tubing and internal channels of the devices to avoid deterioration of the optics. Ethylene oxide gas sterilization and airing procedures take 12 to 18 hours, so each nondisposable angioscope can usually be used in only one procedure each day.

Angioscopy Technique

For percutaneous angioscopic examination, access to the lumen is obtained by passing the scope through an introducer catheter.[8] Percutaneous angioscopy is currently being used as a method to define the mechanisms and accuracy of angioplasties, and to evaluate vessel trauma when angiograms provide equivocal information. Achieving good images with percutaneous angioscopes is currently limited by relatively stiff systems that make control and centering of the devices difficult. To aid manipulation of the scope tip, a steering mechanism in at least one plane is desirable. Control of proximal blood flow in the area being examined can be achieved by inflation of a balloon on the catheter tip. A rapid flow of irrigating solution through the delivery catheter is then required to control blood from collateral vessels. Backbleeding from distal circulation can be limited by having an assistant apply external pressure over the vessels in a distal extremity.

Angioscopy can be performed intraoperatively in 10 minutes or less through an opening in the vessel. To perform intraluminal inspection, vascular occlusion is obtained by conventional operative means or by a balloon on the angioscope's end. Infusion of saline under approximately 300 mm Hg pressure (30–75 ml/min) through an irrigation channel in the angioscope or by a coaxial catheter clears intraluminal blood and enables visualization in approximately 80–90% of cases.

Several investigators have reported that angioscopy reveals clinically important information that is not apparent by extraluminal inspection, probing, or angiography in 15–30% of vascular procedures. The angioscopic findings have altered surgical therapy in a significant number of these cases. In prospective evaluations, angioscopic findings differed significantly from preoperative or intraoperative angiograms in 24% of cases, resulting in an alteration in the operation in 17%.[9]

Angioscopic Thrombectomy

Embolectomy and thrombectomy of peripheral vessels is greatly enhanced by angioscopy.[10] Unless the whole length of vessel is occluded, the angioscope may initially be introduced through the arteriotomy to inspect the lumen and define the exact site and extent of thrombosis or embolism, and to determine whether there is pre-existing atherosclerotic disease. Fogarty catheters may then be passed beside the angioscope if the arterial lumen is large enough to accommodate both devices. Balloon inflation, and detachment and removal of thrombus and debris can then be visually monitored.

Direct visual observation of the degree of balloon inflation is quite important since it allows determination of the amount of balloon distension necessary for adequate removal of the lesion without causing injury to the vessel related to overinflation (Fig. 22.4). Balloon catheters frequently slide over thrombus that adheres to the wall, leaving large fragments, which may or may not be removed with repeated passes and which are frequently not demonstrated adequately by completion arteriograms. When adherent thrombus is observed, further attempts at retrieval can be made by positioning the balloon just distal to the clot and by oscillating it back and forth over the site. If this is not successful, a decision is required to determine if further attempts at extraction are warranted using other

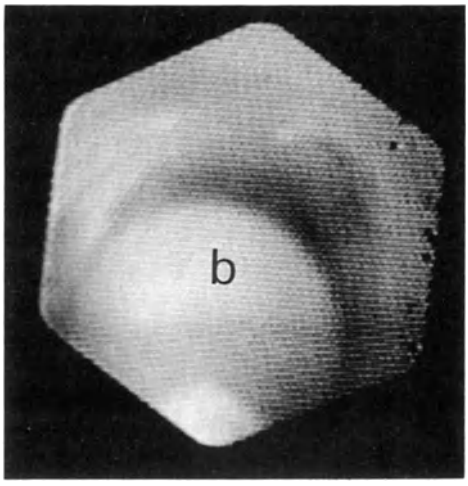


FIGURE 22.4. A Fogarty balloon (b) visualized with angioscopy while it is being inflated in the vessel lumen (Reprinted with permission from White RA, White GH: Angioscopy in the management of infrainguinal occlusive arterial disease in current therapy. In: *Vascular Surgery II*, 2nd edition, 1990, eds., Ernst C, Stanley J, BC Decker, Inc., Philadelphia, PA, pp 544–549.)

instruments, such as flexible grasping forceps, rotary atherectomy devices, or vascular brushes. An additional possible alternative is the intraoperative use of fibrinolytic agents.

When thromboembolectomy is considered complete, a final angioscopic inspection of the entire artery is made. Angiographic examination of smaller runoff vessels may be obtained by injection of contrast medium through the fluid channel of the angioscope before the scope is withdrawn. One of the advantages offered by angioscopy is that complications and technical errors, such as retained thrombus or intimal flaps, can be corrected while the arterial lumen is still open and before blood flow is restored. The angiogram usually fails to demonstrate smaller irregularities of the wall caused by intimal flaps or adherent thrombus and may underestimate larger lesions as well.

Transfemoral thromboembolectomy of the iliac artery is performed by initially clearing about 80% of the occlusion length with the Fogarty catheter and then introducing the angioscope to inspect the lumen for retained thrombus, mural defects, and atherosclerotic plaque. After the remainder of the iliac segment has been reopened, a Fogarty catheter is inflated at the level of the aortic bifurcation to impede blood flow and enable inspection of the thrombectomized vessel. If there is good collateral flow through branches of the iliac artery, it may be quite difficult to overcome the blood in the field to obtain an adequate view. Similar techniques are used for thromboembolectomy in an occluded limb of an aortobifemoral graft. In this case the absence of blood flow from branch vessels ensures a relatively blood-free field.

Femoral-popliteal-tibial thromboembolectomy is performed through a groin incision and by obtaining exposure and control of the common femoral, profunda and superficial femoral arteries. An arteriotomy is performed and the angioscope is first inserted into the profunda femoris artery, where it will usually pass easily to a distance of 20 to 25 cm. Because of the narrow diameter of this artery, the Fogarty catheter cannot be inserted at the same time, so that angioscopic monitoring of profunda thromboembolectomy is limited to inspection before and after passage of the balloon.

In the superficial femoral artery and within prosthetic bypass grafts in this position, the Fogarty catheter will pass comfortably alongside the angioscope so that the whole procedure can be monitored visually. At the distal popliteal level, the orifices of each of the tibial arteries can be identified, and selective cannulation with the Fogarty catheter is often possible. This is achieved by slightly bending the catheter tip to enhance cannulation. It is more difficult to achieve access to the anterior tibial artery because of the severe angle. On occasion, selective cannulation of the tibial artery can avert exposure of the vessels below the knee to retrieve embolic or thrombotic material.

In prosthetic grafts, extensive buildup of neointimal hyperplasia may be difficult to differentiate from chronic thrombus. Use of alternative instruments such as forceps, curettes and brushes to extract intraluminal lesions is probably safer here than in the native vessels. Inspection of the distal anastomosis is important since this is often the site of hyperplastic stenosis. Twists or kinks of the graft can also be identified by angioscopic inspection.

Thrombectomy of the iliac and femoral veins using angioscopic visualization has been reported to be superior to venograms for demonstration of residual thrombus, poor operative result, and the presence of venous spurs in the left common iliac vein.[10] When incomplete thrombectomy is demonstrated endoscopically, a temporary peripheral arteriovenous fistula may be indicated as a means of improving patency rates.

Residual thrombus has been identified angioscopically within arteries and grafts after standard thromboembolectomy procedures in approximately 80% of cases.[11] The direct, three dimensional view obtained provides significantly more information regarding luminal compromise than does contrast radiologic imaging. In many cases the missed clot is quite small and probably is of no significance. This is most likely with soft mural thrombus that is closely attached to the wall and is not stenotic. In some instances, thrombectomy catheters have also been observed to pass between the vessel wall and the clot without dislodging the clot. With lesser amounts of thrombus, especially nonobstructing mural thrombus, it is difficult to judge whether further attempts at removal are indicated. Mural thrombus often simulates spasm on angiograms, and thus good backflow does not necessarily correlate with a satisfactory removal of thrombus. It is likely that many of these minor defects would normally be resolved by the natural fibrinolytic and healing processes. During angioscopic thrombectomy, infusion of irrigating fluid simulates blood flow, thus giving a dynamic representation of potential flaps and loose debris. In some cases, severe dissections, or atherosclerotic stenosis or occlusion, which are identified by endoscopy, cannot be treated endoluminally, indicating that the best therapy is to proceed immediately to vascular bypass rather than persist with unproductive attempts at thrombectomy.

Angioscopy-assisted In-situ Vein Bypasses

Observing the completeness of valvulotomy in in-situ vein bypasses has improved the technical accuracy of the procedure and reduced the operative time by assuring complete incompetence of valve cusps. The valvulotome is inserted through a side branch of the upper segment of the saphenous vein or through the vein lumen at the distal end. The valvulotome is passed proximally from the distal vein through the most proximal valve, and the angioscope is inserted proximally and passed distal until the valvulotome can be visualized at the valve site. Low profile valvulotomes, such

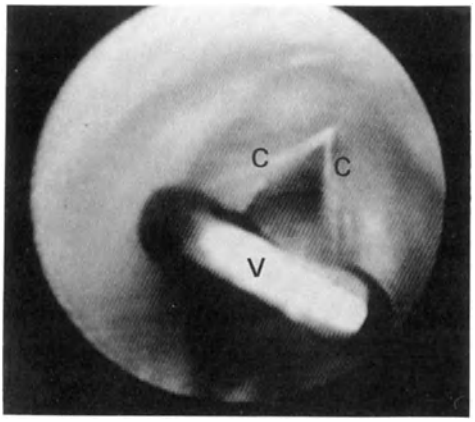


FIGURE 22.5. Angioscopic inspection of a venous valvulotomy procedure (V—valvulotome, C—valve cusp). (Reprinted with permission from White RA, White GH: Angioscopy in the management of infrainguinal occlusive arterial disease in current therapy. In: *Vascular Surgery II*, 2nd edition, 1990, eds., Ernst C, Stanley J, B.C. Decker, Inc., Philadelphia, PA, pp 544–549.)

as a Mills valvulotome, can be easily visualized and controlled by angioscopic inspection (Fig. 22.5). Incompetence of valve cusps is easily tested under direct vision by distending the vein with saline infusion and compressing the vein lumen by external pressure. As valve cusps are serially disrupted, the valvulotome and angioscope are advanced distally to the next valve.

Intraluminal identification of tributary veins during the procedure can help limit dissection and isolation of the vein and prevents tears in the vein caused by hooking side branch with the valvulotome. Several manufacturers are developing microinstruments that can be passed through a lumen in an angioscope or coaxial to the scope to enhance accurate and expedient valvulotomy under direct vision. During the procedure, extreme care must be taken to prevent damaging the vein with intraluminal instruments. Angioscopes too large to pass easily through the lumen can produce severe trauma to the vein wall.

Angioscopic Monitoring of Angioplasty Procedures

Angioscopic monitoring of angioplasty procedures has advantages over arteriography because it removes the hazards of radiation exposure and contrast reactions and allows immediate detection and correction of technical complications. Arteriography tends to underestimate wall irregularities and stenosis. Angioscopic inspection under magnification and video control enables placement of angioplasty devices without deviation into

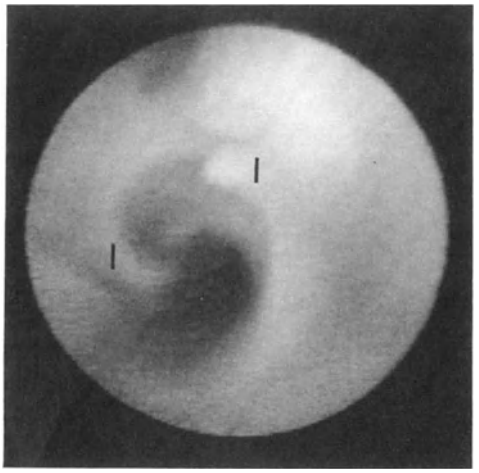


FIGURE 22.6. Intimal flaps (I) which protrude into the vessel lumen after balloon angioplasty of a superficial femoral artery lesion.

collateral vessels. Proximal stenotic lesions or tapering of the vessel near the site of an occlusion limits angioscopic inspection and prevents clear visualization of an occlusion. Angioscopy following angioplasty has been extremely helpful in determining adequacy of recanalizations, inspecting the surface for fragments, and in determining the mechanism of action of recanalization devices.[12] Inspection of treated arteries frequently demonstrates intimal flaps, mural thrombus, balloon dilatation cracks and other intraluminal features not apparent by angiographic studies (Fig. 22.6).

An additional benefit of angioscopy during angioplasty procedures is that the examination is conducted before restoration of blood flow and may help prevent embolization of arterial wall fragments or thrombus. Angioscopy examinations are limited in that assessment of distal smaller vessels is usually not possible because of angioscope diameters. Examination of normal vessels beyond the treated segment may cause intimal lesions or induce spasm. Angiography of the distal vasculature can be performed by injecting contrast through a channel in the scope. This allows direct correlation of the angiogram and angioscopic image.

Angioscopy has limited benefits during an angioplasty procedure or in preventing perforations since visualization of the device is obviated once it has entered an occlusion. An additional obvious limitation of angioscopy is that there is no way to evaluate vessel thickness or concentricity of lesions. In the future, a combination of angioscopy with intraluminal ultrasound may provide a 3-dimensional perspective of the vessel lumen and transmural vessel wall anatomy.

Balloons

Transluminal dilatation of stenotic atherosclerotic arterial lesions was initially advocated by Dotter in 1964.[13] Dilatation was performed using successively larger coaxial polyethylene catheters (a 12F catheter over an 8F catheter). Staple and van Andel later used tapered-tip catheters of 5F to 12F diameter with similar effect.[14] The taper configuration reduced arterial wall trauma that had been common with Dotter's blunt-nosed catheters.

Dotter subsequently postulated that balloon dilatation of an atherosclerotic lesion might effectively enlarge the lumen. In 1965 he successfully dilated a stenosed iliac artery using a Fogarty balloon embolectomy catheter. The concept of balloon dilatation was advanced by Andreas Gruntzig with his 1974 report documenting use of a double lumen polyvinyl balloon catheter.[15] Since then design and construction of angioplasty balloons has advanced, particularly with development of stronger noncompliant, nonelastomeric balloons that can withstand high inflation pressures without distortion of shape or overstretching of diameter. Early polyvinyl chloride balloons stretched easily with moderate pressure, whereas the more recent expandable polymer balloons are stronger and provide forceful radial dilatation of lesions. Increased catheter flexibility and decreased catheter diameters have improved maneuverability of the devices and the ability to cross tight stenotic regions of stenosis, including those in small tortuous arteries of the coronary circulation and the lower leg.

Balloon catheter characteristics that must be considered in the choice of equipment for a particular patient are balloon diameter, balloon length, catheter diameter, catheter length, catheter flexibility, profile and diameter of the catheter tip and the deflated balloon. Recent high pressure balloons may allow inflation to pressures of 12 to 15 atmospheres without increase in diameter. This means that a greater dilating force is achieved without overexpansion—this characteristic may be particularly favorable in densely fibrotic atherosclerotic stenoses.

Following placement of the balloon at the site of an atherosclerotic narrowing, the weakest point in the plaque is fractured by expansion of the balloon[16] (Fig. 22.7). Balloon angioplasty is especially effective for localized, short stenotic lesions (or short length occlusions) of the iliac arteries, with good rates of initial recanalization (> 90%) and long-term patency. Cumulative patency rates of approximately 60–70% at the 4-year interval have been reported.[17,18] A retrograde femoral approach is usually used, and frequently, bilateral iliac angioplasty may be performed via one femoral access site. Measurement of pressure gradients are very helpful in determining the success of an angioplasty procedure. Pressure gradients across a lesion may be measured simultaneously with dual catheters or separately, measuring the pressure as the catheter is passed through the stenosis.

The results of percutaneous angioplasty of infrainguinal arterial disease are not as good as for aortoiliac disease. In this site, if PTA fails then

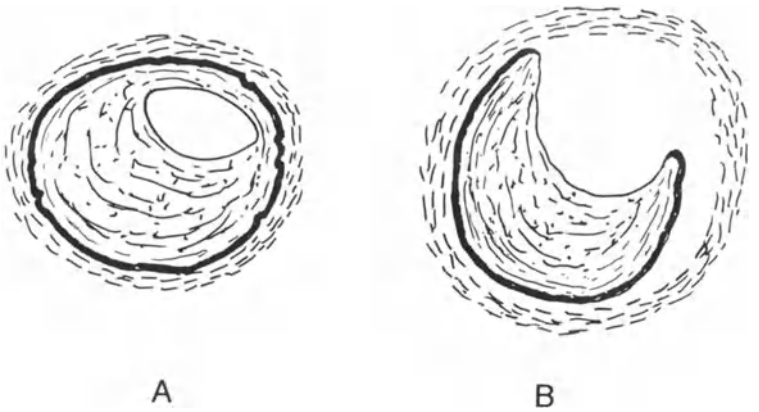


FIGURE 22.7. Mechanism of dilatation of the arterial lumen by transluminal balloon angioplasty. (Reprinted with permission from White RA, White GH: *A Color Atlas of Endovascular Surgery*, 1991, Chapman & Hall, Ltd., London, England.)

approximately 10% of patients are made clinically worse, usually with coincident reduction of the ankle-brachial systolic blood pressure ratio. Factors that predict success are for the lesion to be short, localized (as compared to generalized stenotic disease), and having patent arteries in the distal vascular bed. In general, satisfactory results can be obtained if patients are selected carefully.

Balloon angioplasty can also be used to treat disease in the branches of the abdominal aorta, particularly the renal and mesenteric arteries. Renovascular disease is found to be the cause of hypertension in approximately 5% of hypertensive patients and may often be amenable to dilatation, especially if there is a short, focal stenosis of the renal artery that does not involve the ostium. Lesions of the renal artery orifice, formed by an extension of aortic plaque, are less favorable and more difficult to treat successfully, as are totally occlusive lesions of the renal artery.

Lasers

Lasers have been investigated as a method to recanalize and debulk atherosclerotic occlusions for less than a decade. These devices, more than any other, have undergone rapid development and application. In this regard, controversy concerning the use of lasers as angioplasty devices highlights the problems and discussions relevant to the use of all endovascular surgical instruments.

The initial laser devices used available energy sources, such as the continuous wave argon and Nd:YAG lasers. Preliminary attempts to recanal-

ize lesions using fiberoptic delivery from these continuous wave, thermal sources produced a high incidence of vessel perforation and thermal damage. To capture the thermal energy and attempt to help prevent perforations, ovoid metal tips were placed on the fiber ends. Although there were enthusiastic preliminary reports, based on treatment of stenotic lesions and short occlusions using laser thermal devices plus balloon angioplasty compared to patency for balloon angioplasty alone,[19] application of this technology to longer lesions conforming to surgical indications for therapy have higher complication and failure rates.[20,21] Even when obstructions are successfully recanalized, long-term patency is quite disappointing. Newer devices include a hybrid concept with partial emission of laser energy combined with the thermal effect, pulsed high energy laser devices for precise ablation of tissue, and guided laser angioplasty systems. At this time, the current role of these devices is undetermined and is dependent upon whether future evolution can eliminate some of the present limitations.

The most frequent complication of laser angioplasty is vessel wall dissection or perforation. Regardless of the device, if the probes are used in lesions where a guidewire cannot be initially passed, perforations are a frequent limiting factor. Laser angioplasty highlights the problems regarding guidance of endovascular devices since most atherosclerotic lesions are positioned eccentrically within the vessel lumen, and failures including perforations, dissections, and short-term reocclusions are due to inaccurate guiding and debulking of lesions.[22,23]

An additional complication of laser angioplasty, which is related to imprecise delivery of energy to normal or minimal diseased artery, is the development of lesions in previously unaffected sites adjacent to the treated segment. The most evident presentation of this complication is apparent when patients who were previously treated for short stenoses or occlusions return with long segments of reoccluded vessels. This limitation emphasizes the importance of treating only well defined lesions that can be precisely ablated with current devices. It also emphasizes that treatment of minimal disease, such as claudication, should be reserved for interventionalists who have technical excellence, vast experience and optimal systems available to treat these lesions.

The benefit promised by laser technology is the ability to recanalize arterial lesions that would otherwise require a surgical procedure to reconstruct the segment. Patency of vessels treated by laser angioplasty at 12 months follow-up appears to be comparable to percutaneous transluminal angioplasty for similar lesions but inferior to surgery for occlusions over 3 cm length.[24] Although the method is applicable to iliac, superficial femoral and popliteal arteries, current results are not optimal because: 1) current devices only make a small channel that must be enlarged by subsequent balloon dilatation, 2) inability to deliver energy precisely at the lesion, and 3) a significant incidence of vessel wall perforation and early thrombosis.

Although clear indications for laser angioplasty are still undefined, pilot studies show possible benefit for a limited number of patients who have: 1) arterial stenoses and short occlusions; 2) iliac artery lesions; 3) PTFE graft occlusions due to anastomotic intimal hyperplasia; and 4) for certain high-risk patients who are not good surgical candidates or who are failures of conventional therapy.[6,20,21]

The major advantage of laser energy is that it can provide precise, high-energy ablation of tissue through miniaturized endoluminal delivery systems requiring only minimal intervention to recanalize occluded arteries and to debulk lesions. It is important to recognize that current laser devices only establish feasibility for future application of advanced systems to angioplasty. For example, the "hot-tip" devices are not a guided laser energy source but rather a laser powered thermal probe.[22,24] Newer prototype instruments, the most promising of which emit free laser energy, demonstrate feasibility for the potential evolution of this technology.

Atherectomy

Atherectomy devices are designed to remove atherosclerotic plaque from the vessel; this is done by cutting, drilling or pulverizing atheroma and produces a luminal surface quite different from balloon angioplasty or open surgical endarterectomy. Some of the currently available atherectomy devices have mechanisms for extraction of the fragments of ablated plaque, while others reduce the plaque to microparticles which are circulated within the blood stream and are removed by the lungs and reticuloendothelial organs. Atherectomy catheters may be used percutaneously through an arterial sheath, may also be introduced via an incision, or may be used as part of a conventional vascular operation. Larger catheter sizes are more suited to intraoperative application.

Atherectomy may be used as sole therapy or in conjunction with balloon dilatation. Most current atherectomy devices are suitable for stenotic lesions only and are inserted over a guidewire. If the stenotic arterial lumen is not sufficiently enlarged after a successful atherectomy procedure, adjunctive balloon dilatation is often performed. As a corollary, angioplasty device complications (particularly dissection or acute occlusion) or inadequate recanalization may be improved by subsequent passage of an atherectomy catheter.

Precise indications for atherectomy have not yet been defined and its true role in the management of vascular disease will be determined by further technological developments in instrumentation and extensive clinical experience. Although initial successes have been achieved with several devices, restenosis has been a problem in some initial clinical studies, particularly when the amount of plaque removed may have been

inadequate. Preliminary data suggests that precise control of the lesion's removal level within the arterial wall dramatically improves follow-up patency and hemodynamic results. If this finding is substantiated, development of this principle could dramatically improve the results of endovascular recanalizations.

Simpson Atherectomy Device

The Simpson atherectomy catheter consists of a catheter with a distal cutting blade and its housing chamber, including an attached positioning balloon. The catheter's proximal aspect is coupled to a hand-held motor unit and has two ports for fluid flush and balloon inflation. A lever on the motor drive unit is used to advance the rotating cutting blade down the distal chamber. The nonelastic balloon pushes the cutaway against the plaque. The tubular cutter shaves a section of plaque and impacts it in the distal chamber.

Simpson catheters for use in the peripheral vascular system range in diameter from 7 to 11 F. and have windows of 15 mm and 20 mm length. Excised atheromatous material is pushed by the blade into the distal containing section of the chamber so that it may be extracted by removal of the catheter. Early device designs had a small chamber that filled after only 5 or 6 blade passes; the more recent design has a larger chamber that will accommodate material from 20 to 25 cuts. Atherectomy devices designed for coronary artery use are narrower (4.6 F. to 7 F.) and more flexible, with window opening length of 10 mm. The housing moves over a guidewire through a central lumen.

This type of atherectomy catheter may be indicated for management of balloon angioplasty complications, particularly dissection, intimal flap, inadequate recanalization, and early restenosis. It has been shown that restenosis tends to occur rapidly if insufficient material is removed from the treated artery. If the residual lumen after treatment has less than 20% stenosis, the incidence of recurrent stenosis is reduced.[25,26]

Kensey Atherectomy Catheter

The Kensey atherectomy catheter uses a different mechanism of action to debulk atheromatous plaque.[27] There is no cutting blade, but a rotating cam tip acts by pulverizing firm or fibrous atheromatous tissue into microparticles. The cam tip is rotated by a central drive shaft, housed within a flexible polyurethrane catheter. An external electric motor drive unit rotates the tip at speeds up of up to 200,000 rpm, but rates of less than half this speed are now recommended. There is a coaxial lumen within the catheter that is used for infusion of fluid. Fluid ejected from the rotating tip generates a radial jet that creates a vortex effect within the vessel, enhanc-

ing the emulsification of dislodged atherosclerotic debris. The fluid also serves to cool and lubricate the rotating cam. Elastic tissue of the normal arterial wall is pushed aside and not damaged, whereas firm or fibrous atheroma is sculpted by the rotating tip and the fluid jet. There is no mechanism for removal of fluid or microparticles from the vessel. Access may be by percutaneous sheath or intraoperative cutdown to the vessel. The fluid injected through the catheter often consists of a mixture of fluoroscopic contrast, heparin, dextran and thrombolytic agent. The fluid is usually injected at a rate of approximately 30 mls per minute.

The catheter is typically advanced in a to-and-fro fashion with gentle and gradual progress down the vessel. A guidewire is not required, and the system is considered to be suitable for treatment of total occlusive lesions as well as arterial stenosis. Initial experiences were complicated by a fairly high rate of vessel perforation, since the catheter tends to follow the plane of least resistence, often taking it into the arterial wall. However, modifications of the instrument and experience with the techniques have considerably reduced the incidence of this complication.

Auth Rotablater

The Auth rotablater consists of a rotating shaft used to drill out a core down the vessel lumen.[28] In this case the tip is a burr, with its leading surface covered by metal impregnated diamond chips. A flexible mechanical drive shaft attached to the metal burr tip is housed within a teflon sheath within the atherectomy catheter. The whole system is designed to be fed down the artery over a guidewire and is essentially suitable for treatment of stenotic lesions alone. For total occlusions, a guidewire must first be passed through the lesion. The drive shaft is controlled by a turbine housed in plastic casing attached to the proximal end of the catheter.

Compressed air powers the turbine with rotation speeds greater than 100,000 rpm; speed of rotation is determined by air pressure. The system also contains an irrigation port for infusion of fluid. Burrs are available in various sizes from 0.7 mm up to 6mm diameter. Small size tips are suitable for tibial and coronary arteries, while larger sizes are designed for use in the peripheral vascular system, particularly the superficial femoral and popliteal arteries. Multiple diamond chips on the tip function as microknives, which fracture and fragment atheromatous plaque. A control knob on the motor drive casing allows the surgeon to advance or retract the burr tip over a guidewire. The drive shaft has a disengagement mechanism that prevents the artery from wrapping around the rotating burr or catheter at low torque. Animal experiments using labeled particles of atheroma have demonstrated that these particles are generally less than 1 micron in size, pass through the small vessels of the leg, and accumulate in the lung, liver, and retriculoendothelial system.[29]

Transluminal Extraction Catheter (Interventional Technologies)

The TEC atherectomy catheter, developed by Interventional Technologies, also uses a rotating burr tip technique. In this case the tip has two very sharp blades for cutting plaque, and the internal lumen of the hollow-tubed catheter is connected to suction, allowing extraction of the fragmented particles. The procedure is performed over a guidewire and is suitable for stenotic lesions only. Preliminary studies using this device have been promising, and ultimate utility awaits further evaluation.

Complications

Complications of transluminal atherectomy are similar to those of other interventional techniques within the peripheral vascular system. The incidence of complications should be less than 10%, with thrombosis and embolization being the most important. In experienced hands, less than 5% suffer complications; the long-term systemic effects of microembolization have yet to be fully determined. Hemoglobinuria and hemolysis have been recorded on several occasions. Microembolization to distal tissues in the leg has not been a serious clinical problem, even though many particles must be embolized to the distal arterial beds.

Future Perspectives

Resolution of current limitations and complications of endovascular surgical procedures and devices must address the problems related to guidance of angioplasty instruments and restenosis subsequent to initial recanalization. The guidance issue is particularly relevant since most atherosclerotic lesions are eccentrically positioned within the vessel wall. Many of the failures, including initial perforations and dissections and short-term reocclusions, are due to inaccurate guidance of angioplasty devices and inadequate debulking of lesions. For this reason, major emphasis is being directed toward development of precise guidance methods for endoluminal instruments.

In those patients who restenose following initial recanalization, close follow-up, particularly with color flow duplex scanning and noninvasive hemodynamic testing, will enable serial percutaneous recanalization before reocclusion occurs. Studies are required to determine the optimal level of lesion debulking to provide the longest intervals before restenosis occurs yet preserve adequate vessel wall to prevent aneurysm formation. Additional work is required to assess the impact of embolized debris, generated during angioplasty procedures, on long-term function of the distal vascular bed. The ultimate step in resolving the problem of lesion re-

currence rests on defining the etiology and establishing methods to arrest restenosis.

Several interesting devices are being developed and tested to address the guidance issue. Target-specific laser angioplasty may become possible through analysis of emitted fluoroscence patterns (laser-induced fluoroscence) from the vessel wall.[30] An interesting prototype, which uses spectroscopic guidance for controlled activation of a multifiber catheter, has been developed and is being evaluated.[31] Using argon laser radiation to excite both normal and atheroscleortic vessels, distinct patterns of fluorescence from each tissue can be disseminated from a 19-fiber array by a multichannel spectral analyzer. The spectra and images produced permit selective identification of lesions and ablation in individual quadrants around the arterial wall circumference. Although the concept is appealing, current devices require frequent calibration to adjust signal sensitivity and are expensive to purchase and maintain.

Other prototype delivery systems for angioplasty devices incorporate multiple modalities, i.e., spectroscopy, angioscopy or intravascular ultrasound to enhance precision of initial vessel recanalizations. Intravascular ultrasound promises to be the most user friendly and accurate way to accomplish this goal in occluded vessels. This technology is available as a component of the tip of 3 to 8 Fr catheters and produces a detailed image of the thickness of the blood vessel wall[32,33] (Fig. 22.8).

Recurrence from thrombosis and organization of residual debris or restenosis from smooth muscle cell proliferation of recanalized lesions remain the most troublesome problems limiting effectiveness of endoluminal surgery devices, particularly once the guidance issues permitting precise direction of devices is solved. Minimal progress has been made in defining either the etiology or the control of this phenomenon. Concepts regarding the etiology and development of recurrent lesions are confused and are based on poorly controlled studies of angiographic measurements of one- or two-dimensional angiogram images of lesions prior to, and following, interventions. Accurate data regarding the intraluminal dimensions of treated lesions, before and following angioplasty, and the types of tissue components that have been ablated or displaced is not available in the reported data. Without controlled studies evaluating these parameters, little progress can be expected regarding control of lesion recurrence.

Combined approaches to disobliterate occluded arteries, utilizing thrombolysis, luminal dilatation, and tissue ablation or removal, all have unique potentials if improved guidance and delivery systems become available. Future angioplasty device delivery catheters may combine angioscopy for visual inspection of the lumen, spectroscopy for characterization of the tissue elements, and ultrasound for determining vessel wall and lesion dimensions. Improved intraluminal ultrasound devices will not only provide improved visualization of cross-sectional vessel all anatomy, but 3-dimensional longitudinal reconstruction of the vasculature by storing a

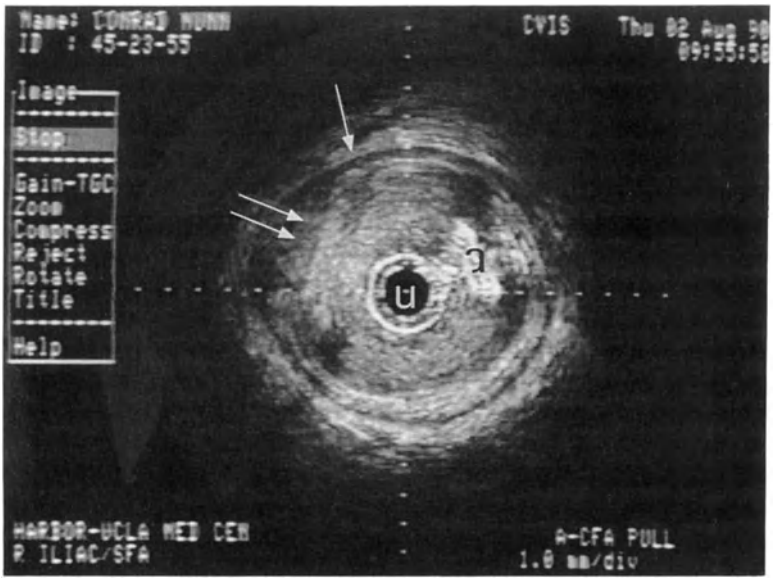


FIGURE 22.8. Intravascular ultrasound imaging of a human iliac artery using an 8 Fr catheter. U, ultrasound probe; single arrow, normal 3-layer muscular artery wall; double arrow, soft plaque or thrombus. Shadow, **a**, is an artifact produced by a thin metallic wire along the axis of a catheter.

sequence of ultrasound images. Improved guidance of angioplasty catheters will help eliminate the primary causes of recanalization failure and recurrence of lesions vessel wall perforation, and inadequate debulking of lesions. This advance is required as the first step toward solving the limitations of current technology. Further improvement will occur as the fundamental processes responsible for restenosis of lesion are elucidated and methods of control become available.

References

1. Clowes A. Theories of Atherosclerosis in *Atherosclerosis and Arteriosclerosis: Human Pathology and Experimental Animal Methods and Models*, 1989, ed., White RA, CRC Press, Inc., Boca Raton, FL, p 3–15.
2. Seldinger SI. Catheter placement of the needle in percutaneous arteriography. Acta Radiol 1953;39:368–76.
3. White RA, White GH. Percutaneous aspiration thromboembolectomy in current therapy. In: Ernst C, Stanley J, eds., *Vascular Surgery-II*, 1990, B.C. Decker, Inc., Philadelphia, PA, pp 598–600.
4. Montarjeme A. Thrombolytic therapy in arterial occlusion and graft thrombosis. Seminars in Vasc Surg 1989;2:155–178.
5. White RA, Kopchok GE, Hsiang Y, Guthrie C, Colman P, Rosenbaum D,

White G. Perspectives for development of angioplasty guidance systems. In: *Lasers in Cardiovascular Disease: Clinical Applications, Alternative Angioplasty Devices and Guidance Systems*, 2nd edition, 1989, eds., White RA, Grundfest WS, Mosby Year Book Medical Publishers, Inc., Chicago, IL, p 207–217.
6. White RA, White GH, Mehringer CM, Chaing FL, Wilson SE. A clinical trial of laser thermal angioplasty in patients with advanced peripheral vascular disease. Ann Surg 1990;212:257–265.
7. White GH, White RA (eds.). *Angioscopy: Vascular and Coronary Applications*. Year Book Medical Publishers, Chicago, IL, 1988.
8. White GH, White RA. Percutaneous angioscopy as an adjunct to laser angioplasty in peripheral arteries. Lancet 1989;2(8654):99.
9. White GH, White RA, Kopchok GE. Intraoperative video angioscopy compared to arteriography during peripheral vascular operations. J Vasc Surg 1987;6:488–495.
10. Vollmar JF, Loeprecht H, Hutschenreiter S. Advances in vascular endoscopy. Thorac Cardiovasc Surgeon 1987;35:334–341.
11. White GH, White RA, Kopchok GE. Angioscopic thromboembolectomy: preliminary observations with a recent technique. J Vasc Surg 1988;7:318–325.
12. White RA, White GH. Angioscopic monitoring of laser angioplasty. In: White GH, White RA, eds., *Angioscopy: Vascular and Coronary Applications*. 1989, Year Book, Chicago, 104–113.
13. Dotter CT, Judkins MP. Transluminal treatment of atherosclerotic obstruction: description of a new technique and a preliminary report of its application. Circulation 1964;30:654–70.
14. Staple TW. Modified catheter for percutaneous transluminal treatment of atherosclerotic obstructions. Radiology 1968;91:1041–43.
15. Gruntzig A, Hopff H. Perkutane pekanalisation chronischen arterieller verschlusse mit einem neuen dilatationskatheter: Modification der Dotter-technik. Dtsch Med Wochenschr 1974;99:2502–05.
16. Block PC. Percutaneous transluminal angioplasty. In: Yu Pn, Goodwin JF, eds., *Progress in Cardiology II*, 1982, Philadelphia, PA, Lea & Febiger, p 1–18.
17. Johnson KW. Balloon angioplasty: Predictive factors for long-term success. Seminars in Vasc Surg 1989;2:113–192.
18. Wilson SE, Sheppard B. Results of percutaneous transluminal angioplasty for peripheral vascular occlusive disease. Ann Vasc Surg 1990;4:94–97.
19. Sanborn TA, Cumberland DC, Greenfield AJ, et al. Peripheral laser assisted balloon angioplasty: Initial multicenter trial in 219 peripheral arteries. Arch Surg 1989;168:121–125.
20. White RA, White GH. Laser angioplasty: Development, current status and future perspectives. Seminars In Vasc Surg 1989;2:123–142.
21. Seegar JM, Abela GS, Silverman SH, Jablonski SK. Initial results of laser recanalization in lower extremity arterial reconstructions. J Vasc Surg. 1989;9:10–17.
22. *Color Atlas of Endovascular Surgery*, 1990, White RA, White GH, eds., Chapman & Hall, Ltd, London, England.
23. White RA, White GH, Vlasak J, Fujitani RM, Kopchok GE. Histopathology of human laser thermal angioplasty recanalizations, Lasers Surg Med 1988;8:469–476.

24. White RA, White GH. Laser-thermal probe recanalization of occluded arteries. J Vasc Surg 1989;5:598–608.
25. Graor RA, Whitlow P, Bartholomew J, et al. Atherectomy of the superficial femoral and popliteal arteries: two year patency and factors influencing patency. Presented at the Society for Vascular Surgery, Los Angeles, 1990.
26. Hinahara T, Robertson GC, Selmon MR, et al. In: Moore WS, Ahn SS, eds., *Endovascular Surgery*, 1989, W.B. Saunders, Inc., Philadelphia, PA, p 310–322.
27. Kensey KR, Nash JE, Abrahams C, et al. Recanalization of obstructed arteries with a flexible, rotating catheter tip. Radiology 1987;165:387–389.
28. Ahn SS. Peripheral atherectomy. Seminars in Vasc Surg 1989;2:143–154.
29. Ahn SS, Auth DC, Marcus DR, Moore WS. Removal of focal atheromatous lesions by angioscopically guided high-speed rotary atherectomy preliminary experimental observations. J Vasc Surg 1988;7:292–300.
30. Murphy-Chutorian D. Laser angioplasty and endarterectomy: Present status and future potential. In: *Current Critical Problems in Vascular Surgery*, 1989, ed., Veith F, Quality Medical Publishers, Inc., St. Louis, MO, p 188–194.
31. Hoyt CC, Richards-Kortum RR, Costello B, et al. Remote biomedical spectroscopic imaging of human artery wall. Lasers Surg Med 1988;8:1–9.
32. White RA. Intravascular ultrasound. In: *Current Practice of Interventional Radiology*, 1989, eds., Kadir S, BC Decker, Inc., Philadelphia, PA, pp 36–41.
33. *Intravascular Ultrasound: Techniques. Developments. Clinical Perspectives*, 1989, eds., Bom N, Roelandt J, Kluwer Academic Press, Dordrecht, The Netherlands.

23
Endovascular Procedures: Current State of the Art

ANTHONY D. WHITTEMORE

Introduction

While autogenous saphenous vein provides the most durable reconstruction for patients with severe peripheral vascular occlusive disease, recent advances in percutaneous endovascular techniques have dramatically expanded alternative approaches. Our past experience with reversed vein grafts demonstrated a 78% primary 5-year patency rate for femoropopliteal grafts and a lower 56% rate for infrapopliteal grafts.[1,2] More recent results with 440 in situ vein grafts provided no significant difference in the femoropopliteal position, but the 70% rate associated with infrapopliteal reconstructions represents a significant improvement, as has been noted in several contemporary series.[3,4–7] Furthermore, these infrapopliteal reconstructions included 18 inframalleolar grafts that provided a 5-year patency rate approaching 80%, a rate confirmed in other recent reports.[8] Secondary patency rates for in situ grafts in all positions exceeded 80%.

In contrast, however, our experience with 300 polytetrafluoroethylene (PTFE) grafts has been remarkably disappointing.[9] After 5 years, 37% of femoropopliteal PTFE bypass grafts and only 12% of infrapopliteal grafts remained patent. While chronic anticoagulation may yet prove efficacious in prolonging patency associated with prosthetic material, the superior durability of autogenous vein seems firmly established.[9,10]

More recently, therefore, in the absence of ipsilateral greater saphenous vein, alternatives to PTFE have been aggressively utilized in 266 consecutive reconstructions for which autogenous tissue proved available in all but 11 (4%).[11] These alternative methods included a variety of endarterectomies and lesser saphenous and/or arm vein grafts, all of which yielded a 66% 4-year patency rate, a significant improvement when compared with previous results achieved with PTFE.

Percutaneous Transluminal Angioplasty

Percutaneous transluminal angioplasty (PTA) has emerged as an alternative approach to the ischemic limb in patients requiring primary revascularization and especially for those without suitable autogenous tissue. Primary intervention for well-localized, short segment lesions has become increasingly successful with the availability of low profile balloons and glidewires. The most favorable results have been observed with localized lesions of the iliac vessels; the efficacy of PTA for infrainguinal disease, however, remains uncertain. Our experience with primary iliac PTA in 184 limbs demonstrated a 5-year cumulative patency rate of 74%. This result, in highly-achieved, selected patients, contrasts with a lower patency rate of 50% observed after 124 infrainguinal angioplasties. There was no significant difference in patency rates observed between 101 superficial femoral and 18 popliteal angioplasties. Infrapopliteal angioplasty has been limited to 5 limbs in our experience and has not been especially successful.

Primary PTA, therefore, seems appropriate for highly selected patients with localized lesions and ia a useful adjunct for patients who are without appropriate autogenous tissue. For example, patients with autogenous tissue limited to short segments of lesser saphenous, arm or residual greater saphenous vein may benefit from balloon dilatation of proximal SFA lesions in an effort to provide a more distal origin for a short vein graft across the knee.

Atherectomy

The suboptimal results associated with infrainguinal PTA and prosthetic material for small-caliber arterial reconstruction have generated a variety of mechanical "atherectomy" devices. These devices remove atheroma by directly engaging the plaque and leave behind a heterogenous luminal surface which differs from that remaining after surgical endarterectomy. The various catheter tips used to engage the plaque are positioned either percutaneously in the angiography suite or under fluoroscopic or angioscopic control in the operating room.

The first such device to be approved by the FDA was the Simpson Peripheral AtheroCath (Devices for Vascular Intervention, Inc., Redwood City, CA). The AtheroCath consists of a cylinder fitted with a fixed terminal guidewire located at the end of a catheter. Within the partially open cylinder there is enclosed a circular cutting element fixed to a central drive shaft that rotates at relatively slow speeds (approximately 1,800 rpm). The cylinder is positioned within a stenotic arterial segment, and after withdrawing the cutter into the proximal end of the cylinder housing, an eccentric balloon is inflated. The housing is thus fixed against the adjacent atheroma and the rotating cutter advanced such that the excised material is

impacted into a storage compartment in the tip of the cylinder for subsequent withdrawal. This device minimizes the risks of peripheral emboli by removing the debris and of intimal dissection by virtue of the guidewire. The AtheroCath may require repeated passages to achieve success, however, and is not as effective when used for a totally occluded artery. An initial experience with 26 patients was reported wherein 51 stenotic lesions requiring 131 passes reduced luminal stenosis from a mean of 79% to 22%.[12] This preliminary experience was then expanded to include 136 lesions in 61 patients for whom atherectomy proved initially successful in 87%.[13] In 30 of these individuals, follow-up angiography after 6 months demonstrated significant recurrent stenosis in the majority of patients unless the initial degree of stenosis was reduced to less than 30%. In the latter case, the rate of recurrent stenosis was only 18%.[2]

The second atherectomy device approved by the FDA was the Kensey Catheter (Theratek, Inc., Miami, FL), which employs two entirely different mechanisms to effect both atherectomy and possibly dilatation. First, a cam tip secured to a central drive shaft is housed within an 8-French flexible catheter and rotates at speeds up to 100,000 rpm, pulverizing atheroma into microparticles. The second mechanism consists of a powered infusion system that spins a radial jet of fluid from the rotating tip against the wall, which not only dilates the lumen but also serves to center the tip and provide much-needed cooling. The advantages of this catheter include its efficacy in vessels totally occluded by either atheroma or thrombus. The catheter is positioned only once within the artery and is advanced steadily until the desired result is achieved. The original device had no guidewire, and the risk of perforation appeared higher than with other devices. The fate and consequence of the pulverized material remain uncertain but have had no apparent clinical impact beyond an occasional distal embolus. While initial experimental results indicated this material may indeed produce microinfarctions, none were of clinical significance in the canine model.[14] In a collective experience of over 100 patients in which this catheter was originally used, only one clinically significant distal embolus was observed. A representative clinical experience, initially reported in a group of 20 patients, has been updated to include 42 patients in which balloon-assisted transluminal atherectomy was attempted in the superficial femoral artery.[15,16] The lesions included complete occlusions and stenotic segments ranging from 1–20 cm in length. Claudication was the primary indication in 33% of extremities, while severe rest pain or tissue necrosis was present in 67%. All procedures were carried out under regional anesthetic in the operating room with open technique and fluoroscopic control. The catheter was successfully passed in 31 of the 46 (67%) and provided initial hemodynamic improvement in 27 (59%) following adjunctive transluminal balloon angioplasty of the residual lumen. Unsuccessful passages resulted from perforation in 11 (25%), only one of which required suture repair, but there were no instances of embolization observed. The incidence of

perforation employing the most recent catheter design, however, was lower (15%). The 1-year cumulative patency rate achieved for these 42 patients appears limited to 37%: if the technically unsuccessful cases are excluded, the 1-year rate increases to 64%. A second single center experience reported from Strasbourg, France, includes 46 balloon-assisted atherectomies in 44 patients using the 8-French Kensey device.[17] The initial technical success rate of 76% was achieved with a lower incidence of perforation (9%) but with three (7%) documented embolic episodes, one of which required popliteal embolectomy. The 1-year patency rate among the 20 eligible patients was 70%, similar to the 64% reported by the Norfolk group if the technically unsuccessful cases are excluded. It is important to note that these results in both recent series were dependent upon adjunctive balloon angioplasty, some form of postoperative antiplatelet therapy, and in the case of the French experience, "antivitamin K" therapy.

The peripheral applications for this device, currently named the Trac-Wright System (Dow Corning Wright, Inc., Arlington, TN) are under continued evaluation, and several design modifications are in progress. The catheter has been made more flexible, a guidewire has been incorporated, and a smaller caliber 5-French configuration is now available.

A third atherectomy catheter under investigation is the Auth Rotablator device consisting of a burr secured to a central drive shaft capable of rotating at speeds up to 150,000.[18] The rotating tip consists of a brass burr into which diamond chips have been embedded. The diameter of the burr ranges from 1.25 to 4.5 mm. The turbine mechanism is powered by compressed air, which also pumps a cooling solution to the tip. The system employs a central guidewire that can be independently manipulated and over which the burr is advanced. In theory, soft viscoelastic tissue is deflected by the tip while hard, fixed atheromatous tissue allows the diamond chips to engage. The primary advantage of the Auth device is a reduced incidence of perforation and the restoration of an adequate lumen of 4 to 4.5 mm in diameter. It is not, however, as effective as the Trac-Wright system in the setting of totally occluded vessels, and its use of necessity results in microembolization of pulverized particles. The incidence of restenosis remains significant. Of the nine patients initially treated in the UCLA experience, four patients developed significant restenosis within 6 months.[19] This restenosis rate is at variance with that reported by Ginsberg, et al., who documented successful results in 19 of 21 atherectomized vessels. After 5 months the restenosis rate was a minimal 10%.[20]

A fifth device has recently been approved for use in the peripheral vasculature and is manufactured by Interventional Technologies, Inc. The conical cutting tip passes over a central guidewire, and debris is constantly aspirated, reducing the chances for microembolization. The incidence of perforation appears quite low at present but more complete follow-up data is required. According to the manufacturer's literature, the TEC peripheral atherectomy system has been used in 93 patients with 143 lesions in 12

clinical centers thus far. As is true with most series, the majority of lesions treated were located in the superficial femoral artery. The initial reported success rate of 92% required adjunctive balloon angioplasty for 35 lesions. The TEC system was initially able to reduce the degree of arterial stenosis by a mean of 59%. Six-month follow-up is available in 57 of the 107 cases, but no consistent angiographic follow-up data is available.

The Bard rotary atherectomy device (Bard, Inc., Cardiovascular Division, Billerica, MA) consists of a cylindrical cutting tip affixed to the end of a flexible catheter rotating at speeds up to 1,500 rpm and is advanced through a lesion over a guidewire. An auger on the guidewire allows retrieval of the incised atheromatous material through the central portion of the catheter. The prime advantages of this catheter appear to be related to low rotational speeds and reduced heat generation; a minimal tendency for perforation since it is guided by a central wire; and the fact that the atheromatous debris is actually removed. The device may not, however, prove efficacious for totally occluded vessels. Preliminary clinical trials are ongoing under an Investigational Device Exemption.

Summary

Provided the hazards of perforation and distal embolization are minimized, the long-term efficacy of recanalization with these atherectomy devices will depend upon the fate of the remaining luminal surface. In the setting of small caliber vessels, it seems unlikely that sustained patency will result without adjunctive pharmacologic control of the fibrous intimal hyperplastic response. Well-controlled prospective studies are much needed to define the role of these instruments in the management of peripheral vascular disease.

References

1. Maini BS, Mannick JA. Effect of arterial reconstruction on limb salvage: a ten-year appraisal. Arch Surg. 1978;113:1297.
2. Kacoyanis GP, Whittemore AD, Couch NP, Mannick JA. Femorotibial and femoroperoneal bypass vein grafts. Arch Surg. 1981;116:1529–1534.
3. Donaldson MC, Mannick JA, Whittemore AD. Femoral-distal bypass with in situ greater saphenous vein: long-term results using the mills valvulotome. Ann Surg. 1991 (in press).
4. Leather RP, Shah DM, Chang BB, Kaufman JL. Resurrection of the in situ saphenous vein bypass. Ann Surg. 1988;208;435–442.
5. Fogle MA, Whittemore AD, Couch NP, Mannick JA. A comparison of in situ and reversed saphenous vein grafts for infrainguinal reconstruction. J Vasc Surg. 1987;5:46–52.
6. Bandyk DF, Kaebnick HW, Stewart GW, Towne JB. Durability of the in situ

saphenous vein arterial bypass: a comparison of primary and secondary patency. J Vasc Surg. 1987;5:256–268.
7. Taylor LM Jr, Edwards JM, Porter JM. Present status of reversed vein bypass grafting: five-year results of a modern series. J Vasc Surg. 1990;11:193–206.
8. Pomposelli FB, Jepsen SJ, Gibbons GW, Campbell DR, Freeman DV, Miller A, LoGerfo FW. The efficacy of the dorsalis pedis bypass for limb salvage in diabetic patients. J Vasc Surg. 1990 (in press).
9. Whittemore AD, Kent KC, Donaldson MC, Couch NP, Mannick JA. What is the proper role of polytetrafluoroethylene grafts in infrainguinal reconstruction? J Vasc Surg. 1989;10:299–305.
10. Veith FJ, Gupta SK, Ascer E, et al. A six-year prospective multicenter randomized comparison of autologous saphenous vein and expanded polytetrafluoroethylene grafts in infrainguinal arterial reconstructions. J Vasc Surg. 1986;3:104–114.
11. Kent KC, Whittemore AD, Mannick JA. Short-term and mid-term results of an all-autogenous tissue policy for infrainguinal reconstruction. J Vasc Surg. 1989;9:107–114.
12. Simpson JB, Zimmerman JJ, Selmon MR, et al. Transluminal atherectomy: initial clinical results in 27 patients. Circulation. 1986;74:Suppl 2:203 (abstract)
13. Simpson JB, Selmon MR, Robertson GC, et al. Transluminal atherectomy for occlusive peripheral vascular disease. Am J Cardiol. 1988;61:96G–101G.
14. Kensey KR, Nash JE, Abrahams C, Zarins CK. Recanalization of obstructed arteries with a flexible, rotating tip catheter. Radiology. 1987;165:387–389.
15. Snyder SO Jr, Wheeler JR, Gregory RT, et al. Kensey catheter—early results with a transluminal endarterectomy tool. J Vasc Surg. 1988;8:541–3.
16. Cull DL, Feinberg RL, Wheeler JR, Synder SO Jr, Gregory RT, Gayle RG, Parent FN III. Atherectomy and laser-assisted balloon angioplasty: boon or bust? J Vasc Surg. 1991 (in press).
17. Desbrosses D, Petit H, Torres E, Barrionuevo D, Figueroa A, Wenger JJ, Ramenah B, Kieny R. Percutaneous atherectomy with the Kensey catheter: early and midterm results in femoropopliteal occlusions unsuitable for conventional angioplasty. Ann Vasc Surg. 1990;4:550–552.
18. Ahn SS, Auth D, Marcus DR, et al. Removal of focal atheromatous lesions by angioscopically guided high-speed rotary atherectomy. J Vasc Surg. 1988;7:292–300.
19. Ahn SS. The rotablator—high speed rotary atherectomy. In: *Endovascular Surgery*. WS Moore, SS Ahn, eds. WB Saunders Co., Philadelphia, 1989, pp. 327–335.
20. Ginsberg R, Jenkins N, Wright A, et al. Transluminal rotational atherectomy: clinical experience in 20 patients. (Abstract) Circulation. 1978 (Suppl II):II-415, 1988.

24
New Developments in Limb Salvage Infrainguinal Arterial Surgery and Underlying Patterns of Disease

FRANK J. VEITH

Introduction

The modern era of direct surgical therapy for lower extremity arterial insufficiency due to arteriosclerosis began barely more than four decades ago with the introduction of femoral artery endarterectomy by dos Santos in 1947[1] and vein bypass by Kunlin in 1949.[2] Since then, advances in surgical technique, diagnostic radiology and patient management have fostered a more aggressive attitude toward limb salvage.[3] Many patients with extensive obliterative arteriosclerotic disease, who were previously advised to undergo primary amputation, are now considered candidates for operative revascularization.

General Approach

Patients with limb-threatening ischemia have multisegmental arteriosclerotic disease and present with severe, unrelenting rest pain, nonhealing ischemic ulcers or frank gangrene. A diagnostic arteriogram is obtained in all patients presenting to us at this stage with few exceptions. Approximately 4% of our patient population are not considered candidates for revascularization due to their nonambulatory status or when gangrene is already extensive, in which case a primary amputation is usually recommended. Of the patients who do have an arteriogram, nearly 6% will not have distal arterial outflow suitable for bypass, and primary amputation may be necessary. Overall, therefore, 90% of the patients presenting to us with limb-threatening ischemia will undergo an attempt at limb salvage.

Revascularization is no longer excluded in patients because of advanced age,[4] poor medical risk, previous contralateral amputation, or gangrene extending to the midfoot or heel. Similarly, revascularization is seldom excluded because of unfavorable anatomy in the distal arterial tree. Ideally, it is important to have adequate distal outflow, but occlusive disease of all three crural vessels or the absence of a patent pedal arch is no longer

considered a contraindication to attempted revascularization. Femoropopliteal bypasses have been successfully performed even when the popliteal artery ends in an isolated or "blind" segment.[5] Collateral branches arising from such blind segments are sufficient to allow blood flow to the leg and foot and to maintain patency. When indicated, bypasses to the distal tibial or dorsalis pedis arteries are now commonly performed. The plantar and lateral tarsal arteries as well as other pedal branches have also been used successfully as sites for distal anastomosis.[6] Greater experience and technical advances have allowed the performance of bypasses to diseased crural and pedal arteries even when they are heavily calcified circumferentially.[7]

We believe that an aggressive approach to limb salvage, including use of modern arteriographic techniques, revascularization, extensive debridement of wounds as necessary and reoperation when indicated, produces vital periods of limb salvage and palliation with low cost in major amputation, morbidity and mortality. This approach, in patient population that includes many referrals for secondary operations, has resulted in an overall limb salvage rate of 75% at one year and 66% at five years.

Best Graft: Vein vs. PTFE

The advantages of using autologous saphenous vein routinely in preference to prosthetic grafts has been widely debated. In a prospective, randomized study[8] comparing autologous saphenous vein (ASV) to polytetrafluoroethylene (PTFE) grafts for femoropopliteal bypasses, a significant difference in patency at 48 months was attained only for bypasses to the below-knee popliteal artery (76% ASV vs. 54% PTFE; $p < 0.05$). Although a trend favoring vein grafts was apparent, no statistically significant difference in patency was obtained for bypasses to the above-knee popliteal artery (61% ASV vs. 38% PTFE; $p > 0.05$), nor was there any significant difference in limb salvage at 48 months whether the bypass was above (78% ASV vs. 77% PTFE; $p > 0.9$) or below (75% ASV vs. 62% PTFE; $p > 0.1$) the knee.

A review of bypasses to the infrapopliteal arteries showed vein grafts to have a significantly better *primary* patency rate at 48 months than PTFE grafts (49% ASV vs. 12% PTFE; $p < 0.001$). Limb salvage, however, again was not significantly different (57% ASV vs. 61% PTFE; $p > 0.05$).

Although PTFE grafts may be acceptable for bypasses above the knee, we preferentially use an autologous vein graft for all bypasses when it is available and adequate. If the greater saphenous vein is unavailable or inadequate, the lesser saphenous or the cephalic veins[9] may be harvested. If necessary, vein may be harvested from the contralateral leg if its circulation is deemed adequate to support healing of the surgical incision. When autologous vein is not available, we prefer PTFE grafts as the best alternate. Although the use of PTFE grafts to distal vessels remains controversial, our results and others[10] indicate that this option is feasible. In the

absence of an adequate vein, we believe that a PTFE graft bypass, even to the crural and pedal arteries, is a better option than amputation.

Distal Origin Grafts

The common femoral artery has generally been regarded as the optimal site of origin for bypasses to the popliteal and infrapopliteal arteries. In selected patients, however, we have found the preferential use of the superficial femoral, deep femoral or popliteal arteries as inflow sources for distal bypasses to be a valuable adjunct in our operative management.[11,12] When these vessels are normal or only minimally diseased proximal to the origin of the bypass, six-year patency rates are equivalent to those for similar operations performed with the common femoral artery as the inflow source.

There are several advantages to the preferential use of a distal inflow source in selected patients. One may avoid dissection in obese, scarred or infected groins. Vein utilization increases, especially in those instances when a limited portion of acceptable vein remains. The shorter length of the bypass may decrease the extent and duration of the operation. Finally, if infection occurs in the area of the proximal anastomosis, management is greatly simplified since continuity of flow to the proximal deep femoral artery is never threatened.

Short Vein Grafts

The use of distal sites for origin of inflow has resulted in late graft patency and limb salvage rates for short vein bypasses that are superior to those of more standard operations (11), particularly in the presence of a disadvantaged outflow tract.[13] Tibiotibial vein bypass grafts yielded a 91% salvage rate and 71% patency rate at 42 months.[14] A patency rate of 81% at 24 months was achieved with short vein bypasses to foot branches (6).

Thus, a short vein bypass is a good operation when vein is limited and may be better than longer grafts in unfavorable circumstances. In addition, although proponents of in situ saphenous vein grafts maintain that these fare better than reversed vein grafts of equal caliber to disadvantaged outflow tracts, the ability to use an ectopic portion of vein often makes this procedure feasible when an in situ bypass is not.

Failing Graft Concept

Infrainguinal bypasses usually result in limb salvage, however, many of these bypasses ultimately fail and the limb may again be threatened. Graft failure may be caused by lesions within an autologous vein graft, anas-

tomotic neointimal hyperplasia, or inflow and outflow lesions produced by progression of atherosclerosis. Close follow-up of patients with physical examination and noninvasive studies, in addition to queries of recurrent symptomatology, is essential to detect hemodynamically significant lesions prior to graft thrombosis.[15,16]

Although reoperation following graft thrombosis can frequently salvage the limb, the procedures required are often complex and technically demanding.[17] Conversely, if the treatment for any of these lesions can be undertaken prior to graft thrombosis, they may often be corrected or improved by percutaneous transluminal angioplasty or a simple operation.[16] Therefore, complete arteriography with visualization of arteries from the renals to the pedal arch is obtained in any patient with recurrent ischemic symptoms or who demonstrates a deterioration in the status of their vascular reconstruction manifested by diminution of distal pulses or noninvasive laboratory studies.

The advantages of early intervention in the management of such "failing" grafts were clearly demonstrated in a study[17] comparing patency rates after reoperation for failed or thrombosed grafts versus patent but hemodynamically failing grafts. A two-year patency rate of 83% was achieved following reoperation for "failing" femoropopliteal extraanatomic bypasses, whereas only 23% of those operated on for thrombosed grafts were still patent two years later.

Unusual Approaches to Infrainguinal Arteries

Even though almost all operations at our institution are performed for limb salvage, subsequent failure of the bypass may not threaten limb viability. In some patients, gangrene and infection is healed by the original operation, and the limb remains intact when the bypass occludes. However, patients with occluded grafts and threatened limbs will require a secondary operation if amputation is to be avoided. Results with these secondary procedures can be surprisingly good.[3]

As secondary operations are required more frequently, it is increasingly common to encounter patients in whom the standard surgical approaches are rendered difficult or impossible because of extensive surgical scarring or persistent infection in a previous incision.

To facilitate reconstructive surgery in the presence of infection or surgical scarring, we have used the following unusual approaches: The *middle and distal portions of the deep femoral artery* were directly approached anteriorly in the midthigh for outflow in 35 cases or inflow in 17 cases.[12] The *above-knee popliteal artery (PA) was approached laterally* in 11 cases, and the *below-knee PA was approached laterally* with resection of the proximal fibula in 10 cases.[18] When the PA was approached laterally for outflow, the bypass inflow was derived from the descending thoracic artery in

two cases, the ipsilateral axillary artery in three cases, the abdominal aorta in one case, the retroperitoneal external iliac in two cases, and the femoral artery in three cases. In 14 cases the *anterior tibial* (five cases), *posterior tibial* (three cases) or *peroneal* (six cases) arteries were *approached laterally*[19] with resection of the fibula. In 10 cases the *medial or lateral plantar branches of the posterior tibial artery* were approached medially for graft insertion. In five cases the *lateral tarsal branch of the dorsalis pedis artery* was approached via a dorsal flap incision for bypass insertion. In four cases the *metatarsal arch branch of the dorsalis pedis artery* was approached anteriorly with resection of the shafts of the metatarsal bones. All these approaches provide access to previously dissected arteries via virginal tissue planes. They are particularly useful for secondary operations after previous bypass failures and permit limb salvage that would otherwise be difficult or impossible.

Conclusions

Treatment of patients with peripheral vascular disease and limb threatening ischemia is both challenging and demanding. The key to limb salvage is commitment, which is often multidisciplinary; the graft is only one small factor. At our institution and many others, an aggressive approach to limb salvage has been greatly rewarding, and we believe that few patients should undergo primary amputation without an attempt at revascularization.

References

1. Dos Santos JC. Sur la desobstruction des thromboses arterielle anciennes. Mem Acad Chir. 73:409–11, 1947.
2. Kunlin J. Venous grafts in therapy of endarteries obliterans. Arch Mal Coeur. 42:371–4, 1949.
3. Veith FJ, Gupta SK, Samson RH, et al. Progress in limb salvage by reconstructive arterial surgery combined with new or improved adjunctive procedures. Ann Surg. 194:386–401, 1981.
4. Scher L, Veith FJ, Ascer E, et al. Limb salvage in octogenarians and nonagenarians. Surgery. 99:160–5, 1986.
5. Veith FJ, Gupta SK, Daly V. Femoropopliteal bypass to the isolated popliteal segment: Is polytetrafluoroethylene graft acceptable? Surgery. 89:296–303, 1981.
6. Ascer E, Veith FJ, Gupta SK. Bypass to plantar arteries and other tibial branches: An extended approach to limb salvage. J Vasc Surg. 8:434–41, 1988.
7. Ascer E, Veith FJ, White-Flores S. Infrapopliteal bypasses to heavily calcified rock-like arteries. Management and results. Amer J Surg. 152:220–13, 1986.
8. Veith FJ, Gupta SK, Ascer E, et al. Six year prospective multicenter randomized comparison of autologous saphenous vein graft and expanded

polytetrafluoroethylene grafts in infrainguinal arterial reconstructions. J Vasc Surg. 3:104–14, 1986.
9. Kakkar VV. The cephalic vein as a peripheral vascular graft. Surg Gynecol & Obstet. 128:551–6, 1969.
10. Flinn WR, Rohrer MJ, Yao JST, et al. Improved long term patency on infragenicular polytetrafluoroethylene grafts. J Vasc Surg. 7:685–90, 1988.
11. Veith FJ, Gupta SK, Samsom RH, et al. Superficial femoral and popliteal arteries as inflow sites for distal bypasses. Surgery. 90:980–90, 1981.
12. Veith FJ, Nunez A, Gupta SK, et al. Direct approaches to the middle and distal portions of the deep femoral artery for use as sites of origin or termination for secondary bypasses. In J Goldstone, ed. *Perspectives in Vascular Surgery*, St. Louis, Quality Medical Publishing, 1988.
13. Ascer E, Veith FJ, Gupta SK, et al. Short vein grafts: A superior operation for arterial reconstructions to poor or compromised outflow tracts? J Vasc Surg. 7:370–8, 1988.
10. Veith FJ, Ascer E, Gupta SK, et al. Tibiotibial vein bypass grafts: A new operation for limb salvage. J Vasc Surg. 2:552–7, 1985.
15. O'Mara CS, Flinn WR, Johnson ND, et al. Recognition surgical management of patent but hemodynamically failed arterial grafts. Ann Surg. 194:467–76, 1981.
16. Veith FJ, Weiser RK, Gupta SK, et al. Diagnosis and management of failing lower extremity arterial reconstructions prior to graft occlusion. J Cardiovasc Surg. 25:381–4, 1984.
17. Veith FJ, Gupta SK, Daly V. Management of early and late thrombosis of expanded polytetrafluoroethylene (PTFE) femoropopliteal bypass grafts: Favorable prognosis with appropriate reoperation. Surgery. 87:581–7, 1980.
18. Veith FJ, Ascer E, Gupta SK, Wengerter KR. Lateral approach to the popliteal artery. J Vasc Surg. 6:119–23, 1987.
19. Dardik H, Dardik I, Veith FJ. Exposure of the tibial-peroneal arteries by a single lateral approach. Surgery 75:337, 1974.

25
Preliminary Clinical Experience with Polyurethane Vascular Prostheses in Femorodistal Reconstruction

PHILIPPE G. BULL, HELMUTH DENCK, ROBERT GUIDOIN, and HELMUTH GRUBER

Abstract

After promising results in animal experimentation, implantation of polyurethane vascular prostheses for femoral reconstruction was performed in 15 patients during a 10-month period. This prospective study was carried out to evaluate patency, limb salvage and complications associated with this type of small diameter conduit for arterial occlusive diseases. Indications for operation included acute and chronic ischemia, nonhealing ulceration, or gangrene. They were five early occlusions, which resulted in a 1-month primary patency rate of 66% and a secondary patency rate of 80%. The mean limb salvage rate at one month was 80% and at this stage of the study, 66%. Infection occurred in one patient following multiple graft thrombectomy attempts. There were no statistically significant differences in patency rates between proximal and distal popliteal graft anastomoses. Two critical phases of healing, in which the graft was prone to flow deterioration, were identified at 4 to 6 weeks and at 4 to 6 months. Electron microscopic studies revealed early endothelial cell formation even under poor hemodynamic conditions, minimal graft-host reaction and good structural resistance at the suture site. Further intensive investigations, however, are required to identify patient groups who would benefit from such a prosthesis.

Introduction

Autologous saphenous vein is the most reliable graft conduit currently in utilization for lower limb revascularization.[1–3] However, since almost a third of peripheral vascular interventions are reoperations, the vein can often be absent or unsuitable as arterial grafting.[4] Many substitutes have gone through extensive clinical trial, but it is apparent that none have been proven to be as effective as the vein.[5–7]

TABLE 25.1. Individual case summaries.

Patients No	Initial	Age	Sex	Previous surgery	Adjunctive surgery	Run-off	Anastomosis Proximal	Distal	Caliber	Complication	Follow-up
1.	J.G.	66	M	Iliac PTA	Profundaplasty	1	CFA	POP I	6	none	12
2.	K.P.	63	M		Profundaplasty	1	CFA	POP I	6	none	dead 10
3.	W.S.	77	M		Iliac PTA	1	CFA	POP III	6	none	11
4.	V.H.	65	F		Profundaplasty	1	CFA	POP III	5	none	11
5.	W.G.	54	M	Fempop	Profundaplasty	1	CFA	POP III	6	occlusion	BKA
6.	F.N.	75	M			1	CFA	POP I	6	occlusion	POP III VJG
7.	H.F.	61	F		POP III PTA	1	CFA	POP I	6	none	9
8.	M.P.	70	F		Profundaplasty	3	CFA	POP I	6	none	8
9.	F.W.	65	M	Aortobifemoral AFS-PTA	PP + POP III PTA	2	Prosthesis	POP I	6	occlusion	Anterior VJG
10.	H.R.	68	F			1	CFA	Peroneus	5	occlusion	BKA
11.	K.B.	53	M	Aortobifem BP Fempop VBP		2	Prosthesis	POP I	5	occlusion infection	AKA
12.	R.B.	53	M	Fempop VBP		1	CFA	POP III	6	occlusion	BKA
13.	J.U.	68	M	Iliac PTA		1	CFA	POP III	6	none	7
14.	W.S.	66	M		POP-TEA	2	CFA	POP I	6	occlusion	Anterior VJG
15.	R.K.	53	M	Fempop VBP PP		1	EIA	POP III	5	occlusion at 2 months	AKA

VBP = venous bypass, PTA = percutaneous transluminal angioplasty, TEA = thrombenarterectomy, CFA = common femoral artery, SFA = superficial femoral artery, EIA = external iliac artery, AKA = above-knee amputation, BKA = below-knee amputation, POP = popliteal artery, POP I = proximal third, POP III = distal third.

Over the past few years polyurethane synthetic materials have been introduced in the field of investigation as a promising substitute for small caliber grafts. They are reported to be relatively nonthrombogenic, but material degradation over time with aneurysmal dilatation was seen early on.[8] Molecular alteration has produced a medical grade polyurethane with better stability and improved resistance to oxidative processes in the biological environment.[9] Promising results in the animal model prompted us to evaluate this new kind of synthetic graft as an arterial substitute.[10–12]

The objective of this study was to evaluate prospectively the early patency rate, graft-host compatibility, and healing process associated with this new synthetic graft.

Patients and Methods

Patient selection was performed under the prerequisites that the autogenous saphenous vein be either absent or unusable, that a chronic or acute ischemia be present, and that a written consent to take part in this study be obtained. The study protocol was sanctioned by the Ethic Commission on Human Experimentation of the Municipal Hospital of Vienna. The pertinent data of all 15 patients (12 men, 3 women) are summarized in Table 25.1. The average age was 63.8 years (range, 53 to 77 years). All underwent unilateral bypass. Associated diseases and risk factors are shown in Table 25.2.

The indications for operation were severe claudication (grade I, category 3) in two cases; acute ischemia (grade II) in four cases, chronic ischemia in three cases, and nonhealing ulceration or gangrene (chronic ischemia, grade III) in six cases.

All patients underwent conventional angiography preoperatively to determine the quality of runoff tibial vessels. In most cases this examination was completed with a selective digital subtraction angiography to visualize

TABLE 25.2. Associated disease and risk factors.

Disease	No	%
Heart disease	5	33
Hypertension	10	66
Diabetes	6	40
Cerebrovascular insufficiency	6	40
Renal insufficiency	3	20
Dysfibrinogenemia	2	13
Paraneoplasmic syndrome	2	13
Smoking	7	47

the pedal arch. Runoff was considered acceptable if at least one tibial or peroneal artery was patent.

Prosthesis

The structural and physical properties of this type of prosthesis have previously been described in detail (9). Briefly, the prosthesis is made by electrostatic spraying of liquid polyurethane on a rotating mandrill, giving the prosthesis a fibrous structure and flexibility. The hydrophilic wall is porous for air but not for cellular elements of the blood. Porosity is produced by phase separation during manufacturing. Very minimal porosity for plasma is present, which makes preclotting unnecessary. The slightly elastic nature of the prosthesis makes it easy to handle surgically. The two types of prosthesis employed had an internal diameter of 6 mm and 5 mm, respectively, whereby the latter was externally reinforced by removable rigid polyurethane rings.

Operative Procedure

All operations were performed following the same scenario: the distal anastomosis was performed first in end-to-side manner—in one case a short segment of autogenous vein was used to form a Linton patch. The sites of the proximal and distal anastomoses are shown in Table 25.1. All anastomoses were performed using continuous monofilament polypropylene sutures, 7-0 for the distal and 6-0 for the proximal anastomosis. The proximal anastomosis was constructed end-to-end to include the origin of the deep femoral artery, creating a profundal augmentation plasty. The anastomoses were performed under factor 2 magnifying glasses. Intraoperative systemic heparinization was achieved with a bolus of 5000 IU heparin at clamping and maintained postoperatively with 20,000 IU per day. Subsequent long-term oral anticoagulation was given if not contraindicated, otherwise a combination of 250 mg acetylsalicylic acid and 25 mg dipyridamol was given. Each patient received a prophylactic dose of 2 g Cefamandol (Mandokef, Lilly) 1 hour before surgery.

Follow-up

Outcome of the procedure was judged by the relative degree of clinical improvement, combined with changes in the ankle/brachial index (ABI), conforming to the guidelines set forth by the Society for Vascular Surgery–International Society for Cardiovascular Surgery.[13] Follow-up examinations were performed by one of the authors (PG Bull) at 1 and 3 months

postoperatively, thereafter every 6 months. Graft patency was confirmed by duplex examination and measurements of distal pressure. Angiography was indicated only if signs of impending graft failure were present.

Statistical Analysis

Life tables for clinical success and limb salvage rates were constructed by life table analysis with calculation of the standard error as recommended by Peto et al.[14] Statistical analyses of the patency rates were performed with the log-rank test. Cumulative patency rates were estimated by the Kaplan-Meier method.

Results

There was no peroperative mortality. One patient (No. 15) suffered a cardiac arrest in the immediate postoperative phase and required resuscitation. Following this the limb was ischemic for a period of 1 hour but recompensated under conservative therapy. Angiographic examination at 4 days demonstrated a patent graft. During the follow-up period one patient (No. 3) died of myocardial infarction at 9 months with the graft patent. One patient suffered a myocardial infarction of the posterior wall at 6 months requiring 2 weeks of CCU. At follow-up, 6 weeks later, the Doppler pressure index had fallen to preoperative value, but the graft was patent in the

TABLE 25.3. Arterial pressure (ankle-brachial indices).

			At follow-up (months)		
Patient no	Preoperative	Postoperative	1	3	6
1.	0.1	0.5	0.6	0.6	0.6
2.	0.0	0.7	0.6	0.5	0.5
3.	0.25	0.8	0.8	0.8	0.8
4.	0.4	0.5	0.5	0.5	0.5
5.	0.6	0.45	AMP		
6.	0.65	0.8	1.0	1.0	1.0
7.	0.6	0.9	0.6	0.7	0.7
8.	0.6	1.5	1.5	1.5	1.5
9.	0.3	1.0	0.3	0.3	0.3
10.	0.0	nm	0.0	AMP	
11.	0.0	0.0	0.0	AMP	
12.	0.3	0.0	0.0	AMP	
13.	0.5	1.0	1.0	1.2	1.2
14.	0.3	0.5	0.2	1.0	
15.	0.6	1.0	0.9	AMP	

duplex examination and gangrene had healed. A treadmill test was not performed because of low cardiac output (see Table 25.3).

Graft Patency

Primary patency is shown in the actuarial curve in Table 25.4. There were five occlusions within the first 4 weeks, resulting in a primary patency rate of 66%. Restoration of patency by thrombectomy alone did not succeed in maintaining the graft patent in any patient. In case No. 5, a femoropopliteal venous graft was implanted 11 years previously and showed intensive aneurysmatic degeneration. Only one tibial artery with multiple stenoses was viewed in the angiogram. Graft occlusion occurred on the seventh postoperative day, and fibrinogen activity was very high with a concentration of 694 mg/dl. In case No. 11 graft occlusion occurred on the day (12) the patient was to be discharged. A series of thrombectomy was attempted without success. Aneurysmatic degeneration of a 7-year-old venous graft bypass, implanted because of bilateral popliteal aneurysms with peripheral embolization, was also the indication for surgery in case No. 12. Occlusion occurred on the 18th day after an uneventful recovery from bilateral femoral reconstruction. Fibrinogen concentration was not measurable, having passed the peak of 700 mg/dl at this stage. A scanning electron micrograph (SEM) of the explanted graft revealed patches of semiendothelialization at the anastomosis site (Fig. 25.1). Indication for surgery in case No. 14 was acute thrombosis following femoral PTA. Graft occlusion was observed on the 10th postoperative day, and thrombectomy was attempted twice without success. Subsequently, a venous jump graft bypass to the tibiofibular trunk was performed with success.

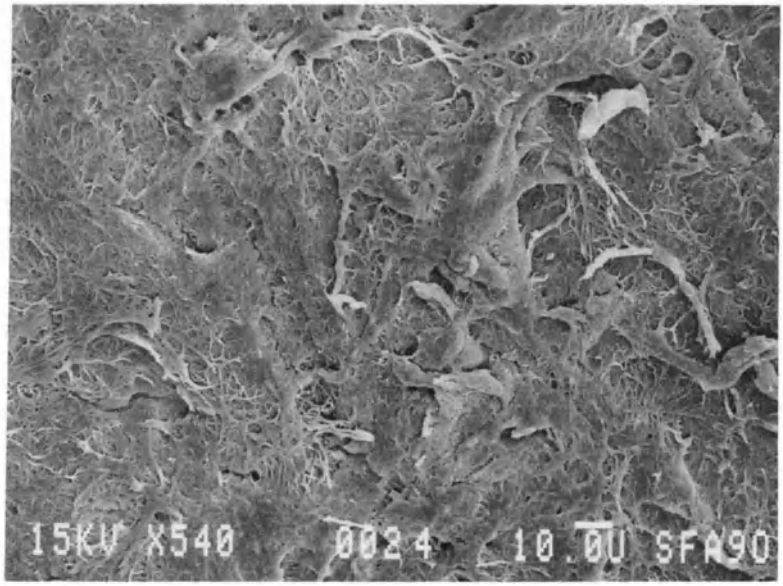
The fifth occlusion (case No. 9) was due to a technical failure in which the prosthesis was pulled accidentally through the adductor muscle. A retransposition of the prosthesis graft was performed successfully. The patient was discharged with a patent graft as documented by angiography, but returned 6 weeks later with a reocclusion. Rethrombectomy was not attempted because it was thought that collaterals could compensate the ischemia, and the patient was put on prostaglandin infusion.

Patient No. 6 complained of rest pain 4 weeks after discharge. Graft occlusion was diagnosed and thrombectomy attempted, but reocclusion occurred in the immediate postoperative phase. A venous jump graft to the distal popliteal segment was performed thereafter with success. Histologic examination revealed fibrotic thrombus adhesion to the prosthetic wall with mural infiltration of foreign-body giant cells (Fig. 25.2).

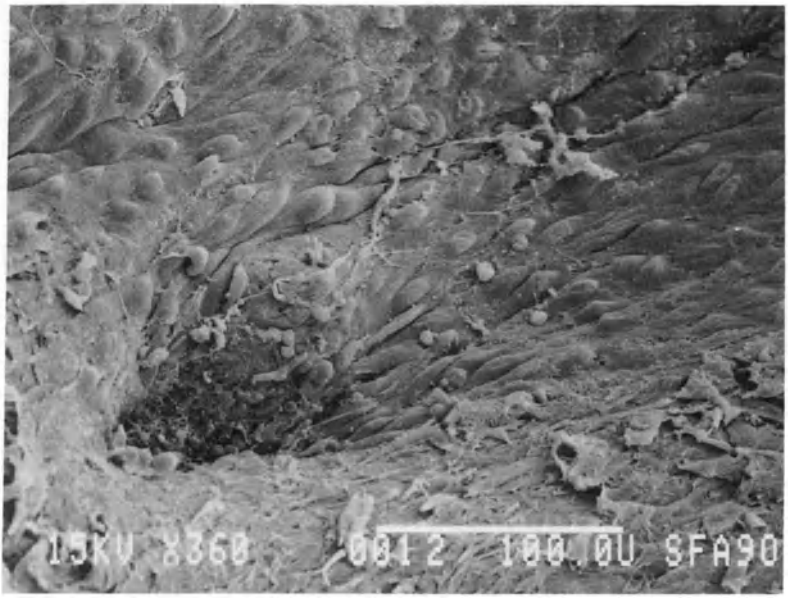
At 6 weeks early graft occlusion occurred in patient No. 10, who had up to then significant amelioration of her gangrenous limb. Lytic therapy was started but interrupted because of intolerable pain resulting in below-knee amputation. SEM studies showed a normal anastomosis. The polyurethane

TABLE 25.4. Primary patency rates.

Interval months	No. at risk	No. failed	Amputation AK	Amputation BK	Withdrawn duration	Patients lost	Due died	Interval patency	Cummulative patency	Standard error
0–1	15	5	1	2	0	0	0	0.66	0.66	0.025
1–2	10	2	0	2	0	0	0	0.80	0.53	0.036
2–3	8	1	0	0	1	0	0	0.87	0.46	0.042
3–6	6	0	0	0	0	0	0	1.0	0.46	0.056
6–9	6	0	0	0	3	0	0	1.0	0.46	0.084
9–12	3	0	0	0	1	0	1	1.0	0.46	0.11
10–12	1	0	0	0	2	0	0	1.0	0.46	0.33



A



B

FIGURE 25.1. Scanning electron micrograph of an occluded polyurethane vascular graft at 18 days with: **(A)** coagulate in the immediate vicinity of the anastomosis, **(B)** despite early subjacent cicatrization with patches of semiendothelialization, compacted fibrin **(C)** is present everywhere.

C

structure was filled with cellular blood elements captured between the graft's fibers (see Fig. 25.3).

One other patient (No. 7) also had a deterioration in arterial pressure at 3 months although the graft was still patent. Case No. 15 had advanced foot gangrene and low cardiac output. Occlusion occurred 2 months postoperatively, at which time the patient was taken over by another institute where an above-knee amputation was performed.

Limb Salvage

Occluded graft remained in spite of thrombectomy in three cases, and led to five amputations. The limb salvage rate at 1 month was 86%, overall limb salvage at this stage of the study is 66%.

Infection

One case of superficial wound infection was documented, which healed without further complication with topical antiseptic therapy. In case No. 11 *Staphylococcus aureus* infection occurred after three unsuccessful thrombectomy attempts (Fig. 25.4). The graft reoccluded the following week. Local excision and drainage were insufficient, and the infection propagated to the bifurcation graft bypass, requiring explantation, and ended with limb loss after 4 months.

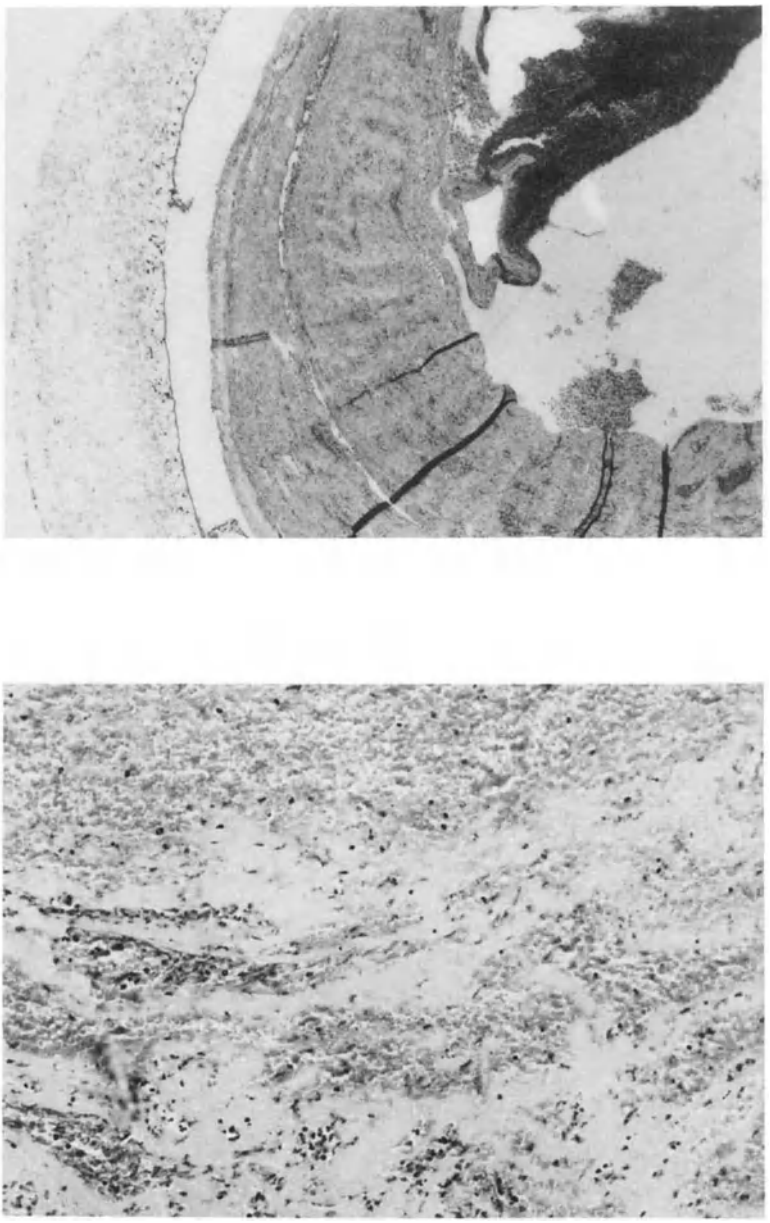
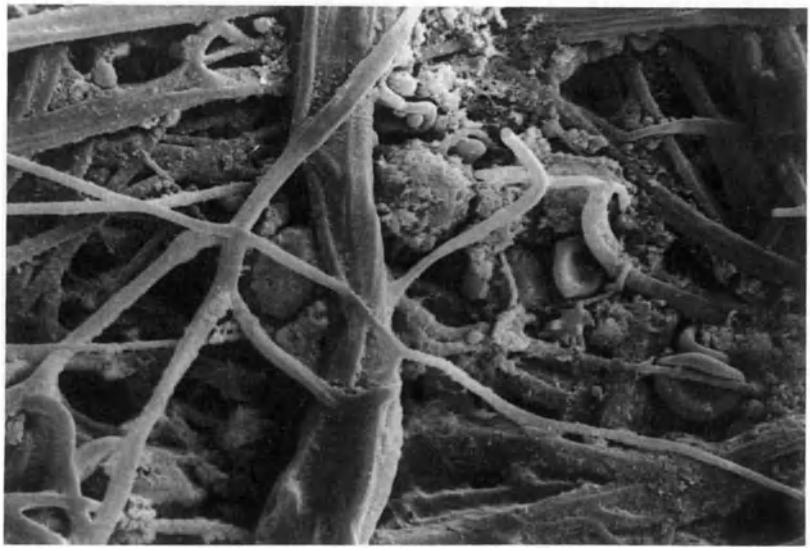


FIGURE 25.2. HE histologic section of distal anastomosis occluding at 4 weeks: Extensive fibrin and cellular elements covering the inner surface of the polyurethane graft with blood-filled residual lumen.

A



B

FIGURE 25.3A–C. Anastomotic area of occluded polyurethane graft (SEM): The anastomosis is normal; the inner structure of the graft is filled with cellular blood elements and fibrin. The external support shows a tendency to compress the outer porous structure.

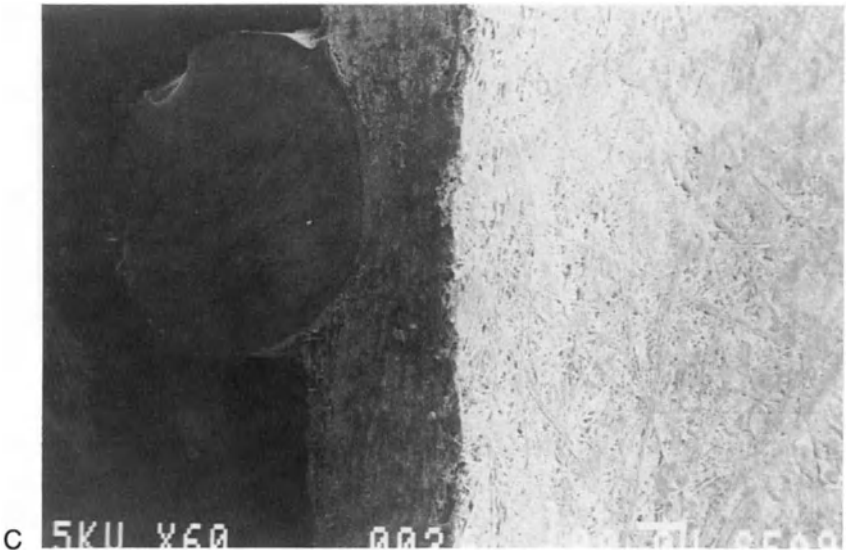


Fig. 25.3C

Discussion

Autologous saphenous vein remains, when available, the optimal small caliber graft. Adequate graft patency was noted in veins with as little as 3.0 mm external diameter.[15] A recent study reviewed all pertinent literature on femoropopliteal grafting in order to obtain patency rate estimates for grafts in different situations.[16] The reported results confirmed the difference in long-term patency between vein and prosthetic grafts and the tendency for a high initial failure rate with a slower, almost constant, attrition rate over subsequent years. Above-knee grafts had an average occlusion in the first year of 20.2%, and prosthetic grafts 23.5%. Below-knee vein grafts had a reported rate of 24.3%, and 39.0% was reported for synthetic grafts. This report is interesting insofar as it can be used as a reference when comparing different graft materials.

The goal is to develop an arterial synthetic substitute that will maintain patency in a diameter of less than 4 mm and with a flow of 50 ml/min.[17] Up to this moment, no synthetic graft material can achieve this performance.

Polyurethane is composed of soft polyether groups and hard urethane-urea groups. Their biocompatibility is partly due to alternation between soft and hard segments. In polymer with predominantly soft components, protein and conformational variations of fibrinogen adsorption onto the surface is increased, whereas if the polymer contains more hard segments, the biocompatibility is increased due to reversible bonding of hydrogen of the secondary amide groups with peptides of fibrinogen.[18]

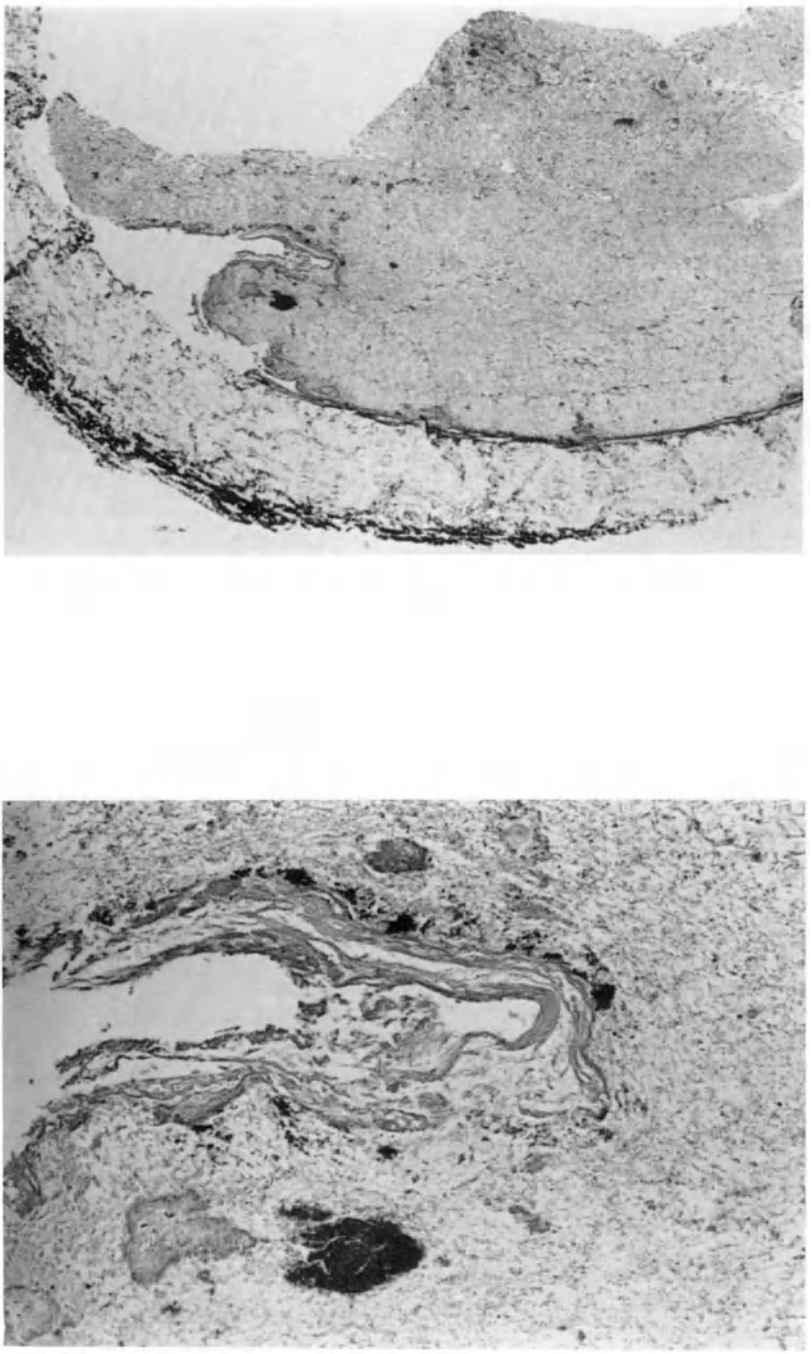


FIGURE 25.4. Occluded polyurethane graft with *Staphyloccoccus aureus* infection in the thrombus.

On the present evidence, no indication is given that polyurethane has a superior patency to other types of synthetic graft material. On the contrary, excessive pannus may form at the proximal anastomosis, as detected on duplex sonography, resolving in 4 to 6 weeks, as detected in two patients, but might reoccur in the following months. Furthermore, obstructive fibrin deposits might be responsible for the deterioration of ankle/brachial pressure with flow disturbances at the site of the proximal anastomosis seen in two patients at 4 and 10 months follow-up, respectively. The occurrence of occlusive thrombi at different times of implantation suggests alternation of the fibrinolytic and thrombogenic phases as observed along this study period and seems to correspond to earlier reports in animal study.[19]

The patency rate reported in our series was 66% at 1 month and 47% at 1 year. Although the failure rate at 1 year was much higher than expected (four of eight above-knee and four of seven below-knee graft bypasses) as compared to the expected average-year failure of 23.5% and 39%, respectively (reported by Michaels), statistical evidence was not enough to reject the hypothesis that the new material is equally good at the 5% level of significance (x^2-test). The log-rank test did not reveal a significant difference at the 5% level between proximal and distal anastomosis. This, however, may be due to the small number of patients in the two groups. Factors found to be associated with decreased patency were poor runoff and a hypercoagulable state that did not respond to heparin or antivitamin K medication.

Conclusions

The authors conclude that, although this type of graft has many interesting features, its general use cannot be recommended until additional investigations are performed. Further studies on protein absorption on the surface of the graft, complement activation, platelet aggregation and fibrinogen consumption, as well as intensive structural testing, are required before definitive recommendation can be made.

References

1. Veterans Administration Cooperative Group 141. Comparative evaluation of prosthetic, reversed, and in-situ vein bypass grafts in distal popliteal and tibial-peroneal revascularization. Arch Surg. 1988;123:434–438.
2. Böhmig HJ, Zeidler G, Berenberg-Goßler J, Schmoller F. Free orthograde saphenous vein bypass: technique and results. Angio Archiv 1989;17:97–101.
3. Leather RP, Shah DM, Chang BB, Kaufman JL. Resurrection of the in-situ saphenous vein bypass: 1000 cases later. Ann Surg 1988;208:435–42.
4. Gosselin C, Guidoin R, Hould F. Evolution of vascular surgery in Quebec 1978–1988. Ministry of Health, Government of Quebec.

5. Veith FJ, Gupta SK, Ascer E, et al. Six-year prospective multicenter randomized comparison of autologous saphenous vein and expanded polytetrafluoroethylene grafts in infrainguinal arterial reconstructions. J Vasc Surg 1988;3:104–114.
6. Batt M, Avril G, Gagliardi JM, et al. Femorodistal bypass using the chemically processed human umbilical vein graft: 9-year experience. Can J Surg. 1990;33(1):61–65.
7. Bull PG, Hagmüller GW, Hold M, Wandschneider W, Denck H. Clinical comparison of biological prosthesis for femorodistal arterial reconstruction: ovine collagen biograft vs human umbilical vein. In: Chang J, ed, *Modern Vascular Surgery*, New York: PMA Publishing Corp, 1990, pp. 269–278.
8. Gruss JD, Braun B, Lehnhardt FJ, Grande PU. Experimental study of a new prosthetic graft. J Cardiovasc Surg. 1981;22:18.
9. Braun B, Grande PU, Lehnhardt FJ, Jerusalem C, Hess F. Herstellung und tierexperimentelle Untersuchung einer kleinlumigen mikroporösen Polyurethangefäßprothese. Vasa 1988; Suppl 22 (entire issue).
10. Hess F, Jerusalem C, Braun B, Grande PU. Three years experience with experimental implantation of fibrous polyurethane microvascular prosthesis in the rat aorta. Microsurgery. 1985;6:155–162.
11. Hess F, Steeghs S, Jerusalem C, Braun B, Grande PU. Implantation of 20 cm long polyurethane vascular prosthesis in the femoral artery of dogs. Thorac Cardiovasc Surgeon. 1988;36:348–500.
12. Hess F, Steeghs S, Braun B, van Det R, Grande PU, Jerusalem C, Skotnicki S. Patency rate of small caliber fibrous polyurethane vascular prostheses implanted in the dog carotid and femoral artery improved by use of acetylsalicylic acid and dipyridamol. Thorac Cardiovasc Surgeon. 1988;36:221–226.
13. Ad Hoc Committee on Reporting Standards, SVS/ISCVS. Suggested standards for reports dealing with lower extremity ischemia. J Vasc Surg. 1986;4:80–94.
14. Peto R, Pike MC, Armitage P, Breslow NE, et al. Design and analysis of randomized clinical trials requiring prolonged observation of each patient. Br J Cancer. 1976;34:585.
15. Wengerter KR, Veith FJ, Gupta SK, Ascer E, Rivers SP. Influence of vein size (diameter) on infrapopliteal reversed vein graft patency. J Vasc Surg. 1990;11:525–531.
16. Michaels JA. Choice of material for above-knee femoropopliteal bypass graft. Br J Surg. 1989;76:7–14.
17. Callow AD. Problems in the construction of a small diameter graft. Inter Angio. 1988;7:246–53.
18. Stupp SI, Kaufmann JW, Carr SH. Interactions between segmented polyurethane surfaces and the plasma protein fibrinogen. J Biomed Mat Res. 1977;11:237–250.
19. Charara J, Beaudoin G, Fortin C, Guidoin R, et al. In vivo biostability of four types of arterial grafts with impervious walls; their haemodynamic and pathological characteristics. J Biomed Eng. 1989;11:416–428.

26
Effects of Morphology of Distal Anastomosis Immediately After Surgery on Intimal Hyperplasia in Femoropopliteal Bypass Graft

KENSUKE ESATO, NOBUYA ZEMPO, MASAKI O-HARA, KENTAROH FUJIOKA, TAKAYUKI KUGA, and HIROAKI TAKENAKA

Summary

Intimal hyperplasia of the distal anastomosis was studied angiographically in 26 patients (31 limbs) who underwent femoropopliteal bypass graft and had more than 1 year patency.

Mean follow-up was 32 months (range, 12–105 months) for autologous saphenous vein graft (ASV group, 15 cases) and 24 months (range, 12–64 months) for polytetrafluoroethylene graft (EPTFE group, 16 cases). The stenotic rate (SR) was calculated from digital subtraction arteriograms and rounded off to 0, 10, 25, 50 or 75%. SR was measured both immediately after surgery and at follow-up in 7 of 15 cases in the ASV group and in 11 of 16 cases in the EPTFE group. Some stenosis of the distal anastomosis was noted in 8 of 15 cases (53.3%) in the ASV group and in 8 of 16 cases (50.0%) in the EPTFE group. The SR in patients with measurements both immediately after surgery and at follow-up, which was 0% in 6 and 10% in one case immediately after surgery, was 0% in 4 cases, 10% in 2 cases, and 25% in 1 case at follow-up. In the EPTFE group, SR immediately after surgery was 0% in 7, 10% in 2, and 25% in 2 cases of 11 cases with measurements both immediately after surgery and at follow-up. SR at follow-up, however, was 0% in 4, 10% in 3, 50% in 3, and 75% in 1 case.

Technical factors causing stenosis when the anastomosis is created appear to be the primary risk factor for intimal hyperplasia as a cause of late graft failure.

The etiology of femoropopliteal bypass vein or EPTFE graft failure remains to be elucidated fully. Generally speaking, early graft failure usually is attributed to technical problems, and late graft failure to progression of atherosclerosis in native vessels proximal or distal to the graft or intimal hyperplasia at the anastomosis. However, the details of these processes are unknown.

This study examines intimal hyperplasia at the distal anastomosis after femoropopliteal bypass and attempts to determine which factors affect it.

Materials and Methods

Primary femoropopliteal arterial bypass was performed in 80 limbs (65 patients) from 1981 to 1989 (age range, 48 to 81 years; mean, 67.5 year). Autologous saphenous vein graft (ASV group) was used in 37 limbs (above-knee in 26 and below-knee in 11) and polytetrafluoroethylene graft (EPTFE group) in 43 limbs (above-knee in 19 and below-knee in 15). Among these, 26 patients (31 limbs) had graft patency for at least 1 year and had undergone arteriography within recent 1 year. Twenty-three patients (88 percent) were men. The indication for arterial reconstruction was claudication in 11, ischemic rest pain in 2, and nonhealing ischemic ulcer in 2 patients in the ASV group; and was claudication, ischemic rest pain, and ischemic ulcer in 9, 4 and 3 cases in the EPTFE group, respectively. The distal anastomosis was above-knee in 14 cases and below-knee in one case in the ASV group, and above-knee in 13 cases and below-knee in 3 cases in the EPTFE group. Mean follow-up was 32 months (range, 12–105 months) in the ASV group and 24 months (range, 12–64 months) in the EPTFE group (Table 26.1).

Intravenous digital subtraction angiography (IVDSA) was performed in the outpatient department to examine changes of distal anastomosis and confirm patency. Contrast material (total volume, 30–35 ml) was administered into the radial vein at a rate of 13 ml/sec. Angiograms were obtained in a lateral projection.

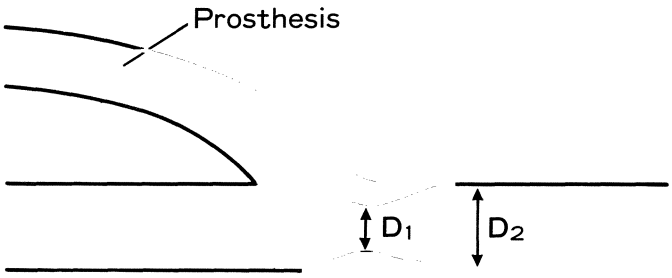
The stenotic rate (SR) was calculated using the following formula:

$$SR(\%) = \frac{D_2 - D_1}{D_2} \times 100$$

where D_2 is the internal diameter of normal host artery distal to the distal anastomosis, and D_1 is the smallest internal diameter of the stenotic host

TABLE 26.1. Severity of symptoms and site of distal anastomosis in patients with patent femoropopliteal bypass.

	Fontaine			Distal anastomotic site (knee)		
	II	III	IV	Above	Below	Follow-up period (months)
GSVG (n = 15)	11	2	2	14	1	32
EPTFE (n = 16)	9	4	3	13	3	24



$$\text{Stenotic Rate (\%)} = \frac{D_2 - D_1}{D_2} \times 100$$

FIGURE 26.1. Formula and schema for calculating the stenotic rate at the distal of femoropopliteal bypass graft:
D_2 = internal diameter of normal distal host artery;
D_1 = internal diameter of maximal stenosis area of the host artery just distal to the anastomosis.

artery just distal to the distal anastomosis (Fig. 26.1). The SR was calculated both immediately after surgery and during follow-up in 7 of 15 cases in the ASV group and 11 of 16 cases in the EPTFE group.

The stenotic rate was rounded off to 0, 10, 25, 50 or 75% (Fig. 26.2).

Results

The cumulative life-table primary patency rate was calculated in all cases (Fig. 26.3). No significant difference between the ASV and EPTFE groups was observed for up to 3 years after operation (64.0% vs. 71.3%, respectively).

Eight of 15 limbs (53.3%) showed some stenosis of distal anastomosis in the ASV group; the SR was 10% in 2, 25% in 3, and 50% in 3 cases. In the EPTFE group, 8 of 16 limbs (50.0%) demonstrated anastomotic stenosis; the SR was 10% in 3, 25% in 1, 50% in 3, and 75% in 1 case (Table 26.2).

TABLE 26.2. Stenotic rate of distal anastomotic site.

	Stenotic rate %				
	10	25	50	75	Total (%)
ASV (n = 15)	2	3	3		8(53.3)
EPTFE (n = 16)	3	1	3	1	8(50.0)

ASV: autologous saphenous vein; EPTFE: expanded polytetrafluoroethylene

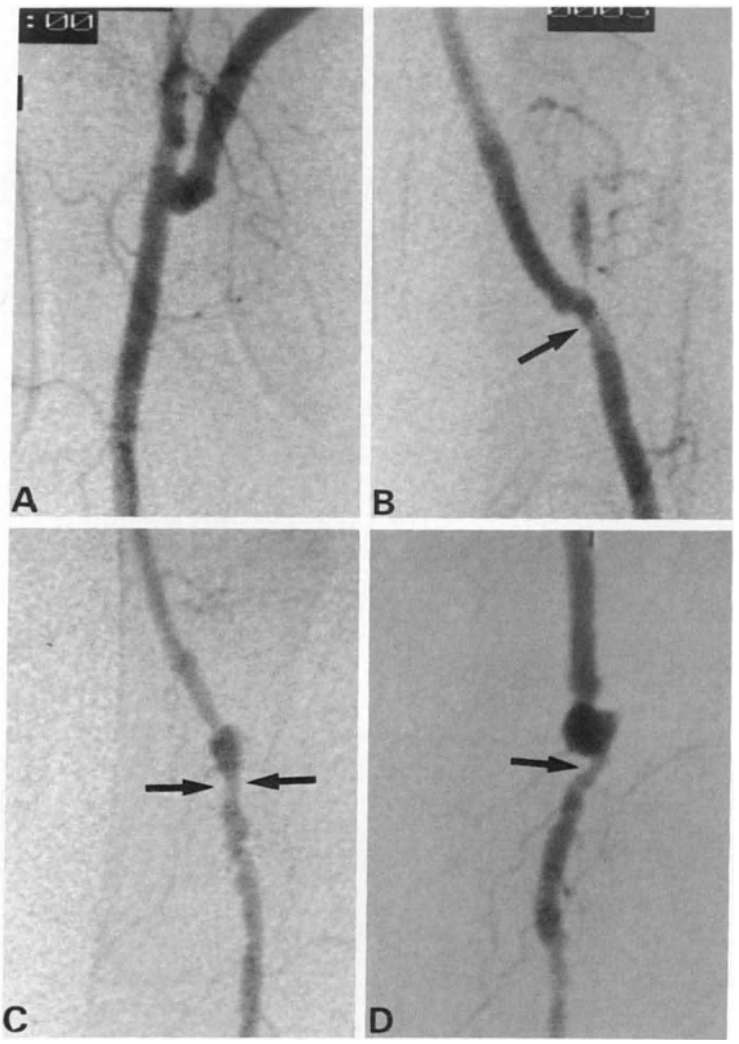


FIGURE 26.2. Arteriograms showing percent stenosis. Stenosis was rounded off to 0(**A**), 10(**B**), 25(**C**), or 50(**D**)%. Arrows indicate stenosis.

The SR immediately after arterial reconstruction in the ASV group was 0% in 6 and 10% in one limb. At follow-up, the SR in these limbs was 0% in 4, 10% in 2, and 25% in one case. The limb with the SR of 25% at follow-up was 10% immediately after reconstruction. Of 11 limbs in the EPTFE group, the SR was 0% in 7, 10% in 2, and 25% in 2 cases immediately after surgery. At follow-up, however, the SR was 0% in 4, 10% in 3, 50% in 3, and 75% in one case. The limbs in which the SR was greater

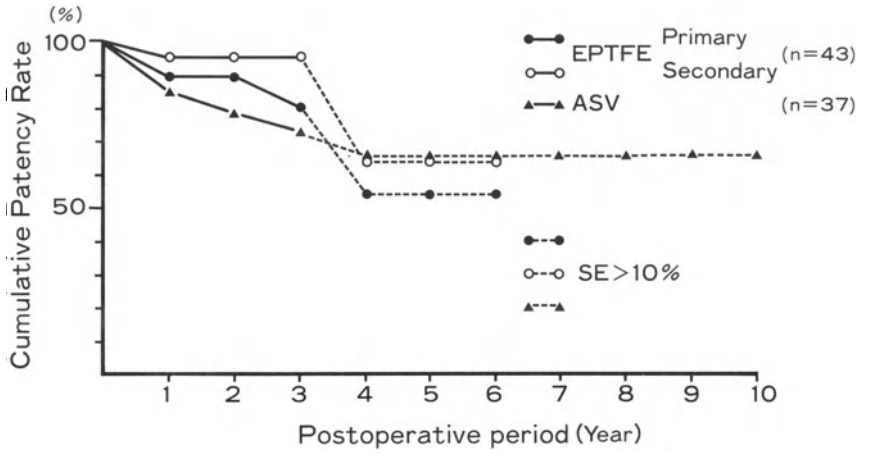


FIGURE 26.3. Cumulative life-table patency rates for autologous saphenous vein (ASV) and polytetrafluoroethylene (PTFE) femoropopliteal bypass grafts. No significant difference in patency rate existed between the ASV and EPTFE groups.

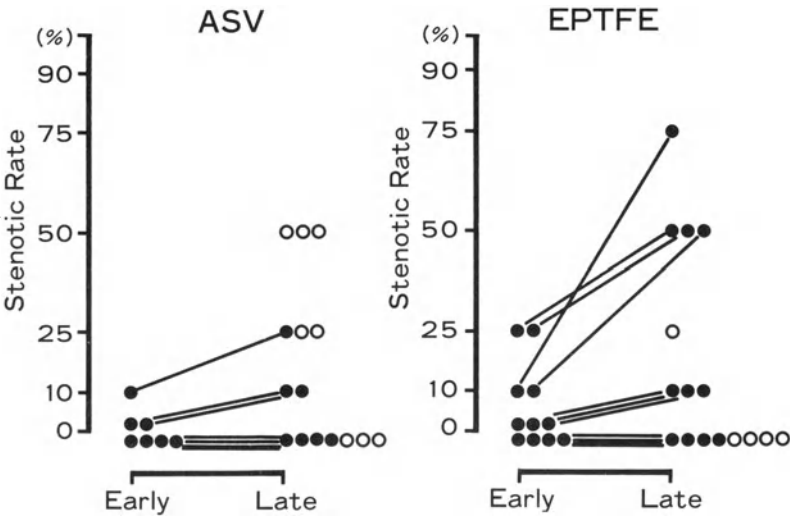


FIGURE 26.4. Comparison of stenotic rate of distal anastomosis immediately after arterial reconstruction and at least one year after surgery in patients with femoropopliteal bypass graft. Patients with slight stenosis of the distal anastomosis immediately after surgery showed high grade stenosis at follow-up. Open circles indicate patients who underwent angiography only at follow-up.

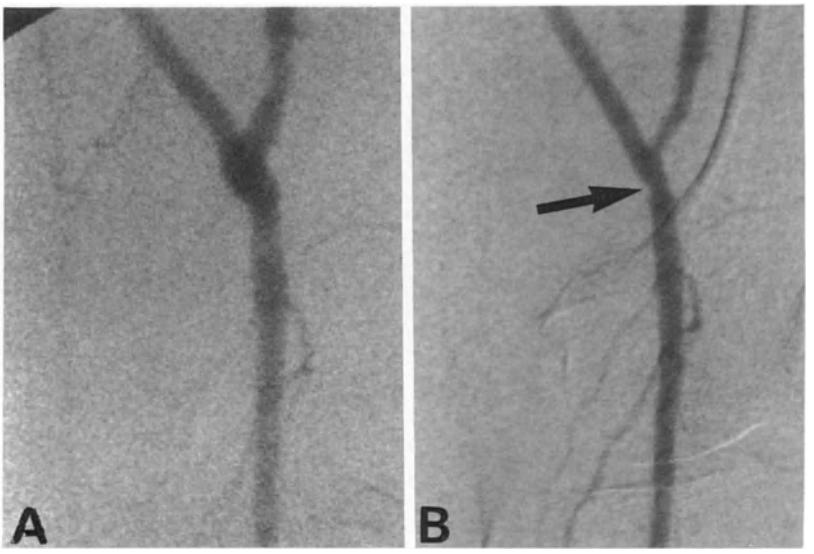


FIGURE 26.5. Arteriograms showing morphologic changes of distal anastomosis in ASV graft. Distal anastomosis, which was no stenosis immediately after surgery (**A**), showed stenosis of 10% at the toe 12 months after surgery (**B**). Arrows indicate stenosis.

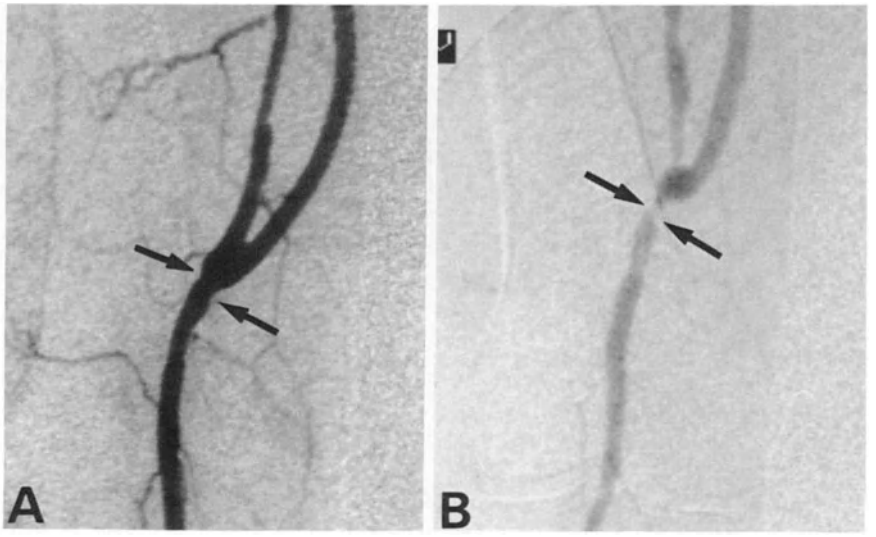


FIGURE 26.6. Arteriograms showing morphologic changes of distal anastomosis in EPTFE graft. The stenotic rate, which was 25% immediately after surgery (**A**), increased to 50% 12 months after surgery (**B**). Arrows indicate stenosis.

than 10% immediately after reconstruction had high grade stenosis, more than 50%, at follow-up (Fig. 26.4).

Changes in distal anastomotic shape in the ASV and EPTFE groups are shown in Figs. 26.5 and 26.6.

Discussion

Despite the generally satisfactory performance of femoropopliteal bypass grafts, a considerable number eventually fail after varying intervals. Important factors correlating with long-term patency are the type of graft used,[1] the site of the distal anastomosis,[2] and arteriographic runoff.[3] According to Veith et al.,[4] EPTFE grafts may be used preferentially for femoropopliteal bypass in selected poor-risk patients because patency rates for ASV and EPTFE grafts are similar for 2 years after operation. However, the overall patency rate for femoropopliteal bypass using EPTFE was inferior to ASV. In our hands the cumulative patency rate for the 2 grafts was similar for 4 years after surgery. This supports the routine use of EPTFE grafts in selected cases. It is believed that long-term antiplatelet therapy results in a high patency rate even with EPTFE femoropopliteal bypass grafts.

Intimal hyperplasia is the most common cause of late graft failure after femoropopliteal arterial reconstruction. Ascer et al.[5] have shown that although no cause for late graft failure could be established in almost one third of cases, intimal hyperplasia was present in 22 of 104 cases (21%) of EPTFE bypass graft occlusion. Periodic arteriography must be performed to determine true incidence of anastomotic intimal hyperplasia. Conventional arteriography is extremely stressful and cannot be performed routinely. However, IVDSA is minimally invasive and can detect morphologic changes at the anastomosis. The incidence of intimal hyperplasia is significantly greater at the distal than proximal anastomosis of in situ vein grafts,[6] and furthermore, distal hyperplasia is more rapidly progressive. This study examined the distal anastomosis only, and intimal hyperplasia originated at the toe of the graft in almost all case.[5]

Almost all patients who developed high grade stenosis of the distal anastomosis had at least slight stenosis immediately after surgery. This highlights the importance of meticulous surgical technique, as even low-grade stenosis will place the patient at risk for eventual graft failure secondary to progressive intimal hyperplasia. Serial changes in morphology and intimal hyperplasia were similar for EPTFE and ASV grafts. Why, then, is the patency rate for ASV grafts higher than for EPTFE grafts? No evidence for a more vigorous reaction by the host artery against EPTFE graft than ASV graft was detected. We believe that the patency rate is most dependent upon the quality of the anastomosis at the time of its creation. Further study is necessary to understand the nature of the host artery's reaction to

the graft and what therapeutic implications such knowledge might bring. Nevertheless, the creation of an unflawed anastomosis remains the surgeon's responsibility.

References

1. Vennesland O, Bay O. Femoropopliteal bypass; factors influencing long term patency. J Oslo City Hosp. 39:137–142, 1989.
2. McLoughlin R, O'Leary G, Fitzgerald LP, O'Donnel JA. The effect of distal anastomotic site on PTFE graft patency in lower extremity bypass.Eur J Vasc Surg. 3:417–419, 1989.
3. Prendiville EJ, Yeager A, O'Donnell TF Jr, Coleman JC, Jaworek A, Callow AD, Mackney WC, Deterling RA. Long-term results with the above-knee popliteal expanded polytetrafluoroethylene graft. J Vasc Surg. 11:517–524, 1990.
4. Veith FJ, Gupta SK, Ascer E, White-Flores SBSN, Samson RH, Scher LA, Towne JB, Bernhard VM, Bonier P, Flinn WR, Astelford PRN, Yao JST, Bergan JJ. Six-year prospective multicenter randomized comparison of autologous saphenous vein and expanded polytetrafluoroethylene grafts in infrainguinal arterial reconstructions . J Vasc Surg. 3:104–114, 1986.
5. Ascer E, Collier P, Gupta SK, Veith FJ. Reoperation for polytetrafluoroethylene bypass failure: The importance of distal outflow site and operative technique in determing outcome. J Vasc Surg. 5:298–310, 1987.
6. Whittemore AD. Late failure of femoropopliteal bypass graft, in Brewster DC (ed); *Common Problems in Vascular Surgery*, Year Book Medical Publishers, Inc, Chicago, 1989, p. 281–288.

27
Femoropopliteal Bypasses to Isolated Popliteal Artery Segments: PTFE vs. Vein Grafts

HARRY B. KRAM, SUSHIL K. GUPTA, and
FRANK J. VEITH

Abstract

A 10-year experience of 217 femoropopliteal bypasses (FPBs) to isolated popliteal artery (IPA) segments in 207 patients is reported. Thirty-three FPBs (15%) were performed with reversed saphenous vein (RSV) and 184 (85%) with polytetrafluoroethylene (PTFE) grafts. Operative indications were gangrene in 121 (56%), nonhealing ulceration in 40 (18%), ischemic rest pain in 51 (24%), and claudication in 5 (2%) cases. The 5-year primary graft patency rate of these bypasses was 59% (RSV = 74%, PTFE = 55%; $p < 0.05$), the secondary 5-year graft patency rate was 61% (RSV = 79%, PTFE = 56%; $p < 0.05$), and the 5-year limb salvage rate was 78% (RSV = 78%, PTFE = 78%). The 30-day operative mortality rate was 10% and the 5-year patient survival rate was 38%. Eleven patients (5%) required lower extremity amputation because of progressive gangrene or extensive infection despite a patent bypass to an IPA segment. Amputation was avoided in 23 patients (11%) by reoperation and secondary graft extension to an infrapopliteal artery with RSV. FPB to an IPA segment combined with graft extension to an infrapopliteal artery with RSV was performed as primary therapy in 19 (9%) patients because of extensive pedal necrosis or infection. We conclude that FPBs to IPA segments: 1) have acceptable 5-year graft patency and limb salvage rates; 2) should be performed with RSV grafts when possible; 3) may be performed with PTFE grafts if necessary, with a resulting limb salvage rate equal to that of RSV grafts; and 4) require sequential extension to an infrapopliteal artery when foot necrosis or infection is extensive. In addition, the presence of an IPA segment is associated with a high operative mortality rate and limited life expectancy due to coronary artery disease.

Introduction

In 1967, Mannick et al.[1] reported that limb salvage could be achieved in patients with an isolated popliteal artery (IPA) segment by performing

a femoropopliteal bypass (FPB) with reversed saphenous vein (RSV). Subsequently, FPBs to IPA segments performed with expanded polytetrafluoroethylene (PTFE) grafts were shown to afford acceptable short-term patency and limb salvage rates.[2] Despite these and other encouraging reports of FPBs to IPA segments,[3–11] the results of FPB are still believed by many to be dependent upon the patency of the runoff bed as determined by arteriography. In addition, there is a dearth of reports of late results with bypasses to IPA segments and a widespread belief that such procedures with PTFE grafts have poor results and should not be performed.

The purpose of the present report is to detail long-term patency and limb salvage results in a series of 217 FPBs to IPA segments. Since most of these procedures were performed with PTFE grafts, it elucidates the value of PTFE grafts when autologous vein is unavailable or of poor quality. In addition, it examines some of the limitations of this operation and emphasizes the need for distal extensions to optimize limb salvage results when foot necrosis or infection are extensive.

Methods

Clinical Data

During the 10-year period 1980 to 1989, 217 FPBs to IPA segments were performed in 207 patients with severe infrainguinal arterial occlusive disease. The indications for operation were gangrene in 121 (56%), nonhealing ulceration in 40 (18%), ischemic rest pain in 51 (24%), and claudication in 5 (2%) cases. The mean age of the patients was 73 ± 11 (1 S.D.) years (range, 38 to 99 years); 103 (49%) were men and 104 (51%) were women. There was a high incidence of associated medical illness, including diabetes mellitus in 142 patients (69%), coronary artery disease in 134 (65%), hypertension in 130 (63%), and a smoking history in 111 (54%).

FPBs to IPA segments originated from the common femoral artery in 160 (74%) and the superficial femoral artery in 57 (26%) cases. The bypass grafts terminated above the knee in 127 (59%) and below the knee in 90 (41%) cases. RSV was used in 33 cases (15%) and PTFE grafts in 184 (85%). Minimum selection criteria for FPB to an IPA segment included 7 cm of patent artery proximal to the terminal arterial occlusion and at least one major collateral branch vessel visualized by arteriography.[2,8] Patients with extensive gangrene of the midfoot or severe organic mental syndromes were excluded from consideration for FPB; these patients usually underwent primary amputation.[7,8,11]

Postoperative Management

Patients were examined every 1 to 3 weeks for the first 3 months after operation and then every 1 to 3 months thereafter. Bypass graft patency

was confirmed by unequivical pulse examination, doppler determined ankle pressure measurements and pulse volume recordings. FPB graft evaluation by Duplex examination or contrast arteriography was performed in patients who had significant changes in any of these parameters or a recurrence of ischemic symptoms.

All patients whose FPB thrombosed within the first 30 days after operation underwent reoperation. Patients whose FPB thrombosed after 1 month were considered for reoperation if the limb was rethreatened and there were no contraindications to another attempt at revascularization. Our strategies for the diagnosis, therapy, and operative management of thrombosed grafts have been described in detail elsewhere.[12,13]

Data Analysis

Primary and secondary graft patency, limb salvage and patient survival were tabulated by the cumulative life table method. All patency intervals were based on results of pulse examinations, vascular laboratory data, or arteriography. Patients who died or required major amputation despite a patent FPB had their patency interval terminated at that time. The significance of differences in graft patency and limb salvage rates between clinical groups was determined by the logrank test.[14] Statistical significance was assumed at the 95% confidence level.

Results

The 5-year primary graft patency rate for FPBs to IPA segments was 59% (Table 27.1). However, the 5-year primary graft patency rate was significantly better in patients who received RSV grafts compared with those who received PTFE grafts (74% vs. 55%; $p < 0.05$) (Fig. 27.1). Similarly, the 5-year secondary graft patency rate for FPBs to IPA segments was 61%, but the secondary 5-year graft patency rate was significantly better in

TABLE 27.1. Primary patency life table for femoropopliteal bypasses to isolated popliteal artery segments (N = 217).

Interval (mos)	At risk	Failed	Duration	Interval patency	Cumulative patency	SEM (%)
0	217	9	24	0.956	0.956	0
6	159	12	38	0.925	0.884	1.9
12	122	10	17	0.918	0.814	3.0
24	72	10	35	0.861	0.723	4.5
36	36	3	29	0.916	0.685	6.4
48	16	3	15	0.813	0.590	9.4
60	7	0	5	1.000	0.590	14.3

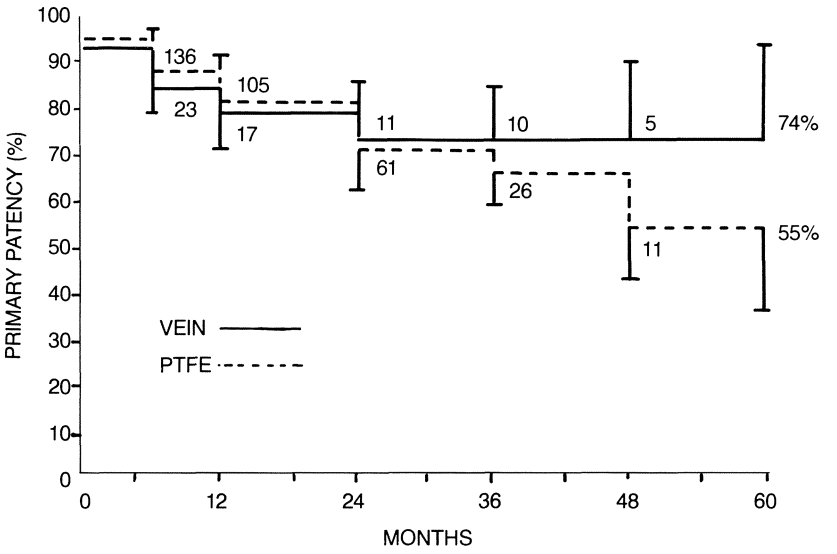


FIGURE 27.1. Cumulative life-table primary patency rates for femoropopliteal bypasses to isolated popliteal artery segments performed with reversed saphenous vein (N = 33) and PTFE (N = 184) grafts. The number at each point represents the number of grafts at risk for that interval, and the bars represent the standard error. There was a significant difference in 5-year primary graft patency rates between the two groups ($p < 0.05$).

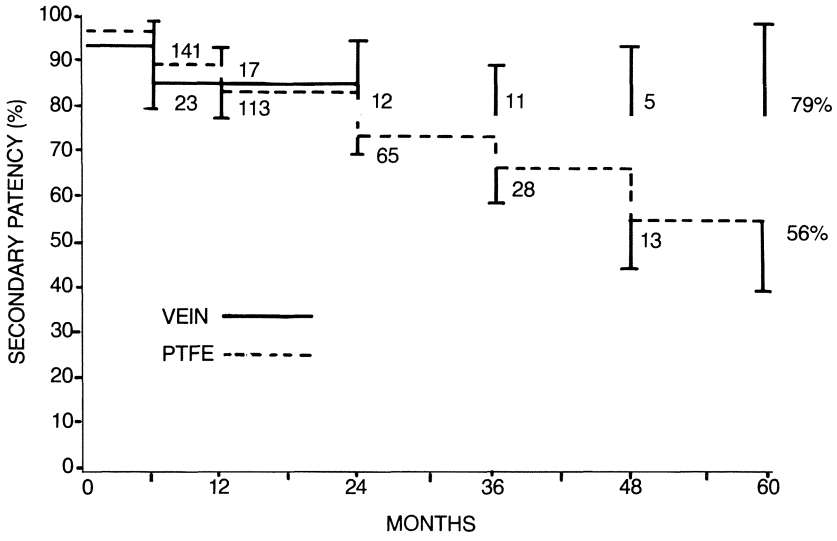


FIGURE 27.2. Cumulative life-table secondary patency rates for femoropopliteal bypasses to isolated popliteal artery segments performed with reversed saphenous vein (N = 33) and PTFE (N = 184) grafts. The number at each point represents the number of grafts at risk for that interval, and the bars represent the standard error. There was a significant difference in 5-year secondary graft patency rates between the two groups ($p < 0.05$).

TABLE 27.2. Limb salvage life table for femoropopliteal bypasses to isolated popliteal artery segments (N = 217).

Interval (mos)	At risk	Amputation	Duration	Interval limb salvage	Cumulative limb salvage	SEM (%)
0	217	3	25	0.985	0.985	0
6	166	9	38	0.946	0.933	1.5
12	133	6	22	0.955	0.890	2.2
24	78	7	40	0.910	0.824	3.8
36	38	3	32	0.921	0.783	5.9
48	16	0	20	1.000	0.783	9.1
60	7	0	5	1.000	0.783	13.8

patients who received RSV grafts compared with those who received PTFE grafts (79% vs. 56%; $p < 0.05$) (Fig. 27.2).

Primary and secondary 5-year graft patency rates were slightly better in patients who underwent above-knee (AK) FPB (60 and 62%, respectively) compared with those who underwent below-knee (BK) FPB (56 and 58%, respectively), but the differences were not statistically significant.

The 5-year limb salvage rate was 78% in patients who underwent FPB to an IPA segment (Table 27.2), but unlike primary and secondary graft

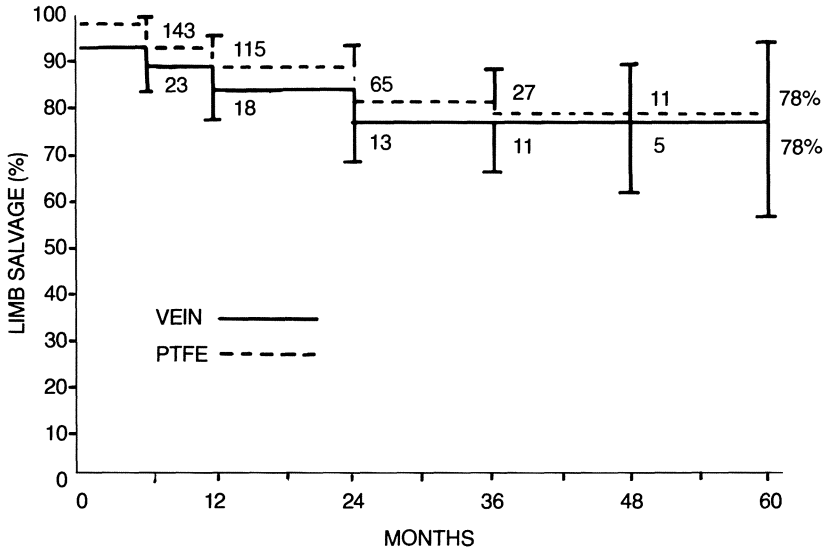


FIGURE 27.3. Cumulative life table limb salvage rates for femoropopliteal bypasses to isolated popliteal artery segments performed with reversed saphenous vein (N = 33) and PTFE (N = 184) grafts. The number at each point represents the number of limbs at risk for that interval, and the bars represent the standard error. The 5-year limb salvage rates of the two groups were identical.

TABLE 27.3. Late results of femoropopliteal bypasses to isolated popliteal artery segments followed for 5-years (N = 217).

	Primary patency (%)	Secondary patency (%)	Limb salvage (%)
Graft			
Reversed saphenous vein	74	79	78
Polytetrafluoroethylene	55	56	78
p value	<0.05	<0.05	NS
Distal anastomosis			
Above-knee	60	62	83
Below-knee	56	58	73
p value	NS	NS	NS

NS = not significant

patency rates, the 5-year limb salvage rates were identical (78%) in patients in whom FPB was performed with RSV or PTFE grafts (Fig. 27.3). However, the 5-year limb salvage rate was better in patients who underwent AK FPB compared with those who underwent BK FPB (83% vs. 73%), but the difference was not statistically significant (Table 27.3).

The 30-day operative mortality rate of patients who underwent FPB to an IPA segment was 10% (21 patients), with most deaths occurring as a result of complications of advanced cardiac disease. The 5-year survival rate of patients in the present series was only 38%. Eleven patients (5%) required major amputation because of progressive gangrene or extensive infection despite a patent FPB to an IPA segment. In 23 patients (11%), amputation was avoided by reoperation and secondary graft extension to an infrapopliteal artery with RSV. FPB to an IPA segment combined with graft extension to an infrapopliteal artery with RSV was performed as primary therapy in 19 patients (9%) because of extensive pedal necrosis or infection.

Discussion

Data from the present retrospective study indicate that FPB to an IPA segment may be performed in patients with limb threatening ischemia with acceptable 5-year graft patency and limb salvage rates. Previously, the durability of bypass operations to popliteal arteries with meager runoff on arteriography was deemed poor, especially when synthetic grafts were used.[5,6] However, the inaccuracy of the arteriogram in estimating actual infrapopliteal runoff has been previously demonstrated by Mundth et al.,[15] who reported a lack of correlation between preoperative arteriography and intraoperative hemodynamic assessment of distal arterial outflow.

Preoperative noninvasive hemodynamic measurements have also been

used to predict long-term FPB graft patency,[16] but data from our institution revealed no differences in preoperative ankle pressures and ankle-brachial pressure indices in patients with and without good distal runoff on arteriography.[8] It is likely that FPB graft failure is a multifactorial problem that defies perfect prediction. Thus, patient selection based on a single expression of distal runoff, be it arteriographic findings or noninvasive hemodynamic measurements, is likely to be misleading.[2,17] Moreover, it is apparent from the present study that the presence of an IPA segment on arteriography should not dictate a pessimistic attitude toward performing FPB for limb salvage.

It is generally accepted that the long-term graft patency rate of FPBs constructed with RSV is superior to that achieved with synthetic graft material.[18] Indeed, our primary and secondary 5-year graft patency rates for RSV grafts to IPA segments were 74% and 79%, respectfully, compared with 55% and 56% for PTFE grafts ($p < 0.05$). However, our results with synthetic grafts conflict with data reported by Corson et al.,[6] who found only a 17% 5-year graft patency rate for prosthetic grafts to IPA segments. However, their series included only 14 synthetic grafts, more than a third of which were constructed with materials other than PTFE. Furthermore, two of their patients received composite grafts constructed of RSV and PTFE, both of which failed within 5 months of operation; the latter patients were included in the synthetic graft group, thereby resulting in a significantly lower 5-year graft patency rate in this small subset of patients.

Based on our favorable experience with the use of PTFE grafts in the management of patients with limb-threatening ischemia, we have often used these grafts for FPBs to IPA segments. A limb salvage rate identical to that obtained with RSV grafts to IPA segments was achieved using PTFE grafts. In addition, the high 30-day operative mortality rate and low 5-year patient survival rate of the present series suggests that patients with IPA segments may have more severe coronary arteriosclerosis and shorter life expectancy than patients with less extensive lower extremity arterial occlusive disease. Because FPBs performed with RSV or PTFE grafts have equivalent primary graft patency rates for up to 2 years,[18] PTFE grafts are preferred over RSV grafts for FPBs to IPA segments in frail high-risk patients in whom a limited life expectancy is probable and a reduction in operating time is desired.

In some patients with an IPA segment and minimal tissue necrosis or rest pain, a dilemma seemingly exists regarding the choice between performing a bypass to the popliteal artery or to a more distal artery. Our preference is to perform a bypass to an IPA segment rather than to a more distal artery, provided the IPA segment is at least 7 cm in length with at least one major collateral branch vessel. This approach is supported by several other studies of patients with IPA segments.[4,10,19] All of these studies revealed better 1-year graft patency rates in patients who underwent FPB com-

pared with those who underwent bypasses to more distal arteries. However, this controversial issue remains unresolved.

On the basis of these observations, our current approach in patients who have minimal tissue necrosis or rest pain, adequate arterial inflow, and an IPA segment measuring at least 7 cm in length with at least one major collateral branch vessel, is to perform an FPB as the initial procedure. Extension of the bypass to an infrapopliteal artery with RSV is reserved for a second procedure in patients whose symptoms fail to resolve following FPB. These sequential bypasses constructed with PTFE and RSV grafts facilitate maximum utilization of limited amounts of adequate vein. Sequential bypasses are also useful in patients who have narrowed or diseased segments of greater saphenous vein, as reduced vein graft diameter has been shown to be associated with decreased infrapopliteal bypass graft patency.[20] In patients who have extensive tissue necrosis or infection, a procedure that supplies direct unobstructed arterial flow to the foot will provide the best chance of healing. This can be accomplished by performing a one-stage sequential femoropopliteal-tibial bypass or a direct femorodistal bypass.[21]

In conclusion, valuable features of FPBs to IPA segments for limb salvage include acceptable late graft patency and limb salvage rates, and identical limb salvage rates when performed with PTFE or RSV grafts. Limitations of FPBs to IPA segments include a frequent need for distal extension of the FPB to an infrapopliteal artery using RSV when foot necrosis or infection is extensive and an association with limited life expectancy.

References

1. Mannick JA, Jackson BT, Coffman JD, et al. Success of bypass vein grafts in patients with isolated popliteal artery segments. Surgery 1967;61:17–25.
2. Veith FJ, Gupta SK, Daly V. Femoropopliteal bypass to the isolated popliteal segment: Is polytetrafluoroethylene graft acceptable? Surgery 1981;89:296–303.
3. Purdy RT, Bole P, Makanju W, et al. Salvage of the ischemic lower extremity in patients with poor runoff. Arch Surg 1974;109:784–6.
4. Davis RC, Davies WT, Mannick JA. Bypass vein grafts in patients with distal popliteal artery occlusion. Am J Surg 1975;129:421–5.
5. Kaufman JL, Whittemore AD, Couch NP, et al. The fate of bypass grafts to an isolated popliteal artery segment. Surgery 1982;92:1027–31.
6. Corson JD, Brewster DC, LaSalle AJ, et al. Comparative analysis of vein and prosthetic bypass grafts to the isolated popliteal artery. Surgery 1982;91:448–51.
7. Veith FJ, Gupta SK, Samson RH, et al. Progress in limb salvage by reconstructive arterial surgery combined with new and improved adjunctive procedures. Ann Surg 1981;194:386–401.
8. Gupta SK, Veith FJ. Fate of patients with a blind popliteal artery segment: Limb loss with a patent femoropopliteal graft. *In*: Veith FJ, ed., *Critical Prob-*

lems in Vascular Surgery. New York: Appleton-Century-Crofts, 1982, pp. 241–50.
9. Brewster DC. Bypass to isolated popliteal artery segment. *In*: Bergan JJ, Yao JST, eds., *Evaluation and Treatment of Upper and Lower Extremity Circulatory Disorders*. Orlando: Grune & Stratton, 1984, pp. 337–49.
10. Darke S, Lamont P, Chant A, et al. Femoro-popliteal versus femoro-distal bypass grafting for limb salvage in patients with an "isolated" popliteal segment. Eur J Vasc Surg 1989;3:203–7.
11. Veith FJ, Gupta SK, Wengerter KR, et al. Changing arteriosclerotic disease patterns and management strategies in lower-limb-threatening ischemia. Ann Surg 1990;212:402–14.
12. Veith FJ, Gupta SK, Daly V. Management of early and late thrombosis of expanded polytetrafluoroethylene (PTFE) femoropopliteal bypass grafts: Favorable prognosis with appropriate reoperation. Surgery 1980;87:581–7.
13. Veith FJ, Gupta SK, Ascer E, et al. Improved strategies for secondary operations on infrainguinal arteries. Ann Vasc Surg 1990;3:85–93.
14. Colton T. *Statistics in Medicine*. Boston: Little, Brown & Company, 1976, pp. 237–50.
15. Mundth ED, Darling DC, Moran JM, et al. Quantitative correlation of distal arterial outflow and patency of femoropopliteal reversed saphenous vein grafts with intraoperative flows and pressure measurements. Surgery 1969;65:197–206.
16. Dean RH, Yao JST, Stanton PE, et al. Prognostic indicators in femoropopliteal artery occlusion. Arch Surg 1975;110:1287–93.
17. Hiatt JG, Raviola C, Baker JD, et al. The limitations of predictability of success of femoro-popliteal bypass grafts. J Vasc Surg 1984;1:617–22.
18. Veith FJ, Gupta SK, Ascer E, et al. Six-year prospective multicenter randomized comparison of autologous saphenous vein and expanded polytetrafluoroethylene grafts in infrainguinal arterial reconstructions. J Vasc Surg 1986;3:104–14.
19. Mason R, Lanfranchi A, Giron F. Isolated popliteal versus distal bypasses for limb salvage. Surg Gynecol Obstet 1982;155:49–53.
20. Wengerter KR, Veith FJ, Gupta SK, et al. Influence of vein size (diameter) on infrapopliteal reversed vein graft patency. J Vasc Surg 1990;11:525–31.
21. Dietzek AM, Gupta SK, Kram HB, et al. Limb loss with patent infra-inguinal bypasses. Eur J Vasc Surg 1990;4:413–7.

28
Intravascular Stent Deployment Across Vein Graft Anastomoses: Acute and Chronic Results

RICHARD F. NEVILLE, YARON ALMAGOR, ANTONIO L. BARTORELLI, RENU VIRMANI, MICHAEL PERLMAN, and MARTIN B. LEON

Abstract

Anastomotic stenosis continues to be an important cause of late graft failure. Intravascular stents may allow the dilatation of these hyperplastic lesions, thereby increasing secondary graft patency rates. To study the acute and chronic effects of intravascular stent deployment across vein graft anastomoses, we implanted balloon expandable stents in 26 femoral venous bypass grafts in 13 sheep. Six weeks after graft construction, intravascular stents (17 slotted, 9 coiled) were deployed across each anastomosis. The stented vein grafts were then harvested immediately or at 3 weeks, 8 weeks, and 6 months postdeployment, with arteriograms obtained and stented segments processed for scanning electron microscopy. Total stent patency was 92%; 88% (15/17) slotted, 100% (9/9) coiled. One slotted stent remained patent despite the presence of chronic thrombus, and distal migration occurred in 33% of coiled stents. Extrinsic compression caused stenoses in 2 stents although both remained patent. Slotted stents exhibited greater endothelialization than coiled stents at 3 and 8 weeks while both were completely covered at 6 months. Stent-vessel wall contact averted thrombosis, migration, and delayed endothelialization. We conclude that stents across vein graft anastomoses demonstrate excellent patency with complete endothelial coverage and minimal intimal proliferation. Stent-vessel wall contact is a critical factor for successful deployment.

Introduction

Anastomotic stenosis with subsequent thrombosis is an important cause of late failure in arterialized vein grafts and hemodialysis access shunts. Smooth muscle cell proliferation and neointimal hyperplasia produce flow-restricting stenoses which decrease long-term graft patency.[1,2] Reported in 10–35% of grafts within the first two years,[3] hemodynamically significant stenoses may be effectively treated by early intervention prior to graft

failure.[4,5] However, surgical revision of these anastomotic lesions is often difficult, and transluminal angioplasty results in an unacceptably high recurrence rate.[6,7] Intravascular stents may provide the endoluminal support necessary to allow successful dilatation of these lesions, increasing secondary graft patency.

We constructed a model to study the acute and chronic effects of intravascular stent deployment across anastomotic sites of end-to-side reversed vein grafts. The model allowed evaluation of stent deployment operational factors, including positioning across the anastomosis and differential stent sizing. Using two different stent designs, we studied endothelialization, incidence of thrombosis, and chronic patency rates at varying time periods after deployment.

Materials and Methods

Bilateral end-to-side reversed vein grafts (n = 6) were constructed in 13 sheep with an average weight of 48 kg. The animals were cared for in accordance with the "Principles of Laboratory Animal Care" and the "Guide for the Care and Use of Laboratory Animals" (NIH Publication 80-23). Each animal was anesthetized with thiopental, intubated, and maintained on halothane anesthesia during the procedure. After surgical exposure of the femoral vessels, the superficial femoral vein was harvested and placed in a cooled bath of lactated ringer's solution, heparin (5000 u/l), and papaverine (30 mg/l). Using regional heparinization of each limb, end-to-side reversed vein grafts were constructed from the proximal superficial femoral artery to the above-knee popliteal artery. The vein grafts were 5–7 cm in length and 3–4 mm in diameter. The native superficial femoral artery was occluded distal to the proximal anastomosis with a suture ligature. The grafts were then allowed to mature for 6 weeks prior to stent deployment.

After 6 weeks, the sheep were returned to the catheterization laboratory, where an arteriogram was performed per the carotid artery to evaluate graft patency and size the anastomotic sites for stent placement. Two different tubular, stainless steel, balloon expandable stents were implanted—15 mm articulated, slotted stents (Palmaz-Schatz) and 20 mm coiled stents (Roubin) (Fig. 28.1). The total deployment cohort (n = 6) included 17 slotted stents and 9 coiled stents. The proximal or distal anastomotic site of each graft was chosen at random for stent placement with the same site used bilaterally in any one animal. Stents were prepared for deployment by crimping (slotted stent) or preloading (coiled stent) on standard balloon angioplasty catheters (3–4 mm). The stent/catheter was then inserted through a guiding catheter to the anastomotic site with stent position across the anastomosis directed by fluoroscopy. Once properly positioned, stents were deployed by expanding the balloon to pressures of

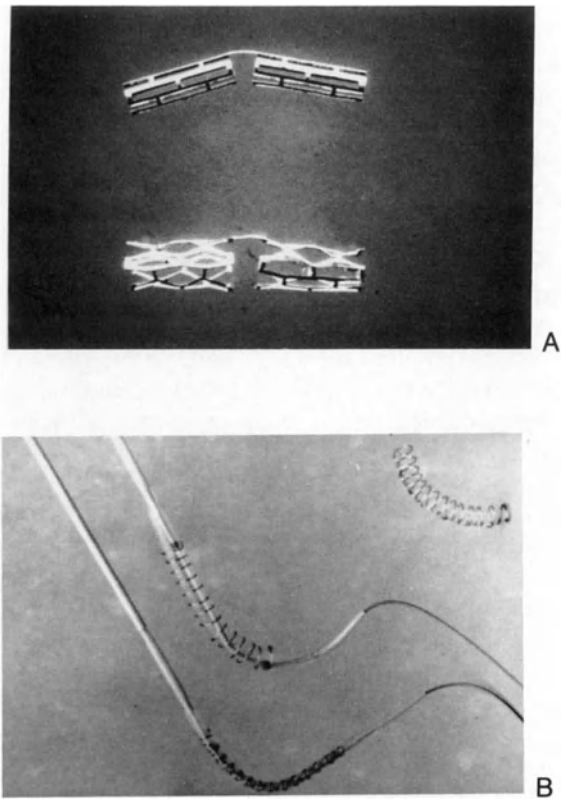


FIGURE 28.1. Balloon expandable, stainless steel stent designs. (**A**) Palmaz-Schatz slotted stent in closed (upper) and expanded (lower) positions. (**B**) Roubin coiled stent showing flexibility while free-standing and loaded on a balloon catheter.

6–8 atmospheres under fluoroscopic visualization. Dextran 40MW was administered intravenously throughout stent implantation, and a completion arteriogram was obtained to assess each stent's position and acute patency.

At predetermined time points (3 weeks, 8 weeks, 6 months), animals were returned to the catheterization laboratory, where an arteriogram was repeated to reassess stent patency, configuration and position. The animals were then sacrificed and each stented vessel segment (artery, anastomosis, vein graft, stent) was harvested using in situ perfusion-fixation techniques. The stents were examined grossly for evidence of patency, thrombosis, stenosis, and endothelialization. The slotted stents were bivalved longitudinally, and the coiled stents cut in cross-section prior to glutaraldehyde fixation. These segments were then examined by scanning electron microscopy.

Results

The flexibility of the slotted and coiled stent designs was adequate to allow equally effective positioning across anastomotic sites and subsequent stent expansion (3.0–4.0 mm) with either stent. Vein graft and native artery size mismatches required occasional balloon reinflation in that portion of the stent deployed in the vein graft to optimize stent vessel wall contact. No acute thrombotic episodes occurred, and all stents were arteriographically patent immediately after stent deployment. Luminal defects were not seen on any postimplantation arteriogram.

At vessel harvest, 88% (15/17) of the slotted stents and 100% of the coiled stents remained patent for a 92% total patency rate (Fig. 28.2). One occluded stent harvested at 3 weeks showed an underlying stenotic area, while the other occlusion occurred in an animal harvested at 6 months

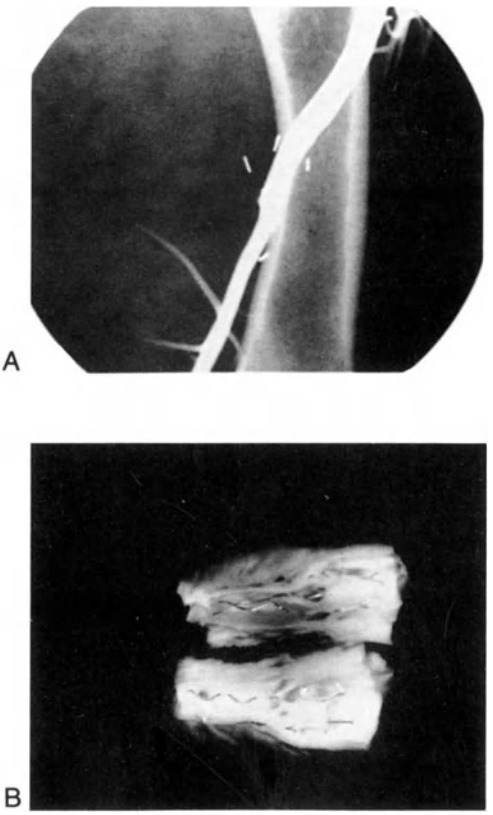


FIGURE 28.2. Arteriogram (**A**) and gross photograph (**B**) of a stent deployed across a vein graft anastomosis demonstrating patency at harvest.

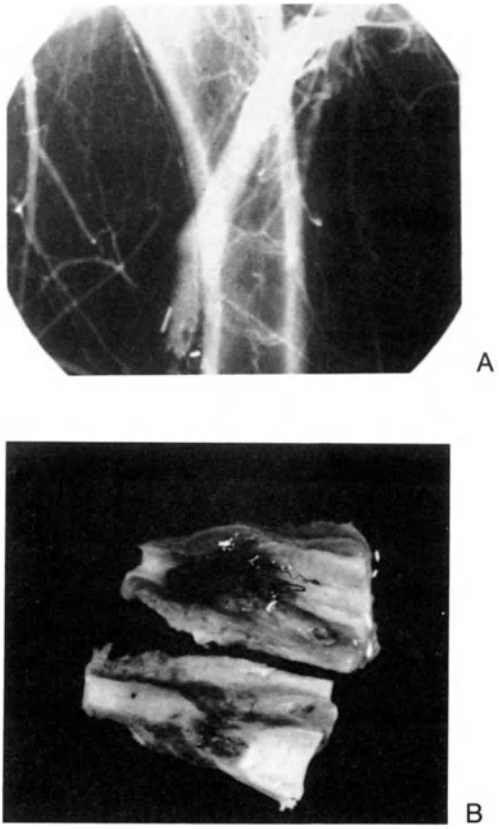


FIGURE 28.3. Arteriogram (**A**) and gross photograph (**B**) of a stent deployed across a vein graft anastomosis demonstrating occlusion at harvest.

without any obvious cause (Fig. 28.3). An arteriographic intraluminal filling defect suggestive of thrombus was noted in 1/17 (6%) of the slotted stents but in no coiled stents. This chronic thrombus was confirmed at harvest (Fig. 28.4) and occurred in a stent that exhibited poor stent-vessel wall contact at initial deployment. Focal arteriographic stenoses were noted at the anastomotic site in 2/17 (12%) of the slotted stents and no coiled stents. Patency was maintained in both of these stenotic stents. Extrinsic compression of the animal's hindlimb or incomplete expansion at the central articulation may have played a role in these noncritical stenoses. Distal migration from the original site of deployment was noted on follow-up arteriography in 3/9 (33%) of the coiled stents and in no slotted stents (Fig. 28.5). All of the migrated stents exhibited evidence of recoil during placement on deployment arteriograms resulting in relative undersizing in the venous portion of the grafts involved.

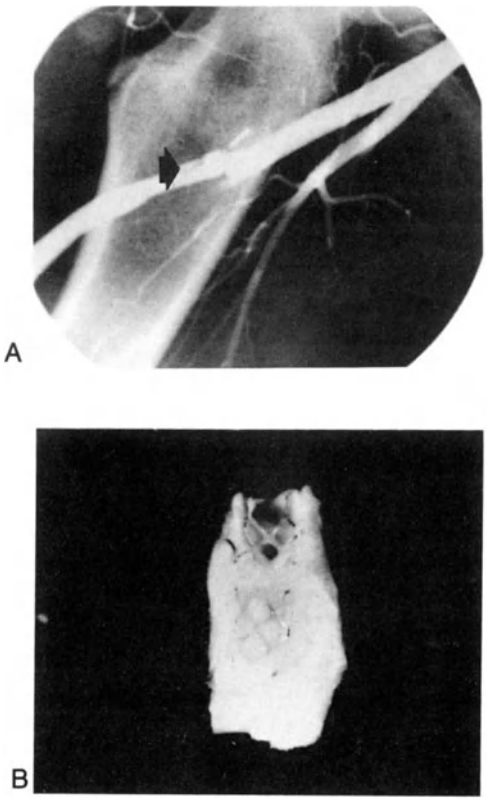


FIGURE 28.4. Scanning electron micrographs (15KV, ×30) showing partial endothelialization of both slotted (**A**) and coiled (**B**) stents at 3-week harvest.

Chronic vessel wall responses were studied in the 3-week, 8-week, and 6-month animals by scanning electron microscopy. Partial endothelialization of the struts of both slotted and coiled stents was evident at 3 weeks (Fig. 28.6). The 8-week animals demonstrated greater endothelial coverage of the slotted stents as compared to the coiled stents from corresponding animals (Fig. 28.7). Both stent designs showed complete endothelial coverage at 6 months (Fig. 28.8). At times, the coiled stents exhibited incomplete stent-vessel wall contact, which may have delayed endothelial cell coverage (Fig. 28.9).

Discussion

Anastomotic stenoses of arterialized vein grafts and hemodialysis access shunts are a frequent cause of late graft failure, often leading to difficult operative revision involving vein patching or an additional bypass

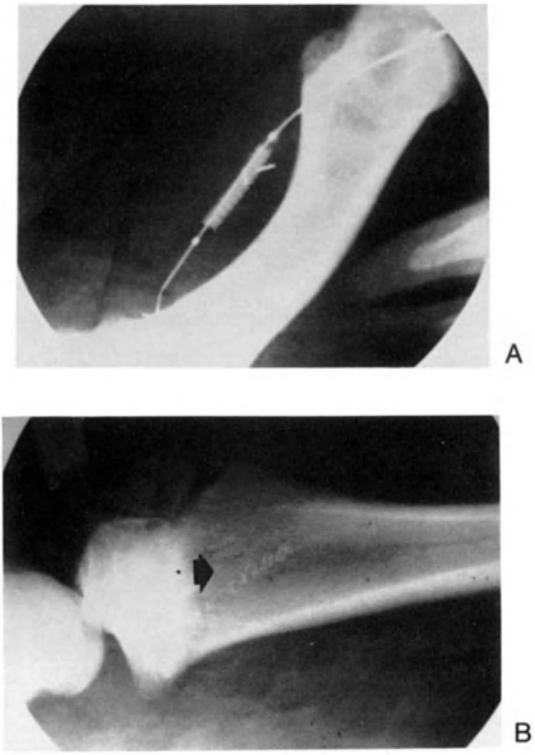


FIGURE 28.5. Scanning electron micrographs (15KV, ×40) at 8-week harvest demonstrating a greater degree of endothelial coverage of a slotted stent (**A**) versus a coiled stent (**B**) from the same animal.

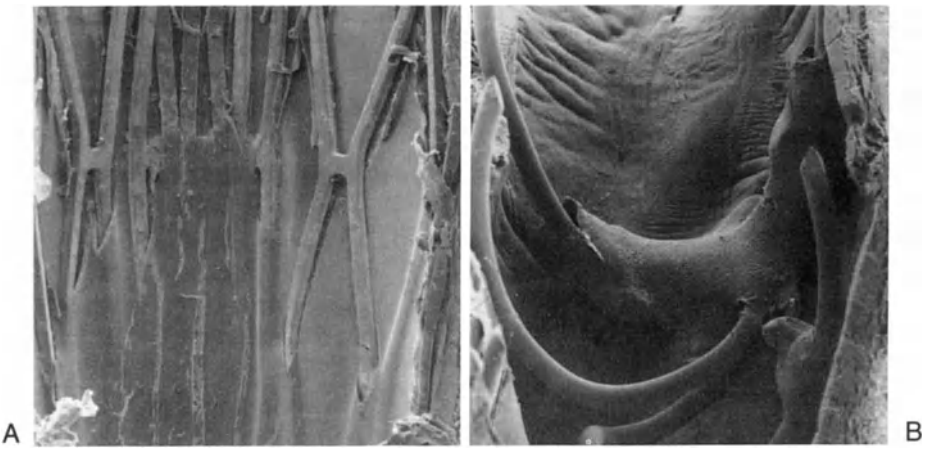


FIGURE 28.6. Scanning electron micrographs (15KV, ×25) showing complete endothelialization of both slotted (**A**) and coiled (**B**) stents at 6-month harvest.

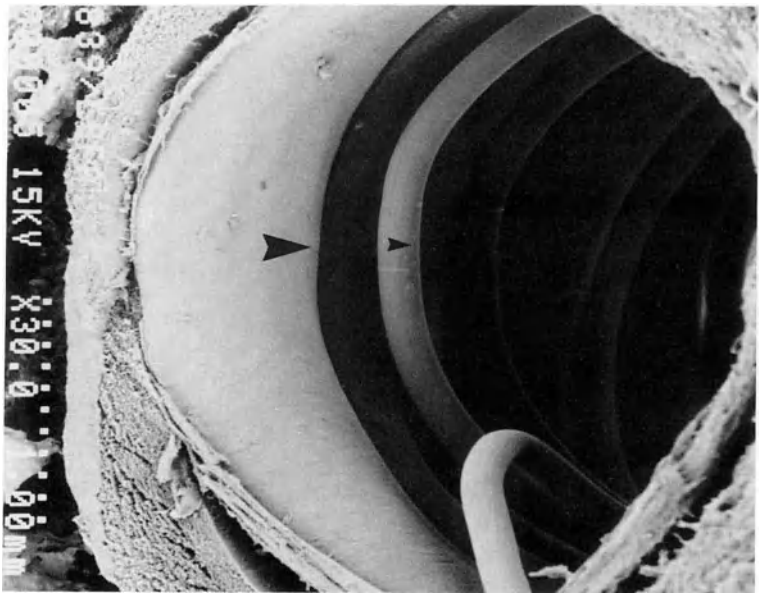


FIGURE 28.7. Scanning electron micrograph (15KV, ×30) demonstrating poor endothelial cell coverage of a coiled stent when the stent struts (small arrow) have incomplete contact with the vessel wall (large arrow).

FIGURE 28.8. Arteriogram (**A**) and gross photograph (**B**) of a slotted stent with mural thrombus (arrow).

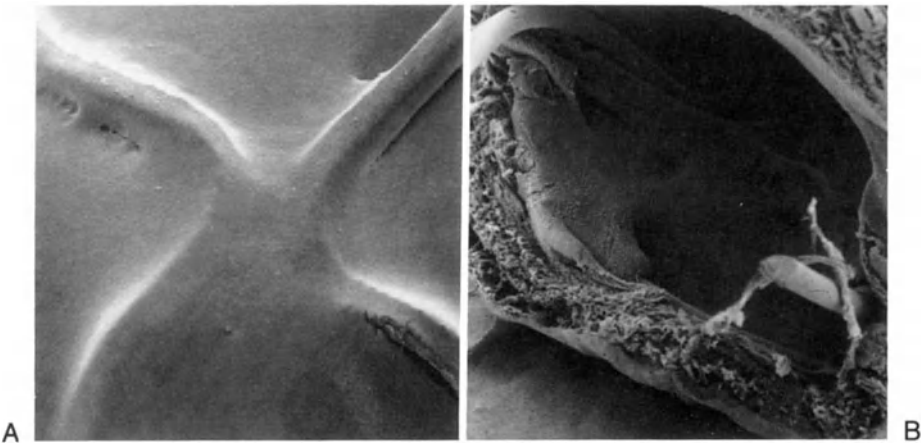


FIGURE 28.9. Arteriograms of a coiled stent during deployment (**A**) and at 8-week harvest (**B**) showing migration to an infrapopliteal position (arrow).

procedure.[3] Attempts at transluminal dilatation of such lesions to increase secondary patency have proved unsatisfactory.[7,8] These lesions frequently recur after angioplasty due to their fibrotic composition and high degree of elastic recoil.[9,10] Longitudinal intimal dissections can also occur leading to platelet initiated thrombosis and stimulation of further smooth muscle cell proliferation with resultant fibromuscular hyperplasia.[11] Intravascular stents may provide the endoluminal support necessary to overcome elastic recoil and seal postdilatation dissections, thereby increasing the acute and chronic patency of these problematic lesions.

Intravascular stents were first implanted in the peripheral arteries of dogs by Dr. Charles Dotter in 1964.[12] Since that time many different stent designs have been developed including self-expanding spirals, thermal memory alloys (Nitinol), and balloon expandable stents. The initial stents were associated with a high incidence of acute thrombosis and unacceptable intimal hyperplasia. These complications were related to stent rigidity and the amount of material exposed to circulating blood elements and requiring neointimal coverage.[8,13] Recent technological developments have produced stents composed of thin wire filaments (0.10–0.15 mm) with a reduced total surface area. These stents provide excellent flexibility while maintaining reliable expansive properties.[14]

Clinical trials using stents to treat recurrent lesions after angioplasty in human iliac and coronary arteries are currently proceeding based on a significant body of data from animal laboratory experience. However, only isolated case reports describe attempts to place stents across the anastomoses of bypass grafts or hemodialysis access shunts.[9,15] Animal experiments involving stent deployment in vein grafts demonstrate rapid endothelialization and good long-term patency when implanted in the body of the graft.[16] The animal work supports the use of dextran during stent

implantation to avoid acute thrombosis. Dextran may function as a buffer between the positively charged metallic stent struts and the electronegatively charged blood elements.[17]

This study was designed to determine the acute and chronic effects of stent deployment across vein graft anastomotic sites as well as examine the operational feasibility of anastomotic implantation. Both slotted and coiled stent designs provided adequate flexibility to maneuver the stent across the anastomotic site with easy expansion in the chosen position. Stents were expanded to a size corresponding to arterial diameter at the anastomotic site. Because of the size discrepancy between arterial diameter and diameter of the vein graft's hood, reexpansion with increased balloon pressures was often necessary in the venous portion of the graft to maximize stent-vein graft wall contact.

Excellent patency rates were obtained with both slotted and coiled stents. Acute thrombosis did not occur and only two stents were occluded at harvest. One of the occluded stents was obviously compressed at the articulation site, as were two stents that remained patent. These stenoses may have occurred due to extrinsic compression as they were located in an area of the hindlimb often grasped by the animal handlers during transport. Extrinsic compression may present a clinical problem for stent deployment in exposed peripheral locations such as the extremities.

Scanning electron microscopy demonstrated endothelial cell coverage of the stents to be partial at the 3-week harvest and complete by 6 months with both stent designs. At the 8-week harvest, the slotted stents endothelialized more rapidly than the coiled stents, which continued to show only partial cellular coverage. The partially covered coiled stents often demonstrated poor contact between stent struts and the intimal surface of the vein graft. Relative stent undersizing in the vein graft due to recoil after balloon deflation was evident on review of the deployment arteriograms of these stents. Similar recoil was noted in arteriograms of the coiled stents that migrated into the distal arterial tree after implantation. Scanning electron microscopy also demonstrated slow or incomplete endothelial coverage of stent struts in the lumen of the vein graft that lacked adequate vessel wall contact.

In summary, we deployed both slotted and coiled endovascular stents across vein graft anastomotic sites in an animal model of reversed vein bypass grafts. The stents demonstrated excellent acute and long-term patency rates (92%) with rare thrombosis, stenosis, or distal migration as evaluated by angiography and gross inspection at harvest. Scanning electron microscopy demonstrated partial endothelialization over both stents by 3 weeks and complete endothelial coverage by 6 months. Slotted stents appeared more susceptible to extrinsic compression, while coiled stents exhibited more frequent migration and delayed endothelialization due to stent recoil at deployment resulting in incomplete stent-vessel wall contact. We conclude that proper sizing of stents to obtain close stent-vessel wall contact played an important role in enhancing the rate of stent endothe-

lialization and prevention of distal migration. Intravascular stents may be deployed across anastomotic sites of end-to-side venous bypass grafts with the expectation of excellent patency rates and favorable long-term host responses.

References

1. Szilagyi DE, Elliot JP, Hageman JH, et al. Biological fate of autogenous vein implants as arterial substitutes. Ann Surg 1973;178:232–244.
2. Imparato AM, Bracco A, Kim GE, Zeff R. Intimal and neointimal fibrous proliferation causing failure of arterial reconstructions. Surgery 1972;72:1007–1014.
3. Grigg MJ, Nicolaides AN, Wolfe JN. Femorodistal vein bypass graft stenoses. Br J Surg 1988;75:737–740.
4. Bandyk DF, Kasbnick HW, Stewart GW, Towne JB. Durability of the in-situ saphenous vein bypass. A comparison of primary and secondary patency. J Vasc Surg 1987;5:256–258.
5. Whittemore AD, Clowes AW, Couch JP. Secondary femoropopliteal reconstruction. Ann Surg 1981;193:35–42.
6. Hunter D, Castaneda WR, Coleman CC, et al. Failing arteriovenous dialysis fistulas: evaluation and treatment. Radiology 1984;152:631–635.
7. Brooks JL, Sigley RD, May KJ, Mack RM. Transluminal angioplasty versus surgical repair for stenosis of hemodialysis grafts: a randomized study. Am J Surg 1987;153:530–531.
8. Zollikofer CL, Largiader I, Bruhlmann WF, Uhlschmid GK, Marty AH. Endovascular stenting of veins and grafts: preliminary clinical experience. Radiology 1988;167:707–712.
9. Glanz S, Gordon GH, Butt KM, Hong J, Adamsons R, Sclafani SA. Stenotic lesions in dialysis access fistulas: treatment by transluminal angioplasty using high pressure balloons. Radiology 1985;156:236.
10. Rousseau H, Joffre F, Raillat, et al. Self expanding endovascular stent in experimental atherosclerosis. Radiology 1989;170:773–777.
11. Dotter CT. Transluminally placed coil spring endarterial tube grafts, long term patency in canine popliteal artery. Invest Radiol 1969;4:329.
12. Cragg A, Lung G, Rysavy JA, et al. Percutaneous arterial grafting. Radiology 1984;150:45–49.
13. Palmaz JC, Sibbitt RR, Reuter SR, Tio FO, Rice WJ. Expandable intraluminal graft: preliminary study. Radiology 1985;156:73–77.
14. Schatz RA, Palmaz JC. New technology in angioplasty: balloon expandable intravascular stents. New Dev Med 1987;2:59–75.
15. Gunther RW, Vorwerk D, Bohndorf K, et al. Venous stenoses in dialysis shunts: treatment with self-expanding metallic stents. Radiology 1989;170:401–405.
16. Prevosti LG, Leon MB, Virmani R, Bartorelli AL, Almagor Y, Teirstein PS. Stent implantation in vein grafts: safety, patency, and host response. Circulation 1988;78(II):407.
17. DePalma VA. Investigation of three surface properties of several metals and their relation to blood compatibility. J Biomed Mat Res Symp 1972;3:37–75.

29
A New Endoscopic Valvulotome for In Situ Bypass Surgery

ARSHAD QUADRI, PARVIZ SADHIGI, and
RICHARD M. BASILE

Summary

A new adjustable endoscopic valvulotome is described in this article. The scientific basis of its development has been discussed in detail by highlighting the historical background and technical problems associated with valvulotomy since the inception of in situ bypass surgery. A concise description of the mechanical properties and physical design of the instrument is disclosed and graphically demonstrated by accompanying illustrations and photographs.

The valvulotome has been used in 12 patients for arterial reconstruction of 14 limbs performed over a period of 1 year and 8 months. The indications for surgery were limb-threatening ischemia (78.5%) and disabling claudication (21.5%). Arterial reconstruction was performed from femoral to distal runoff vessel of either anterior or posterior tibial arteries in 78.5% of the patients. The remaining number of patients had either femoral to above-knee popliteal or below-knee popliteal bypass grafts. Limb salvage rate was 100%; primary patency of these grafts was 85.7%; and cumulative patency rate was 92.8%. The average graft velocity at the distal end of the graft at a 6-month interval was 73.4 cm/sec without significant change compared to earlier postoperative values.

Introduction

With the advent of video angioscopy, availability of smaller caliber angioscopes, and computer image digitalization systems, it has become possible to visualize the intravascular anatomy in greater detail than ever before. One exciting area of the clinical application of this technical advancement is in performing valvulotomy for in situ bypass grafts. Mahigan et al., 1986,[1] and LaMaraglia et al., 1990,[2] have published their experiences with direct vision valvulotomy. Their conclusions have been inspiring. It has thus become more and more apparent that valve lysis is safely achieved

under direct vision of video angioscope while respecting all the time-honored principles of in situ bypass surgery.

All the presently available valvulotomes are either a modification of traditional types or a variation on the same theme, the only exception being that of the Chin-Fogarty System, which uses a different concept of valvulotomy from our valvulotome. Keeping in mind all the lessons learned from past experiences in vascular surgery with in situ bypass grafts, we have developed an endoscopic valvulotome. Our early clinical experience with this system is also described below.

Materials and Methods

The adjustable endoscopic valvulotome is a dual-blade system. Each blade measures less than 1 mm in thickness and has two functional sides: the valve side and the intimal side. The valve side has two cutting edges, with each cutting edge oriented at a right angle to the other. The intimal side is highly polished (Fig. 29.1). The two blades are mounted on separate spring-loaded struts connected to a long stainless steel wire. The blade-strut-wire assembly is passed through a 4F carrier sheath made of medically approved PVC, the proximal end of which carries a blade housing made of surgical steel. This housing is highly polished and rounded, measuring 2.2 mm at its widest part. The blades can thus be telescoped from a cutting to a noncutting position from the sheath's distal end. The spring-loaded struts

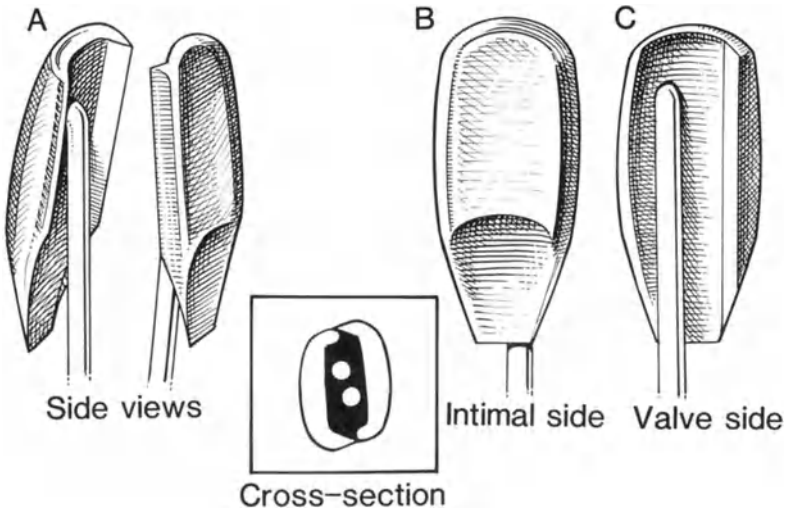


FIGURE 29.1. Showing the intimal and valve side in detail. The horizontal and vertical cutting edges are demonstrated. Inset shows the coaptation of two blades.

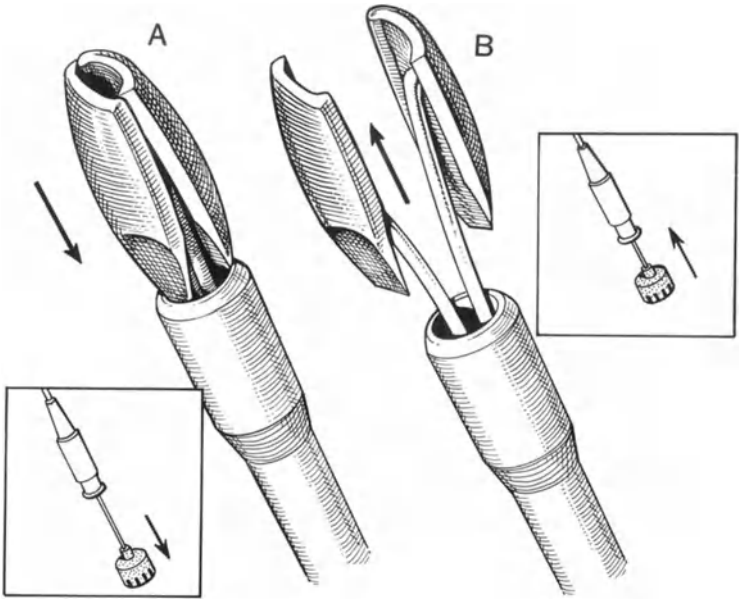


FIGURE 29.2. The valvulotome assembly is shown. The blades are shown in (**A**) cutting and (**B**) noncutting position (note the rounded edges of the distal end). Inset demonstrates the distal end of the valvulotome.

make an arc, so that when the blade is telescoped out of the housing, one can reach a maximum span distance between blades of 5–6 mm and a minimum of 2 mm (Fig. 29.2). Figs. 29.3–29.5 provide different views of the prototype.

The technique adopted in performing valvulotomy is as follows: The angioscope is passed into the proximal end of the saphenous vein and the first set of valves is sighted. The valvulotome is passed into the vein's distal end in a noncutting position so that it passes by the first set of valves and is in the visual field of the scope (Fig. 29.6). The blades are telescoped out of the housing and oriented correctly into a cutting position. The valvulotome is then pulled in a retrograde fashion so that the blades engage with the valve cusps and lyse them in two planes, rendering them incompetent (Fig. 29.7). The valve lysis is achieved by incising the valve cusps as demonstrated in Fig. 29.8.

The valvulotome has been experimentally tried on cadaver veins to test its ease of function and to demonstrate completeness of valvulotomy. Experiments were performed and the data of postvalvulotomy flow rates, velocity of flow, and completeness of valvulotomy were scrutinized. Based on these experiments, we created prototypes of the adjustable endoscopic valvulotome that were to be used clinically. The experimental data is not

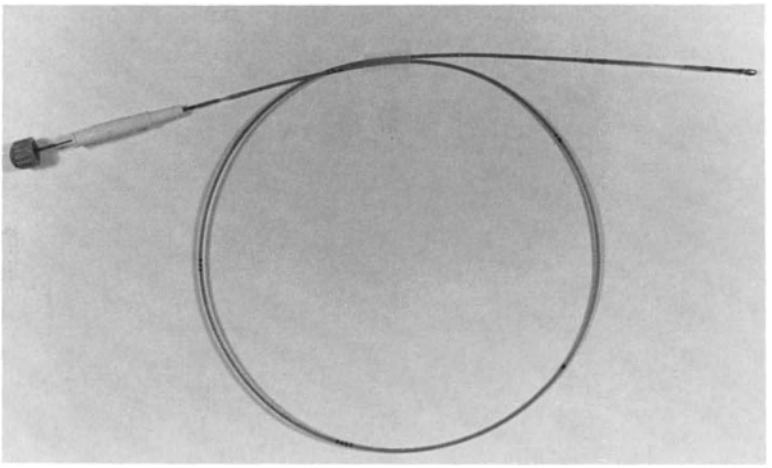


FIGURE 29.3. Valvulotome in closed position.

included in this paper because we feel that the clinical data provided below is sufficient.

Fourteen in situ femoral to distal bypass grafts have been performed over a period of 1 year and 8 months at the Berkshire Medical Center, Pittsfield, MA. In all these cases, our endoscopic adjustable valvulotome was used. It is appropriate to point out here that the valvulotome is designed to be used only once; therefore, each case was performed by a new

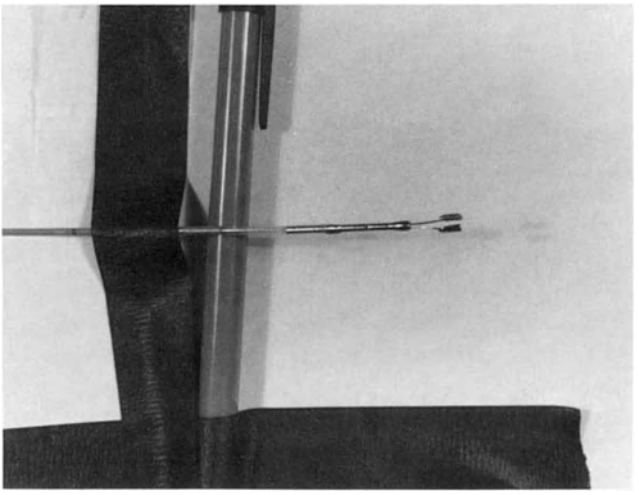


FIGURE 29.4. Valvulotome in open position.

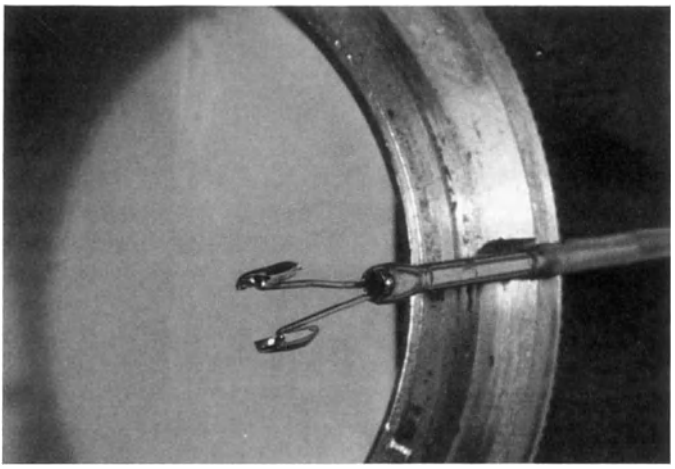


FIGURE 29.5. Close-up of the instruments.

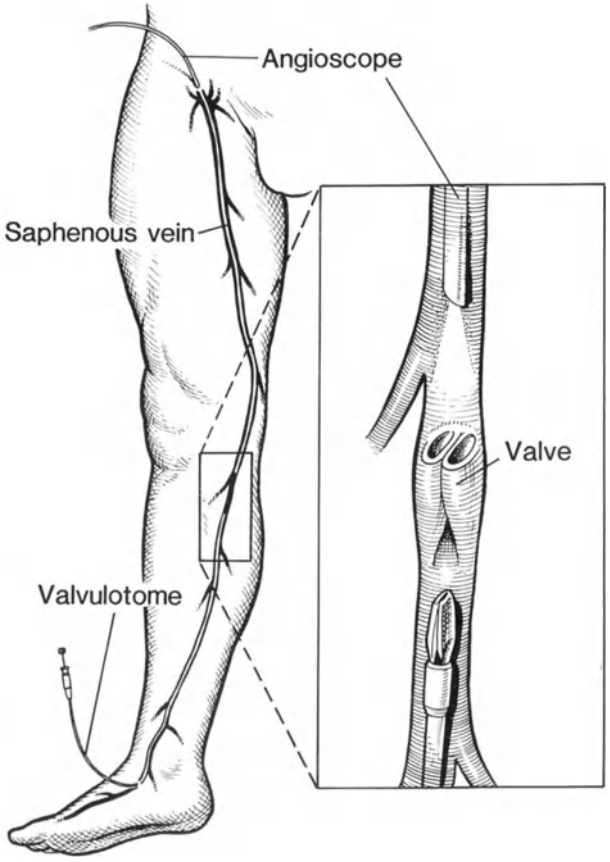


FIGURE 29.6. Artist's impression of the technique (self-explanatory).

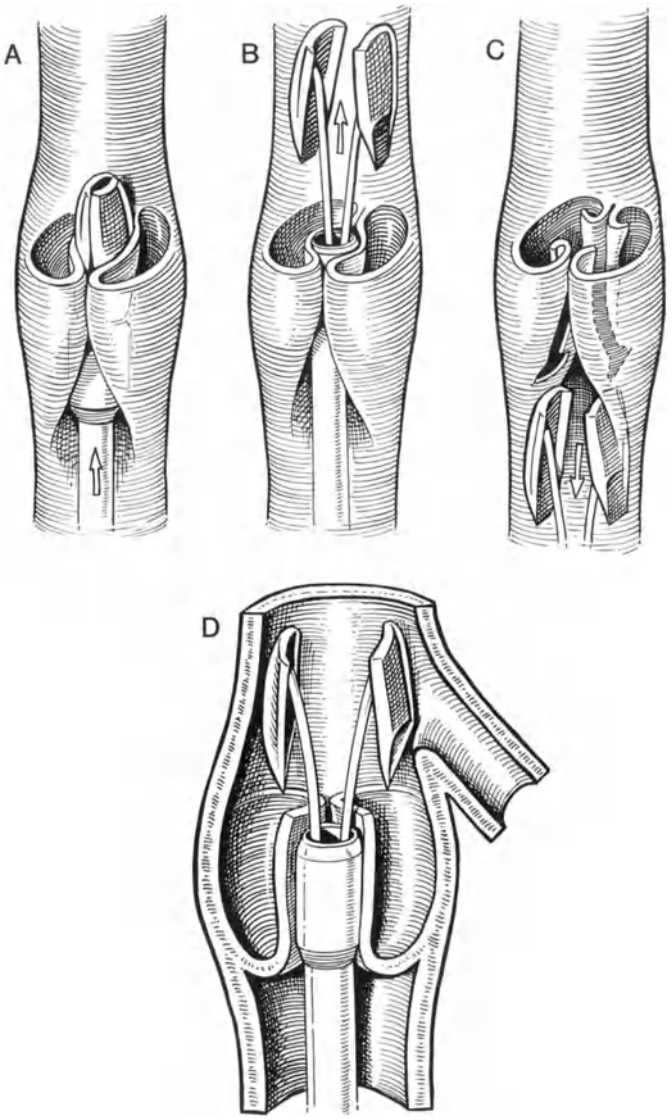


FIGURE 29.7. (**A**) Valvulotome in a noncutting position between valve leaflets; (**B**) valvulotome in a cutting position and oriented parallel to the valve cusps; (**C**) valve incision achieved in two planes; (**D**) side branch safety when valvulotome is at full span.

valvulotome made in our R&D Lab. The patient population consisted of 4 females and 8 males whose ages ranged from 38 to 81 years (average age, 71.5 years). The most common indication for arterial reconstruction in this small series of patients was nonhealing ulcer of the foot (8 patients, 57.1%). Other indications were disabling claudication (3 patients, 21.4%),

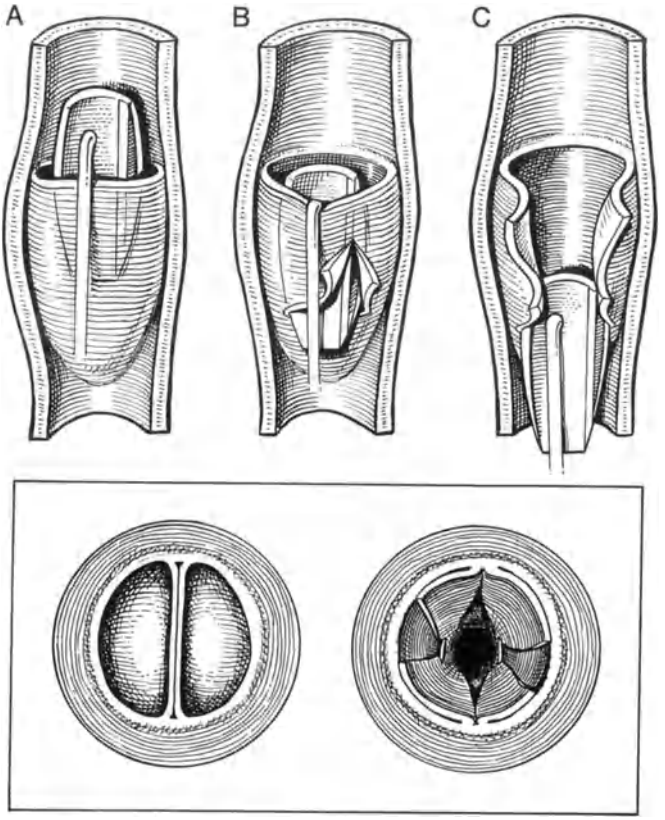


FIGURE 29.8. (**A**, **B**, **C**) Showing details of valve incision. Inset: Cross sectional view pre- and post-valvulotomy.

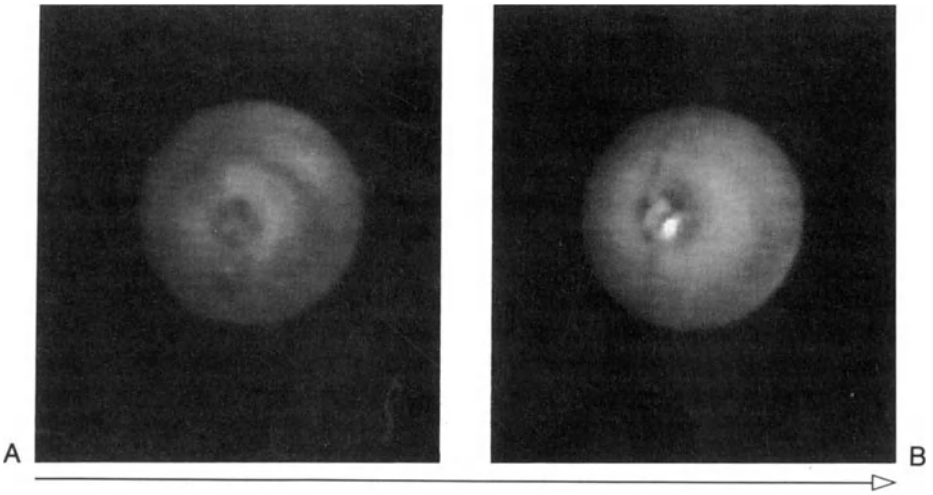


FIGURE 29.9A–G. Showing the sequence of valvulotomy.

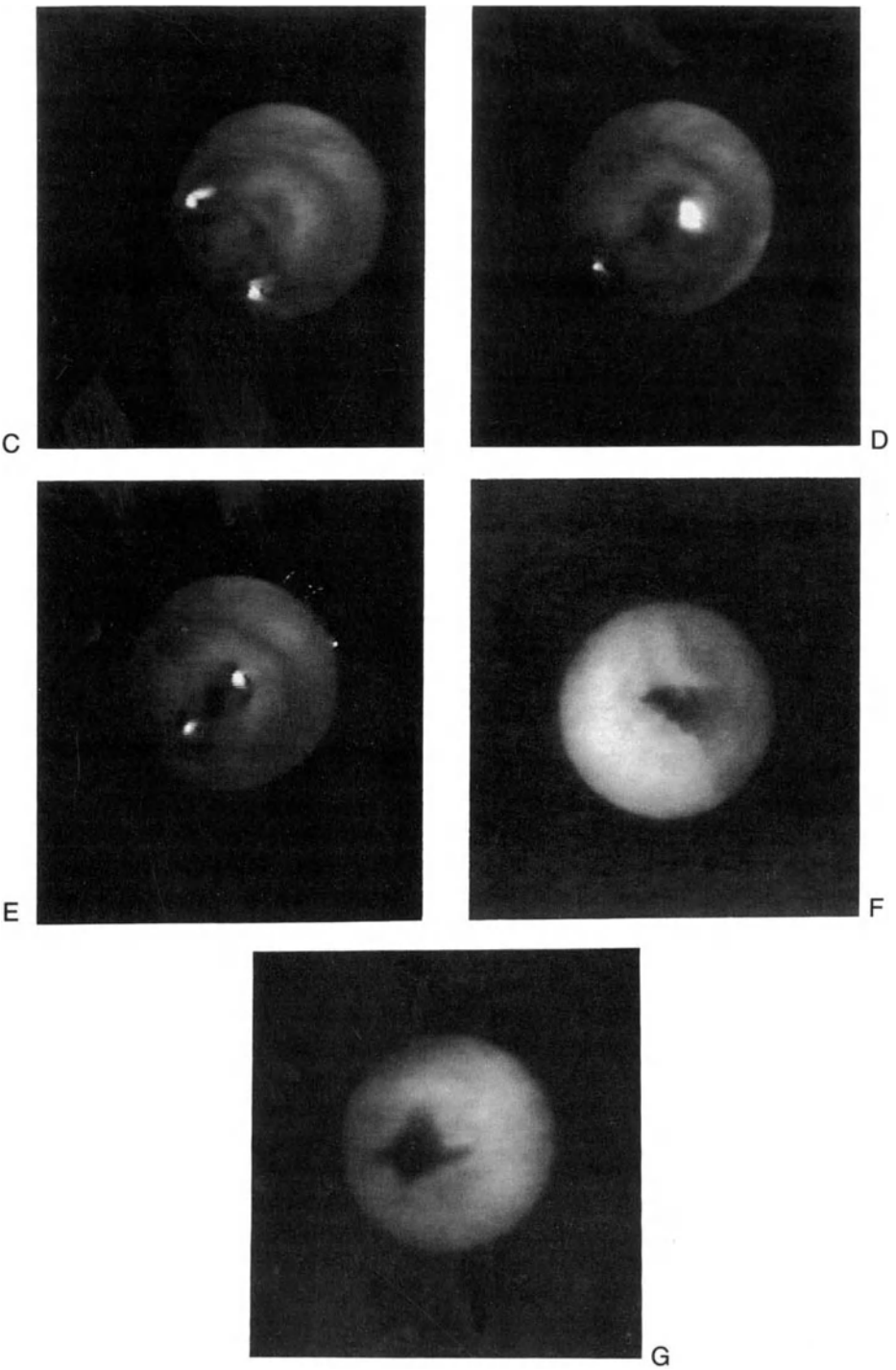


FIGURE 29.9. (**A**) Valve in vision. (**B**) Valvulotome between leaflets (noncutting). (**C**) Valvulotome across leaflets (cutting position). (**D**) Orienting the valvulotome. (**E**) Performing valvulotomy. (**F**) Detail of valvulotomy. Only one leaflet (cusp) is shown in close-up to demonstrate lysis by incision and intact valve vein wall junction. (**G**) Completed valvulotomy.

rest pain (2 patients, 14.3%), and acute and chronic occlusion of the femoral popliteal artery (1 patient 7.2%). Average preoperative ABI was .39 and the range was from .30 to .62. Upon review of the angiographic findings of these patients, it was found that 10 patients (71.4%) had SFA occlusion with significant popliteal and trifurcation disease and single vessel runoff below the knee joint. Two (14.2%) had SFA occlusions with reconstitution of popliteal artery at the knee joint. One (7.2%) patient had isolated SFA occlusion, and the other had acute arterial occlusion of the femoral popliteal system.

The femoral artery and saphenofemoral junction were exposed through a vertical incision in the groin. The common, superficial, and profunda femoris arteries were dissected and controlled. The inflow source was then determined, thus dictating the length of saphenous vein required. The distal anastomotic site was decided by angiographic information and the outflow vessel was accordingly exposed. The distal saphenous vein in the leg was exposed according to anatomic location of the outflow vessel. An angioscope (Baxter 2.3 mm scope with irrigating side channel or Olympus 2.8 mm angioscope) was introduced from the proximal end of the vein and video angioscopy was begun. The saphenous vein was flushed with 60 mg of papaverine in 10 cc normal saline before beginning the valvulotomy. Continous irrigation of the vein was maintained by irrigating with heparinized lactated Ringer Solution at 200–300 mmHG. In some instances, Olympus angiopump was also used to facilitate venous distension. The valvulotome was inserted into the vein through the distal end in closed position and valvulotomy performed as described previously. The sequence of events during valvulotomy are depicted in the photographs. The first set of valves are sighted (Fig. 29.9A) and oriented to perform valvulotomy (Fig. 29.9B–C); valvulotome is pulled back to perform valve lysis (Fig. 29.9D); valvulotomy is thus complete by using retrograde incision technique (Fig. 29.9E–F). Fig. 29.9G shows both leaflets lysed to confirm valvulotomy completeness. These photographs were taken by using Sony Mavigraph from a video movie made in real time while surgery was in progress.

Using the aforementioned technique, femoral to distal anterior or posterior tibial bypass grafts were performed in 11 cases (78.5%). Two patients (14.2%) received femoral to below-knee popliteal bypass, and one (7.2%) underwent a translocated nonreversed femoral to above-knee popliteal bypass.

Results

It is our belief that efficacy of this valvulotome in performing valvulotomy atraumatically is best assessed by following the primary graft patency over the first year postoperatively. Therefore, all grafts underwent noninvasive hemodynamic testing at varying intervals after the first month. Criteria

TABLE 29.1. Pre- and postoperative ABIs.

No.	Pre-op ABI	Surgery	Post-op ABI	Graft velocity cm/sec. (11 days. 6 mos)	Clinical evidence of graft function
1	0.33	R. fem.-ant. tibial BPG.	0.74	40 cm/sec/	Palpable pulse
2	0.4	L. common fem. to distal ant. tibial	0.96	75–100	Palpable pulse
3	0.38	R. SFA. to ant. tibial	—	100	Palpable pulse
4	0.35	L. femoral to bil pop.	0.90	40	Graft stenosis
5	0.38	LFA to AK pop. (translocated)	0.64	40	Palpable pulse
6	0.30	R. CFA. to DP.	1.0	91	Palpable pulse
7	—	L. CFA to ant. AT.	—	60	Palpable pulse
8	0.31	Common to distal posterior tibial	0.98	75	Palpable pulse
9	0.33	CFA to prox. tib.	—	—	Palpable pulse distal to graft
10	0.43	SFA to DP	1.15	81	Palpable pulse
11	—	L SFA to DP	—	—	Palpable at 6 month
12	0.44	L SFA to DP	—	—	Failed
13	0.47	R CFA to distal ant. tibial	0.98	100	Palpable pulse
14	0.60	L CFA to post tibial	1.00	80	Palpable pulse

followed for determining continued graft patency were based on graft flow studies and postoperative ABIs.[3] Since all the grafts were subcutaneous, palpable pulse in the graft and Doppler signals were also used as clinical evidence of graft patency.

Based on the above criteria, the primary patency rate was 85.5% and cumulative patency at the end of $1\frac{1}{2}$ years is 92.8%. At the 4-month interval, one graft failed due to graft entrapment and stenosis, and secondary patency was restored by a wide-patch angioplasty. One graft failed at 3-months due to retained AV fistulas. Average graft velocity at the distal end of the graft was 73.4 cm/sec (range 40 cm/sec to 100 cm/sec). Postoperative flow velocity, ABI, and physical status of the graft are summarized in Table 29.1. Pre- and postoperative ABIs were compared using student t-test for paired data, and we found a p-value of less than .0005. At each follow-up the grafts had palpable pulse and the Doppler signal had a good diastolic component evidencing good forward flow. The limb salvage rate was 100%.

Discussion

In situ bypass grafts have withstood the test of time ever since this technique was first performed by Karl Victor Hall in 1962 in Vienna.[4] Although several excellent clinical studies, Leather et al.[5] and Buchbinder et al.,[6] have demonstrated that in situ bypass grafts have excellent short and long-term patency rates, vascular surgeons have argued that ISBPG has no real advantage over the gold standard, reversed saphenous vein bypass grafts. Porter et al.[7] have demonstrated that RSVG has comparable patency rates to ISBPG, if not better. If the historical development of in situ saphenous vein grafts is traced over the past quarter century, the principle advantage of in situ grafts over conventional reverse vein grafts is in the increased flow occurring in the tapered channel. Both the advantages of this hemodynamic observation in the in situ graft and its disadvantages in the reversed vein grafts are accentuated in the longer bypass grafts as discrepancies in the proximal and distal vein diameter increase. Furthermore, there is new evidence that unusual shear and stress tend to occur at the site of severe vein artery discrepancies, as seen in reversed vein grafts but less so in the in situ grafts. Experience with ISBPG has shown that another important advantage is less chance of vein trauma from overdistension and rotation, inherent in the operation since the vein is not removed from its bed. Besides the above hemodynamic advantage, theoretical advantages of endothelial preservation have been recognized by surgeons. Since the inception of ISBPG, endothelial preservation has been considered crucial because graft failure due to thrombosis has been shown to be associated with platelet aggregation on damaged endothelium.[8] This is especially true in advanced atherosclerotic and diabetic patients. Several studies have demonstrated that removal of vein from its native bed leads to endothelial sloughing[9] within minutes. Studies have also demonstrated that warm as well as cold ischemia of the vein causes damage to both intima and smooth muscles in the vein wall.

Despite the abovementioned advantages of in situ bypass grafts, it has only gained acceptance by vascular surgeons with great reluctance and scepticism. We believe that technical demands of this operation and inadequacy of instrumentation provided to perform such intricate surgery have been major factors in the slow acceptance of in situ technique. Technical problems of in situ bypass have centered around methods of removing valvular obstruction to arterial flow and occlusion of venous branches that otherwise could become AV fistulas. The issues involved in occlusion of side branches are not the subject of this paper. We will, therefore, concentrate on methods of removing valvular obstruction to arterial flow.

The first method of valvular disruption, described and suggested by Rob, was the valve fracture technique. This caused subendothelial damage and thus the technique was not popularized. However, many techniques have

evolved since then. To mention some earlier methods of valvular disruption, Hall and Samual et al. described valvular disruption through the distal venous end; May, Dweese, Rob, Connolly, Stemmer, and Barnar et al. demonstrated valvular disruptions from the proximal venous end; Connolly, Harris and Mills stated that passage of a blunt instrument into a vein produces valve eversion. The valve eversion technique was also not popularized due to similiar fears of endothelial and subendothelial damage. This was an overly traumatic method for achieving valvular incompetence. Besides causing endothelial trauma, the degree of incompetence obtained by all the above methods was uncertain and resulted in tearing valve leaflets at the junction with the vein wall rather than eversion only. Barner et al. were of the opinion that passage of a blunt instrument into the vein from the proximal end resulted in valvular tear. This report was highly critical of the in situ method; the authors also suggested that the subsequent failures of these grafts were especially related to the purposeful damage of the valves, which were the site of thrombus deposition or intimal proliferations or both.

The other method of valvular disruption is that of valve excision as described by Hall. In this method, each valve is sequentially exposed and excised via a transverse veinotomy, which is closed by fine interrupted sutures after valve excision. This was an excellent technique and the only one that performed valvulotomy under direct vision. As a consequence, Hall produced excellent short- and long-term results. However, this technique suffered from several potential disadvantages. Namely, infliction of trauma to vein wall while performing veinotomy (as many as 13), trauma to endothelial surface in excising valve leaflets; potential for technical errors during closure of veinotomies; need for lifting the vein from the bed, clamping above each valve, and the operative tedium involved in excision of each valve. The two most popularly used valvulotomes presently are the Lametré and the Mills-type instruments, which are a big contribution to blind valvulotomy of in situ bypass graft surgery. However, their major criticism has been due to increased incidence of retained valves and damage to veinous endothelium by entering into side branches. Not to overstate the tedium involved in using the Mills or the Lametré valvulotome in long bypasses, Leather and Carmody made an invaluable contribution to in situ bypass surgery by introducing what they called the valve incision technique.[10] In this technique, the valve cusps were engaged by microvascular scissors passed through side branches and exerting no shear on the intima. The valve cusps were clearly divided, rendering them incompetent without damaging the valve vein wall junction.

Learning from the experience with in situ bypass surgery as discussed, we conclude that the following principles have evolved for ideal valvulotomy: 1) valve lysis must be performed under direct vision, 2) endothelial preservation should be of the utmost importance while performing valvulo-

tomy, 3) side branch entry must not be a concern, and 4) valvular incompetence should be achieved by valve incision rather than eversion, fracture, or excision.

The adjustable endoscopic valvulotome performs valve lysis by using valve incision technique; this is assured by the two cutting edges oriented at right angles to each other such that when a downward traction is applied, the valves are incised along the longitudinal axis of the valve cusps. The ability to control the span between the two blades aids in proper engagement with valve cusps and avoids undue intimal contact while performing valvulotomy. An advantage of this valvulotome, which improves endothelial protection, is the fact that all exposed portions of the instrument coming in contact with the intima are highly polished so as to impart minimum shear. Also, a built-in feature in the design of the blade is a gentle curve at the transverse cutting edge. This slope separates the cutting edge from intimal contact and makes the first cut transversely away from the valve ridge. As the valvulotomy is continued, a longitudinal incision in the valve cusp is achieved without disrupting the vein wall junction. Another important feature is the ability to bring the blades to a noncutting position while a subsequent set of valves are sought by the angioscope. This further assures intimal protection. The inherent shape of the blade provides an added protection from entry into the side branch.

Lastly, due to the small size of the valvulotome (2.2 mm in its broadest part), vein utilization rate is maximized. This valvulotome circumvents all inherent technical problems of valvulotomy afforded by other presently available valvulotomes. The instrument is simple to use and minimizes the learning curve required of vascular surgeons wishing to incorporate the in situ bypass technique into their surgical armamentarium.

References

1. Mahigan JT, Olcott C. Video Angioplasty as an alternative to the operative arteriography. Am J Surg 1986;152:139–145.
2. LaMaraglia GM, Cambria RP, Brewster DC, Abbott M. Angioscopy guided semi-closed technique for in situ bypass. J Vasc Surg 1990;12:601–604.
3. Bandyk DF, Kaebnick HW, Stewart GW, Towne JB. Durability of in situ saphenous vein arterial bypass: a comparison of primary and secondary patency. J Vasc Surg 1987;5:256–268.
4. Hall KV. The great saphenous vein used in situ as an arterial shunt after extirpation of the vein valves. Surgery 1962;51:492–495.
5. Leather RP, Shah DM, Chang BB, Kaufman JL. Resurrection of the in situ saphenous vein bypass. Ann Surg 1988;208:435–440.
6. Buchbinder D, Dasch AR, Rollins DL, Dillon BC, Douglas DJ, Schuler JJ, Flanigan D. Results of arterial reconstruction of the foot. Arch Surg 1986;121:673–677.
7. Taylor LM Jr., Edwards JM, Phinney ES, Porter JM. Revised vein bypass to

infra-popliteal arteries, modern results are superior to or equivalent to in situ bypass for patency and for vein utilization. Ann Surg 1987;1:90–97.
8. Bush HL, Graber JN, Jaubowski JA, et al. Favourable Balance of prostacycline and thromboxane A_2 improves early Patency of humans in situ vein grafts. J Vasc Surg 1984;149–159.
9. Cambria RP, Abbott WM. Translocated autogenous vein grafts. In: Rutherford, RB, Ed., *Vascular Surgery*, Philadelphia: WB Saunders Company, 1989, pp. 425–434.
10. Leather RP, Shah DM, Buchbinder D, Annest SJ, Karmody AM. Further experience with the saphenous vein used in situ for arterial bypass. Am J Surg 1981;142:506–510.

30
In Situ Bypass Procedures (How I Do It) and Long-term Results

BENJAMIN B. CHANG, ROBERT P. LEATHER, and DHIRAJ M. SHAH

Introduction

The in situ saphenous vein arterial bypass has gradually been accepted into the surgical community as the procedure of choice for chronic occlusive disease of the lower extremities, especially at the level of the tibial vessels. However, the unique and often intricate problems posed by this procedure make the in situ bypass a potentially frustrating and time-consuming operation, especially to the occasional vascular surgeon. Our 15-year experience with the valve incision method as developed at Albany Medical Center Hospital, now comprised of over 1400 such bypasses, has allowed us to identify and solve many of the common problems seen with this procedure. Herein, we will describe various aspects of the in situ bypass that have proven to be especially crucial for successful completion of this operation.

Patient Selection

The vast majority (>93%) of our patients undergoing this procedure have had limb-threatening ischemia. Although the in situ bypass functions well in the claudicator, few of these patients will be so incapacitated by their occlusive disease as to warrant the risks of surgery.

For patients with extensive foot gangrene, careful evaluation of the extremity is necessary prior to surgery. As successful limb salvage in these cases is often predicated upon a familiarity with unconventional partial foot amputations (Chopart, Syme, Pirogoff, etc.) and a willingness of both the patient and surgeon to devote the time and energy required by these difficult problems, a clear commitment to limb salvage is necessary before actual surgery is performed.

Conversely, patients with indolent foot ulcers—many of whom may appear to have palpable pulses—may well have unrecognized foot ischemia precluding healing. Early noninvasive evaluation with pulse volume

recordings to the transmetatarsal level, followed by aggressive revascularization, can greatly speed ulcer healing in many cases.

Preoperative Angiography

Inadequate or incomplete angiography of the tibial and plantar vessels may well cause the surgeon to abandon limb salvage efforts. Because the in situ bypass has the ability to remain patent even when taken to limited outflow tracts, complete visualization of all infrageniculate vessels is mandatory for proper selection of the optimal outflow site. This requires specific discussion with the angiographer to indicate the surgeon's needs. If necessary, intraoperative prebypass angiography or direct exploration of the tibial vessels may be necessary in demonstrating distal arterial patency, many times predicted by careful Doppler (8 MHz) examination.

Angiographic criteria formerly used as a guide in the prediction of reversed saphenous vein bypass patency have no bearing in the in situ population. Thus, lack of patency of the pedal arch or the presence of only an isolated tibial vessel for runoff does not preclude a patient from anticipated limb salvage.

Preoperative Vein Imaging

Saphenous vein system anatomy is quite variable and complex. In addition, safe use of the intraluminal valve cutter demands a clear definition of the vein anatomy prior to surgery. Because of this, use of invasive or noninvasive saphenous vein imaging techniques are just as important as adequate arterial angiograms for successful completion of the in situ bypass.

In the past, saphenous contrast phlebography was used at our institution with great success. In this procedure a remote branch of the saphenous vein in the foot is cannulated. After the contrast is injected, sufficient heparinized saline is used to flush the contrast from the saphenous vein. By minimizing the possibility for injury to the endothelial layer of the saphenous vein, there were no complications with this method in a series of over 300 patients.

Application of B-mode duplex ultrasound to venous imaging has led us to prefer this method to phlebography in most cases. In addition to being noninvasive, this study may be performed quickly (<30 minutes by experienced personnel) and can be used to generate a three-dimensional map of the saphenous system that can be traced onto the patient's skin with a surgical marker. By indicating the presence of major fistulae and parallel venous systems, such a map is a great aid in positioning of incisions and selection of the most satisfactory portion of the vein for use as an arterial

bypass. In less than 5% of cases, the venous system is so complex that phlebography is still necessary and useful for the surgeon.

With an experience of over 1000 limbs using this modality, several words of caution are appropriate. A familiarity with the anatomic variants of the saphenous system, especially as it affects the operation, is mandatory for the vascular technician. Therefore, technician education through review of pertinent articles and initial comparison with phlebography, coupled with constant feedback of the actual operative anatomy, is necessary in order to gain reliable information from this examination.

The presence of branches and double systems is reliably detected by either imaging modality. Although the vein diameter can be roughly estimated preoperatively, the actual size of the distended vein can only be appreciated through direct visualization. Therefore, a vein that is reported as being too small by either technique still warrants exploration before any decision is made as to adequacy of size. When exposed, if the vein compares favorably with a #8 French catheter, it is more than adequate and will measure >3 mm OD under arterial pressure.

Information gained by saphenous vein imaging is useful in minimizing the dissection necessary for in situ bypass. Selection of the proper venous system, especially by avoiding narrow segments and complicated, interconnecting parallel veins, allows for use of the intraluminal cutter; without this knowledge, the likelihood of endothelial damage and bypass failure is greatly increased.

Selection of Inflow

Selection of the proper inflow vessel depends on both venous and arterial anatomy. Although in situ bypass patency is identical if any of the three usual inflow arteries is used, we prefer using the superficial and profunda femoris arteries when possible. This allows for a tension-free anastomosis. In addition, this allows the surgeon to excise the proximal several millimeters of the saphenous vein which during preparatory manipulation, invariably suffers injury to the endothelium. However, if these arteries are unsuitable for use as inflow vessels, the saphenous vein can always be made to reach the common femoral artery, albeit with considerable mobilization and some tension on occasion. Endarterectomy of the proximal superficial femoral artery, although tempting, has proven, in our hands, to have an increased failure rate and is to be avoided if possible.

Selection of Outflow

Of the three tibial vessels, the posterior tibial is most conducive to in situ bypass as it is the most easily exposed and requires a very short distal

segment of mobilized saphenous vein for anastomosis. The anterior tibial artery requires the longest length of mobilized distal vein, whether tunneled through the interosseous membrane or brought anteriorly over the tibia (preferred), and is therefore the least favorable. The peroneal artery falls between these two choices; however, as it is the most likely tibial vessel to remain patent, it is therefore the most likely vessel to be selected for tibial bypass.

Another consideration for outflow selection is the concept of bypassing all occlusions when possible. Thus, unless inescapably limited by a lack of adequate vein, a bypass to a tibial vessel is usually preferable to an isolated popliteal artery. Direct perfusion of the foot, especially in the face of tissue necrosis, is the driving tenet of outflow selection, even if this necessitates the use of ankle and foot arteries.

Open vs. Closed Valve Incision

There are two major methods of valve incision. The "open" technique involves direct exposure of the vein and use of the modified Mills valvulotome to render valves incompetent. The advantages of this technique are that this is the least traumatic method of any kind for valve incision and is the only safe method in small (<3 mm OD) veins. In addition, the surgeon can visualize the vein during valve incision. Unfortunately, use of this technique in the thigh often results in large, deep incisions. A number of wound problems will result. In addition, this method is relatively time-consuming.

The "closed" technique, utilizing the intraluminal valve cutter, while not only faster, obviates the need for long incisions along the entire vein. However, this technique is "blind." In order to safely utilize this technique, preoperative vein imaging is mandatory to aid in selection of the proper vein and to avoid narrow areas and closed loops better treated by the open technique. Furthermore, use of the valve cutter should be strictly limited to the thigh portion of the saphenous vein with the cutter being introduced and retrieved through the vein's proximal divided end. In addition, the cutter must be used in a dilated vein to avoid a lethal circumferential injury to the intima and should be oriented such that its blades are perpendicular to the plane of the skin in order to engage the valve leaflets. In spite of these cautions, the closed technique can safely be used in the thigh portion of the saphenous vein in 95% of cases.

Venospasm

Spasm of the exposed vein is the most likely source for conduit injury. While spasm in and of itself can cause damage to the endothelial mono-

layer, more importantly it renders intraluminal instrumentation of any kind unsafe. Avoiding and treating venospasm is of paramount importance for the surgeon.

Prior to skin incision, perivenous tissue should be infused with a dilute solution of papaverine (0.02 mg/ml in saline). In addition, this powerful vasodilator can be applied topically to all exposed segments of vein.

Once venospasm has occurred, the vein must be hydrostatically dilated. This may be performed safely using a mixture of 500 cc of Dextran 70, 1000 units of Heparin and 120 mg of papaverine brought to a pressure of <200 mmHg introduced into the vein via a branch or the transected end of the vein. After occluding the spasmed segment proximally, the surgeon must wait for the segment to dilate. If there is continued flow with both ends of the segment occluded, a fistula is present that should be ligated. Dilatation may take as much as 10 minutes. However, use of pressures of greater than 200 mmHg will cause endothelial injury. Manual hydrostatic dilatation may unwittingly produce extremely high pressures and severe endothelial disruption.

Use of the Modified Mills Valvulotome

The valvulotome is the least traumatic method of valve incision available. Again, it should only be used in a dilated vein in order to minimize intimal injury. The most common problem encountered is laceration of a vein branch orifice. This can be prevented by visualizing each valve incised and, in particular, the posterior leaflet as there is often a branch immediately proximal to the insertion ridge. If the vein is lacerated in this manner, the defect should be patched, either with a separate piece of vein or with an in-continuity patch using the lacerated branch itself.

The most reliable test for absence of a flow-limiting stenosis in the bypass conduit before construction of the distal anastomosis is to observe free flow through the distal divided end of the arterialized in situ vein. Strong, persistent pulsatile flow (over 5–10 seconds) is absolute evidence that no proximal hemodynamically significant stenotic lesion exists.

If, however, there is initial strong flow (1–2 pulses) followed by diminished flow, palpate along the conduit until a pulse is palpable. This is the site of obstruction or stenosis. If no valve leaflet is encountered by reintroduction of the valvulotome, then spasm or a platelet plug is the cause. Before the distal anastomosis is constructed, deliberate closure of any incompletely lysed valves can be precipitated by rolling a folded towel or lap pad along the in situ conduit from top to bottom while there is high flow either to a perforating vein or the distal divided end.

Arterial Control

Tibial arteries can be difficult to control atraumatically. Usually, use of small neurosurgical aneurysm clips (Yasargil, Heifetz) suffices. However, calcified arteries may preclude their effectiveness but should not be a reason to abandon the operation. There are several alternative methods available to aid the surgeon, all of which are useful at one time or another.

An orthopedic tourniquet as suggested by Bernhard and Towne is safe, reliable, and effective, albeit somewhat cumbersome. Crushing the artery with a large clamp as suggested by Ascer may work, but we have been reluctant to use this technique. Small Fogarty catheters or Flo-Resters can give intraluminal control but also are difficult to sew around and can cause damage to the arterial intima. If all these maneuvers fail, then sewing while the blood is sucked from the field is an infallible, if messy, alternative.

Anastomotic Technique

The proximal anastomosis is usually performed with 7-0 continuous prolene, while the distal is sutured with 8-0 prolene. One to two centimeters of vein end should always be amputated and discarded as the intima is usually injured in the course of instrumentation. Thus, somewhat more vein than is necessary should always be prepared initially. Using the injured segment of vein can lead to early bypass failure or formation of stenoses leading to late bypass failure.

The "parachute" technique for anastomosis is the most accurate method of suture placement in the anastomosis heel. There is no effort made to construct a large anastomosis; the arteriotomy is usually 1.5–2.5 times vein diameter. A great deal of effort is spent to avoid handling or touching the intima with forceps. Use of loupe magnification (2.5–3.5×) and headlight illumination should be regarded as mandatory.

Fistula Localization

Iatrogenic arteriovenous fistulas produced by arterialization of the vein in situ are often a source of great concern for the surgeon. Identification and ligation of these fistulas is quite simple with the use of an intraoperative Doppler.

After the proximal anastomosis is constructed, the Doppler signal in the proximal segment of the saphenous vein is noted. A high flow signal denotes the presence of significant fistulas. If the vein is compressed proximal to all fistulas, there will be a cessation of flow as detected by the Doppler. By moving the compressing finger gradually more distally, eventually the Doppler will suddenly detect flow. The underlying fistula can then be dis-

sected and ligated. Only those branches with high flow need be ligated. There is no need to ligate every branch of the saphenous vein; in fact, doing so will lengthen the operative procedure and require needlessly extensive dissection. A completion angiogram will identify residual major fistulas. Usually fistulas from the bypass to the deep system (perforators) are ligated while cutaneous fistulas are ignored. Persistence of a small amount of fistula flow at the completion of the procedure is of no great concern, as the natural history of these fistulas is usually one of progressive asymptomatic occlusion.

Completion Angiography

On-table completion angiograms are useful in identifying certain problems. Small, irregular intraluminal defects may be due to platelet aggregates in the area of intimal damage; these areas should be examined with the Doppler. If there is a corresponding flow acceleration (stenosis), this area should be opened and patched. Areas of persistent spasm should be hydrostatically dilated. Retained valve leaflets often will not be visualized by angiography but are detectable by Doppler as areas of stenosis.

Follow-up

An adequate follow-up program is just as important as performing a proper operation. Pulse volume recordings are done on the first or second postoperative day. In addition, the distal end of the bypass is examined by Doppler each day postoperatively. Any bypass occlusion is immediately returned to the operating room, as the occlusion is initially segmental and needs only a short jump graft or patching. Although blood will remain liquid within the uninjured part of the in situ bypass, often for many days, any delay of revision may lead to loss of the entire bypass.

After discharge the patients are seen within two weeks and every three months thereafter for the first year; subsequent visits every six months are recommended. At each visit, segmental pressures, pulse volume recordings and Doppler examination are performed. In addition, if possible, examination with duplex ultrasound is done as this has proven to be the most sensitive test for identifying early stenoses or other occult problems.

Conclusion

Successful utilization of the in situ technique requires strict adherence to the principles of avoiding intimal injury because preservation of the intact endothelial monolayer is probably the key to the superior performance of

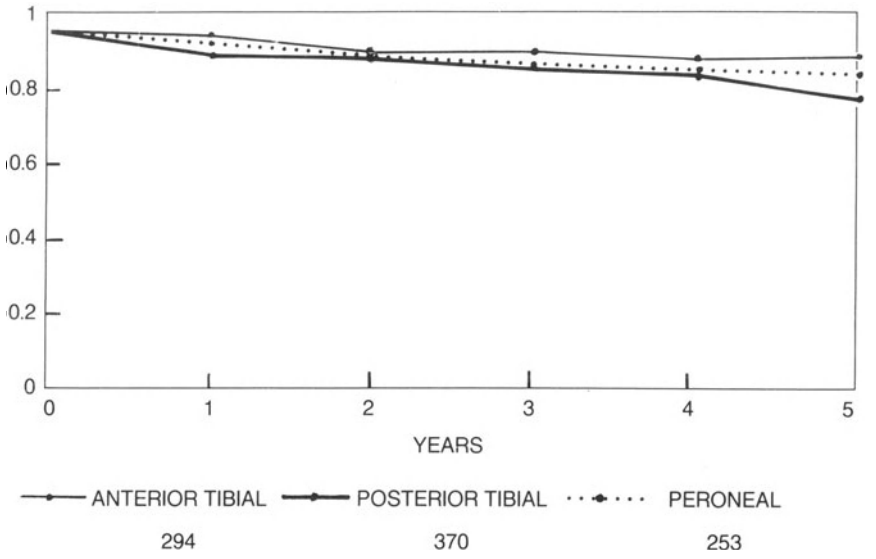


FIGURE 30.1. In situ saphenous vein bypass—effect of distal vessel perfused.

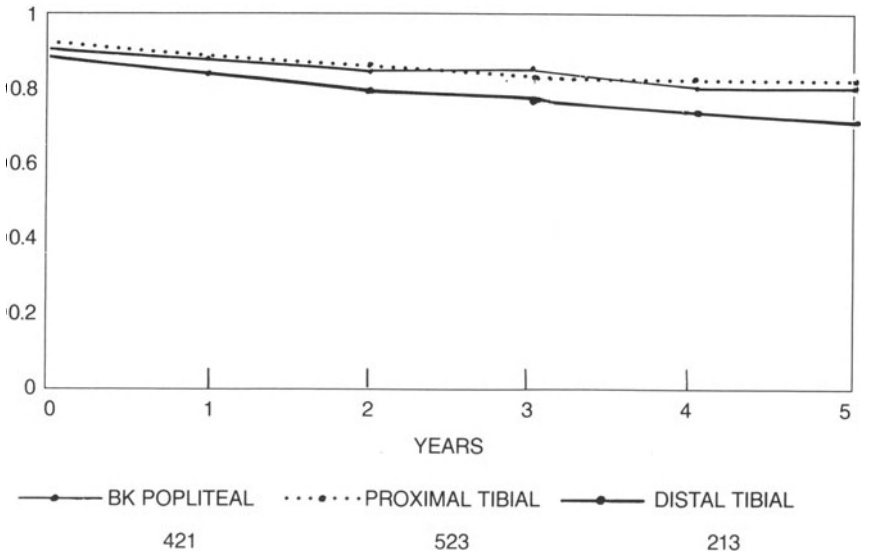


FIGURE 30.2. In situ saphenous vein bypass: levels of distal anastomosis.

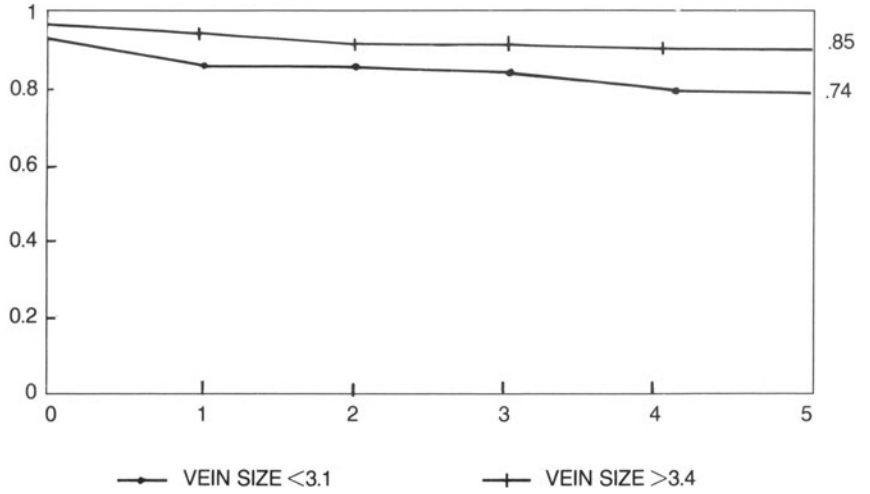


FIGURE 30.3. In situ saphenous vein bypass: effect of vein size.

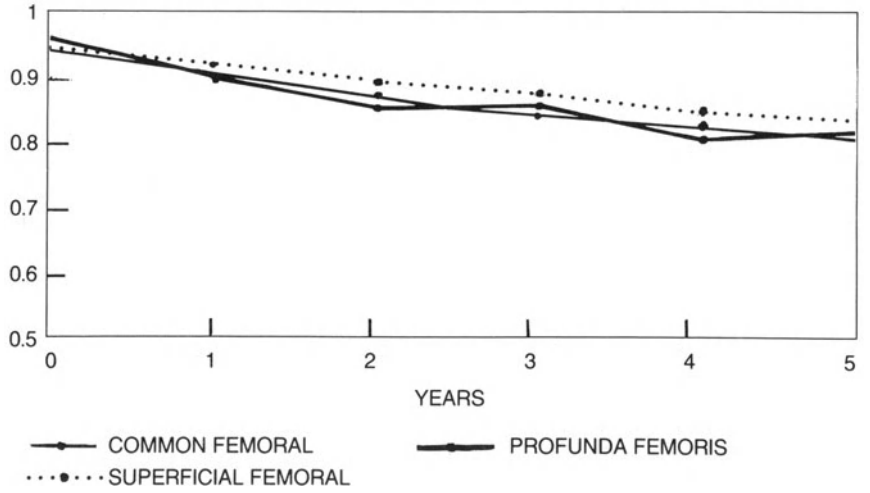


FIGURE 30.4. In situ saphenous vein bypass: effect of inflow source.

TABLE 30.1. Life table analysis of 1431 in situ bypasses.

Interval (months)	Grafts at risk	Occlusions	Interval patency	Cumulative patency
0–1	1431	56	0.959	0.959
2–12	1215	50	0.950	0.911
13–24	745	24	0.962	0.876
25–36	501	9	0.979	0.858
37–48	333	11	0.961	0.824
49–60	224	4	0.978	0.807

TABLE 30.2. Life table analysis of 967 tibial in situ bypasses.

Interval (months)	Grafts at risk	Occlusions	Interval patency	Cumulative patency
0–1	967	36	0.960	0.960
2–12	813	36	0.946	0.908
13–24	481	15	0.962	0.874
25–36	302	8	0.969	0.847
37–48	202	5	0.971	0.822
49–60	135	3	0.973	0.800

the in situ bypass. Atraumatic valve incision, use of fine anastomotic technique, and recognition and correction of problems will ensure a high success rate for the surgeon.

References

1. Leather RP, Powers SR, Karmody AM. A reappraisal of the in situ saphenous vein arterial bypass. Surgery 1979;86:453–60.
2. Leather RP, Shah DM. Experiences with in situ lower extremity saphenous vein bypass procedures. In: Nyhus LM, ed., *Surgery Annual 1988*. Norwalk, CT: Appleton-Century-Crofts, Inc., 1988:259–71.
3. LoGerfo FW, Quist WC, Crawshaw HW. An improved technique for endothelial morphology in vein grafts. Surgery 1981;90:1015–24.
4. Shah DM, Chang BB, Leopold PW, et al. The anatomy of the greater saphenous venous system. J Vasc Surg 1986;3:273–83.
5. Leopold PW, Shandall AA, Corson JD, et al. Initial experience comparing B-mode imaging and venography of the saphenous vein before in situ bypass. Am J Surg 1986;152:206–10.
6. Leather RP, Shah DM. Reoperation after infrapopliteal in situ saphenous vein bypass. In: Trout HH, ed., *Reoperative Vascular Surgery*. New York: Marcel Dekker, Inc., 1988:193–209.
7. Leather RP, Shah DM, Chang BB, Kaufman JL. Resurrection of the in situ saphenous vein bypass: 1000 cases later. Ann Surg 1988;208:435–42.
8. Leather RP, Shah DM. In situ saphenous vein arterial bypass. In: Rutherford RB, ed., *Vascular Surgery*. Philadelphia: WB Saunders, 1989:414–24.

31
Modern Concepts of Vascular and Microvascular Disease in Diabetes Mellitus: Implications for Limb Salvage

FRANK W. LOGERFO and FRANK B. POMPOSELLI, JR.

Foot problems are one of the most common reasons for hospitalization of patients with diabetes mellitus. The incidence of major amputation is approximately 1% per year for the diabetic population over the age of 65.[1] Ischemia, often in combination with neuropathy and infection, is a major factor in the pathogenesis of diabetic foot problems. The purpose of this article is to describe the mechanisms leading to ischemia and to provide a plan for optimum clinical management.

One of the greatest impediments to appropriate care of foot problems in patients with diabetes is the misconception that they have an untreatable occlusive lesion in the microcirculation.[2] This idea originated over 30 years ago from a retrospective histologic study that demonstrated the presence of PAS-positive material in the arterioles of lower extremity amputation specimens from patients with diabetes mellitus. This lesion was described as an "arteriolosclerosis" of diabetes. However, subsequent prospective histologic studies of amputation specimens failed to demonstrate an arteriolar occlusive lesion characteristic of patients with diabetes.[3] In addition, a prospective study of amputation specimens employing a sensitive arterial casting technique[4] confirmed the absence of any small artery or arteriolar occlusive lesion associated with diabetes mellitus. Physiologic studies of patients undergoing femoropopliteal bypass demonstrated no fixed resistance characteristic of arteriolar arteriolosclerosis,[5] and a study employing foot plethysmography concluded there was no evidence of a diabetes-specific small vessel disease.[6] In view of this evidence, accumulating over several years and originating from different experimental approaches, it appears safe to conclude that there is no occlusive lesion in the microcirculation of patients with diabetes mellitus. This is critically important in clinical management, since if such a lesion existed, it would preclude any effectiveness for arterial reconstruction and would connote a poor prognosis for arterial graft patency.

While there is no occlusive lesion in the microcirculation, there does appear to be some evidence of microcirculatory dysfunction. The muscle capillary basement membrane is thickened in patients with diabetes, and

while this is not an occlusive lesion, it may influence the exchange of nutrient materials and/or white blood cells between the capillary and the interstitial fluid. Nonenzymatic glycosylation of basement membrane components replaces sulphur groups and reduces the charge on the basement membrane, which may explain the increased capillary leak of highly charged molecules such as albumin[7] often noted in patients with diabetes mellitus. However, there does not appear to be any impairment of oxygen diffusion since diabetic patients presenting with foot ulcers actually have a higher transcutaneous pO_2 than nondiabetic patients with foot ulcers.[8] In addition, anatomy of the capillaries appears to be different, with more tufted or glomerular-like capillaries noted on video capillary densitometry.[9] This, combined with the autosympathectomy that occurs in diabetes, may interfere with normal distribution of blood flow, particularly in response to pressure points. Thus, it is logical to conclude that there may be functional abnormalities, not occlusive, in the microcirculation that contribute to development of ulceration in the diabetic foot, although a precise link has not been demonstrated.

The clearest differences between patients with and without diabetes mellitus occur in the realm of peripheral vascular occlusive disease (atherosclerosis). For example, prevalence of occlusive disease as demonstrated by noninvasive tests was five times higher among patients with noninsulin dependent diabetes mellitus as compared with normals.[10] Furthermore, 87% of patients with diabetes mellitus and occlusive disease had evidence of progression within a two-year period. The anatomic pattern of atherosclerotic occlusive lesion is different in patients with diabetes mellitus, and this difference has important clinical implications. With diabetes there is a propensity for the occlusive lesion to involve the infrageniculate arteries, often referred to as "tibial vessel disease." Surprisingly, however, this propensity appears to end at the ankle, so that there is actually less occlusive disease involving the foot arteries in patients with diabetes mellitus. This observation was originally made by Strandness[3] on histologic studies and confirmed by Conrad[4] by arterial casting, and more recently demonstrated by Menzoian et al.[11] by arteriography. Once again, it would be inappropriate to label this atherosclerotic occlusive process as a "small vessel" pattern since its influence appears to be confined to the proximal and midtibial vessels, with sparing of the smallest arteries, the foot arteries. This observation is extremely important because it allows for successful arterial reconstruction to the distal tibial peroneal or pedal arteries in the ischemic diabetic foot.

In general, the absence of palpable foot pulses, in the presence of tissue loss, is sufficient confirmation that ischemia is a contributing factor. The use of Doppler ankle pressures is an effective assessment of ischemia in over 90% of patients with diabetes mellitus. However, about 10% of patients will have calcification of the tibial vessels to such an extent that they cannot be occluded by a blood pressure cuff, the so-called "noncompres-

sible" vessels. In such instances, compression of the toe vessels is almost always possible[12] or, alternatively, pulse volume recordings may be helpful. Once it has been determined that ischemia is a contributing factor to tissue loss, prompt arteriography should be performed, followed by arterial reconstruction.

Accurate and complete arteriography is the cornerstone of success in arterial reconstruction, and in the diabetic patient this requires an understanding of the anatomic pattern of occlusive disease. Thus, it is essential that arteriography include the foot vessels, even when the more proximal tibial and peroneal arteries are completely occluded. A common mistake is to note the infrageniculate artery occlusion and make the assumption that "small vessel disease" is present and proceed no further with the arteriogram. Fear of contrast-induced renal failure is often invoked as a limitation on arteriography. However, this is not true in patients without preexisting renal failure, seldom occurs even in patients with preexisting renal failure, and rarely requires dialysis for management.[13] Selective femoral injection, combined with use of digital subtraction arteriography, when necessary, will lead to visualization of the foot vessels in virtually all patients, even those with severe tibial occlusive disease. The use of nonionic contrast material appears to offer no significant clinical advantage as far as the development of renal failure is concerned, and the greatly increased cost of this agent does not seem to be justified for that purpose.[14]

Many diabetics in need of revascularization present with severe, limb-threatening infection that must be treated aggressively with appropriate antibiotics, local debridement and adequate drainage as a first priority. Once infection is under control, arteriography and revascularization can and should be performed. By prompt initiation of these steps, many threatened limbs can be salvaged with only minor toe amputations. Neuropathy is an important additional mechanism in the pathogenesis of diabetic foot problems.[15] Although neuropathy may produce an insensitive foot, may alter or mask the classic manifestations of claudication and ischemic rest pain, the presence of neuropathy should not preclude diagnosis and treatment of ischemia with threatened tissue loss.

In contrast to some earlier reports, more recent experiences with lower extremity arterial reconstruction demonstrate better results in patients with diabetes mellitus than in nondiabetic patients.[16] We have found this to be true even though diabetics more frequently require distal tibial, peroneal or pedal reconstruction as compared with standard femoropopliteal reconstruction in nondiabetics.[17,18,19] Overall vein graft patency at four years was 88% in diabetics as compared with 82% in nondiabetics. While minor amputations were more frequently necessary, major amputations were required less frequently in diabetic patients undergoing arterial reconstruction. Excellent results can be obtained even in patients presenting with a strong, palpable popliteal pulse, severe midtibial disease, and reconstitution of the distal tibial/pedal vessels.[20] In fact, the excellent re-

sults obtained using the popliteal artery for inflow have been an important advance in simplifying arterial reconstruction in diabetics. When constructing vein grafts to the dorsalis pedis artery, we have not found any significant difference in outcome as a function of the site of inflow or the technical approach, i.e., in situ vs. reversed or nonreversed. Thus we advocate a flexible approach to these reconstructions, taking advantage of the technical strategy best suited to the vascular anatomy at hand.[21]

This favorable outcome of arterial reconstruction demonstrates in a different way the absence of any microvascular occlusive lesion impeding outflow from arterial reconstructive procedures in patients with diabetes mellitus. Control of infection, expert arteriography, and technical expertise in the performance of extreme distal reconstruction are essential to successful limb salvage.

Summary

This is no evidence confirming existence of an occlusive lesion in the microcirculation in patients with diabetes mellitus as a source of ischemia in the foot. When ischemia occurs, it is due to atherosclerosis, which has a propensity to involve the infrageniculate arteries but not the arteries in the foot, especially the dorsalis pedis. Results of vein graft reconstruction to these distal arteries are as good as or better than results of more standard arterial reconstructions in nondiabetics. Success in management of the diabetic foot requires early drainage and control of infection followed by diagnosis of ischemia if present. Arteriography should always include foot vessels to determine whether distal arterial reconstruction is possible. With this approach, recent results of limb salvage in patients with diabetes have improved markedly.

References

1. Bild DE, Selby JV, Sinnock P, Browner WS, Braveman P, Showstack JA. Lower-extremity amputation in people with diabetes. Epidemiology and prevention. Diabetes Care 1989;12:24–31.
2. LoGerfo FW, Coffman JD. Vascular and microvascular disease of the foot in diabetes: Implications for foot care. N Engl J Med 1984;311:1615–9.
3. Strandness DE Jr, Priest RE, Gibbons GE. Combined clinical and pathologic study of diabetic and nondiabetic peripheral arterial disease. Diabetes 1964;13:366–72.
4. Conrad MC. Large and small artery occlusion in diabetics and nondiabetics with severe vascular disease. Circulation 1967;36:83–91.
5. Barner HB, Kaiser GC, Willman VL. Blood flow in the diabetic leg. Circulation 1971;43:391–4.
6. Irwin ST, Gilmore J, McGrann S, Hood J, Allen JA. Blood flow in diabetics with foot lesions due to 'small vessel disease.' Br J Surg 1988;75:1201–6.

7. Parving HH, Rasmussen SM. Transcapillary escape rate of albumin and plasma volume in short- and long-term juvenile diabetes. Scand J Clin Lab Invest 1973;32:81–7.
8. Wyss CR, Matsen FA, Simmons CW, Burgess EM. Transcutaneous oxygen tension measurements on limbs of diabetic and nondiabetic peripheral vascular disease. Diabetes 1964;13:366–72.
9. Fagrell B, Hermansson IL, Karlander SG, Ostergren J. Vital capillary microscopy for assessment of skin viability and microangiopathy in patients with diabetes mellitus. Acta Medica Scandinavica 1984;687:25–8.
10. Beach KW, Bedford GR, Bergelin RO, Martin DC, Vandenberghe N, Zaccardi M, Strandness DE. Progression of lower-extremity arterial occlusive disease in type II diabetes mellitus. Diabetes Care 1988;11:464–472.
11. Menzoian JO, LaMorte WW, Paniszyn CC, McBride KJ, Sidawy AN, LoGerfo FW, Connors ME, Doyle JE. Symptomatology and anatomic patterns of peripheral vascular disease: Differing impact of smoking and diabetes. Ann Vasc Surg 1989;3:224–228.
12. Apelqvist J, Castenfors J, Larsson J, Stenstrom A, Agardh C-D. Prognostic value of systolic ankle and toe blood pressure levels in outcome of diabetic foot ulcer. Diabetes Care 1989;12:373–378.
13. Parfrey PS, Griffiths SM, Barrett BJ, Paul MD, Genge M, Withers J, Farid N, McManamon PJ. Contrast material-induced renal failure in patients with diabetes mellitus, renal insufficiency, or both. A prospective controlled study. N Engl J Med 1989;320:143–49.
14. Stacul F, Carraro M, Magnald S, Faccini L, Guarnieri G, Dalla Palma L. Contrast agent nephrotoxicity: comparison of ionic and nonionic contrast agents. Am J Roentgenol 1987;149:1287–9.
15. Gibbons GW. The diabetic foot: amputations and drainage of infection. J Vasc Surg 1987;5:791–3.
16. Hurley JJ, Auer AI, Hershey FB, Binnington HB, Woods JJ, Nunnelee JD, Milyard MK. Distal arterial reconstruction: Patency and limb salvage in diabetics. J Vasc Surg 1987;5:796–800.
17. Sidawy AN, Menzoian JO, Cantelmo NL, LoGerfo FW. Effect of inflow and outflow sites on the results of tibioperoneal vein grafts. Am J Surg 1986;152:211–214.
18. Rosenblatt MS, Quist WC, Sidawy AN, Paniszyn CC, LoGerfo FW. Lower extremity vein graft reconstruction: Results in diabetic and non-diabetic patients. Surg Gynecol Obstet 1990;171:331–335.
19. Pomposelli FB, Jepsen SJ, Gibbons GW, Campbell DR, Freeman DV, Miller A, LoGerfo FW. Efficacy of the dorsalis pedis bypass for limb salvage in diabetic patients. J Vasc Surg 1990;11:745–752.
20. Cantelmo NL, Snow JR, Menzoian JO, LoGerfo FW. Successful vein bypass in patients with an ischemic limb and palpable popliteal pulse. Arch Surg 1986;121:217–20.
21. Pomposelli FB Jr, Jepsen SJ, Gibbons GW, Campbell DR, Freeman DV, Gaughan BM, Miller A, LoGerfo FW. A flexible approach to infra-popliteal vein grafts in patients with diabetes mellitus. Arch Surg (in press).

32
Technique of Reversed Vein Bypass to Distal Leg Arteries

RONALD L. DALMAN, LLOYD M. TAYLOR, and JOHN M. PORTER

Detailed attention to technical precision in the performance of popliteal and infrapopliteal bypass operations using autogenous vein as the conduit of choice has produced marked improvement in the patency results of lower extremity bypass operations in recent years. Using either the in situ or the reversed technique, most vascular surgeons have recognized significant improvement in their ability to salvage ischemic limbs threatened with amputation. The purpose of this communication is to describe our preferred technique for reversed vein bypass to popliteal and infrapopliteal arteries. Hopefully, the description is sufficiently detailed to permit the reader to benefit from our experience while avoiding some of the learning curve pitfalls we encountered.

Patient Selection

Almost all patients with salvageable limbs threatened by ischemia are potential candidates for bypass. Multiple factors, such as those listed in Table 32.1, previously considered by many as relative or absolute contraindications to limb salvage surgery, have not deterred us from the performance of leg bypass. In particular, failure of previously performed bypass grafts, even to infrapopliteal arteries, and presumed lack of suitable autogenous venous conduit are not contraindications to repeat bypass grafting.

However, two patient groups do present absolute and relative contraindications, respectively, for lower extremity revascularization. Institutionalized, neurologically impaired, nonambulatory patients obviously will receive little benefit from limb revascularization. We do not routinely recommend or perform limb salvage surgery in this patient group. A relative contraindication occurs in diabetics with end stage renal disease who present for treatment with large (>30 cm^2) gangrenous ulcers of the foot. Despite continued graft patency, we have been unable to consistently or predictably achieve wound healing or limb salvage in this setting.[1]

TABLE 32.1. Factors that are *not* contraindications to distal bypass.

1. Failure of previous bypass
2. Lack of sufficient saphenous vein
3. Failure to visualize pedal arch on arteriogram
4. Lack of angiographic runoff to foot vessels
5. Gangrenous ulcer involving calcaneus
6. Failure to visualize patent distal vessels on previous arteriogram
7. Calcified infrapopliteal arteries
8. Excessive anesthetic risk

Amputation, the inevitable alternative to lower extremity revascularization, constitutes an operative risk at least equal to that of bypass surgery with a far less desirable outcome.[2,3] Thus, we perform diagnostic arteriography in almost all patients with limb-threatening ischemia, regardless of perceived risk.

Arteriography

High quality distal leg arteriography is essential for optimal bypass surgery. Detailed views of tibial and pedal arteries opacified by high contrast density permit precise selection of anastomotic sites. Adequate visualization of distal leg vessels below multiple levels of proximal occlusion is a demanding exercise in angiographic technique. Selective catheterization of downstream vessels, delayed filming, use of intraarterial vasodilators, and external warming may all be necessary to provide precise anatomic detail.[4] Figure 32.1 demonstrates the detail obtained using these techniques. Digital subtraction techniques may supplement conventional arteriography, but the surgeon must use caution in interpreting these images. Poor contrast density often precludes precise selection of anastomotic sites using digital subtraction, and anatomic relationships to surrounding landmarks frequently are obscured. Operative prebypass arteriography should also be avoided. We find it necessary in far less than 1% of our patients. Need for prebypass operative arteriography[5] in more than occasional patients clearly indicates inadequate preoperative arteriography, a problem that must be corrected. High quality preoperative arteriography is essential for timely completion of operative planning prior to taking the patient to the operating room.

With adequate arteriograms, patients with chronic lower extremity ischemia in whom some revascularization operation is not possible are increasingly rare. We recently reported our experience with 627 critically ischemic limbs treated since 1980.[6] Primary amputation was performed only when no graftable distal vessels were present on good quality arteriograms and was required in only 14 patients (2.8%) and in none in the past three years.

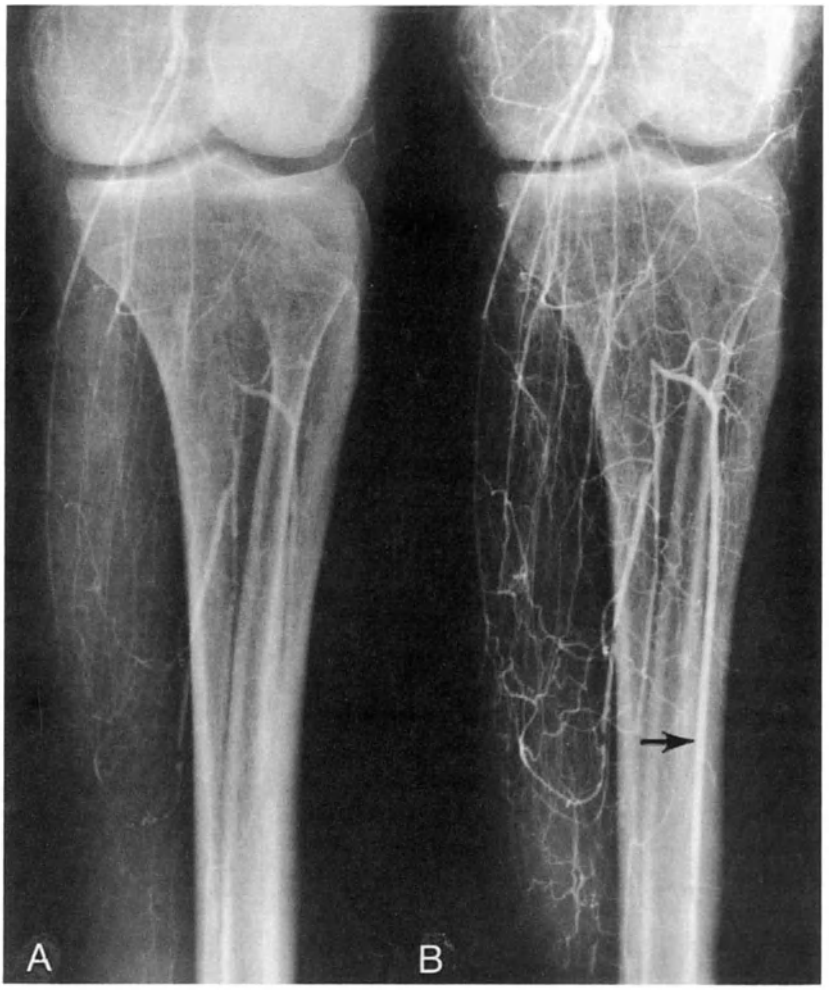


FIGURE 32.1. **A** and **C**: Conventional arteriogram fails to visualize patent distal vessels. **B** and **D**: Arteriogram following tolazoline injection and external warming demonstrates widely patent anterior tibial artery (arrows) suitable for bypass. **E**: Operative arteriogram. Reprinted with permission from Kozak BE, Bedell JE, Rosch J: Small vessel leg angiography for distal vessel bypass grafts. J Vasc Surg 1988;8:711–715.

Anesthesia

An infrainguinal bypass operation must be considered a major undertaking by both the surgeon and the anesthesiologist. The majority of patients are elderly and infirm, and severe multisystem diseases, especially coronary artery disease, are uniformly present. Haste is the enemy of a carefully constructed bypass. All provisions must be made to permit safe conduct of

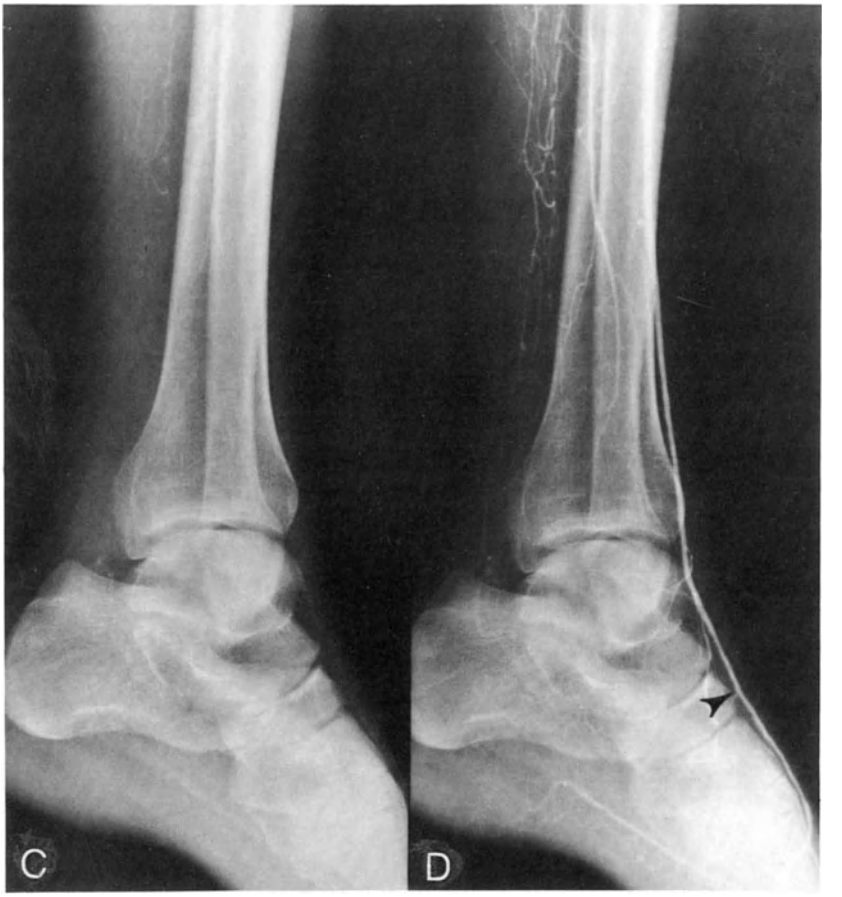


Fig. 32.1C–D

an unhurried procedure. Regional and general anesthetic techniques are both satisfactory. Full hemodynamic monitoring is appropriate in many patients. We routinely use the autotransfusion device for reoperative surgery. In selected patients, we administer nitroglycerin continuously during anesthesia and during the first postoperative day in an attempt to prevent and/or treat myocardial ischemia, as myocardial infarction remains a frequent complication of lower extremity bypass surgery.

Conduct of the Operation

Many leg bypass procedures are extensive and time consuming. Optimal surgical orientation must be toward performance of a technically satisfac-

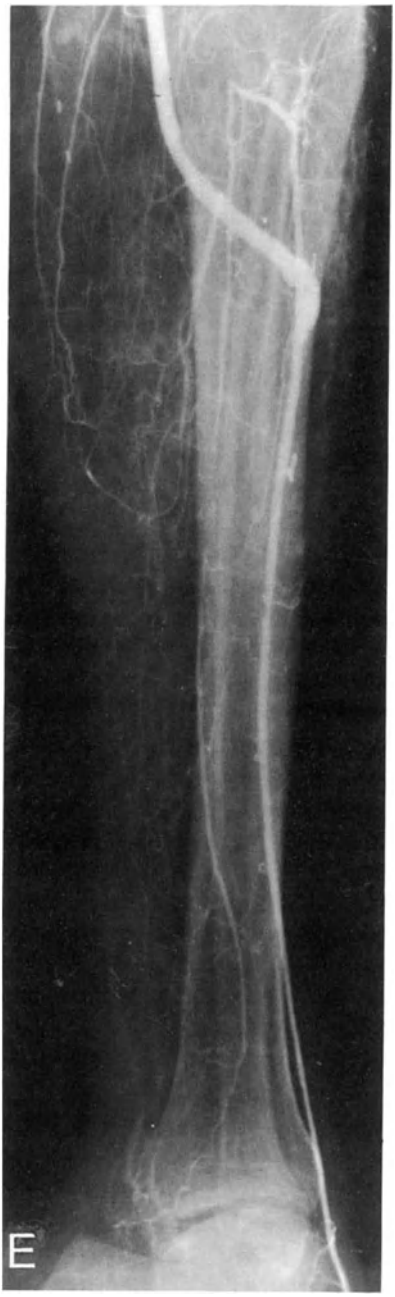


Fig. 32.1E

tory bypass without regard to time or scheduling demands outside the operating room. We routinely perform lower extremity bypass operations with two complete operating teams, each composed of senior surgeon and resident trainee. This approach minimizes both operating time and time from vein removal to reimplantation while maximizing exposure of all residents to the operation and insuring that adequate expertise is concentrated on all aspects of the procedure.

The synergistic effect of the presence of two senior surgeons is important to us and justifies the significant resource commitment. The added manpower allows simultaneous timely performance of multiple vein harvests, unhurried redissection of previously operated areas, extensive endarterectomies, etc., all of which may be necessary to achieve a satisfactory autogenous reconstruction. The investment in time and effort for successful limb salvage surgery in our experience is greater than, for example, that required for aortic aneurysm surgery, although the latter is uniformly regarded as a procedure of greater magnitude.

Equipment

Magnifying loupes, high quality miniature instruments, and bright, even lighting (frequently using a headlight) are essential for optimal results. Appropriate x-ray equipment must be available for operative arteriography. We use a separate sterile work table with seating for two persons, instruments, sutures, and adequate lighting, for preparation of the venous conduit (Table 32.2).

Vein Harvest

Procurement and preparation of the venous conduit are the sine qua non of bypass surgery. We perform this portion of the procedure first. The length of usable vein available frequently dictates necessary variations in the

TABLE 32.2. Equipment needed for distal bypass.

Magnifying loupes
Headlight
Miniature needle holders
Miniature forceps
Silastic vessel loops
Balloon catheters
Portable x-ray equipment
Electromagnetic flow meter
Sterile Doppler probes

TABLE 32.3. Alternate vein sources in patients with absent ipsilateral greater saphenous vein in decreasing order of preference.

Contralateral greater saphenous vein
Cephalic vein
Basilic vein
Lesser saphenous vein
Saphenous vein branches
Superficial femoral vein

planned arterial portions of the procedure. The operation is tailored to accommodate the vein, not the opposite. The approach to vein harvest differs slightly between patients with intact greater saphenous vein and those without. For all patients, however, the operative field is prepped to allow access to the extremity which contains the vein intended for use as well as at least one other extremity as a source of additional vein, if needed.

In patients with intact ipsilateral greater saphenous vein, simultaneous proximal and distal vein harvest is performed at the saphenous bulb and as far distally as necessary for the contemplated procedure. Great care is taken to avoid undercutting of skin flaps in an effort to minimize wound healing problems. Accurate dissection of the vein and division of side branches requires wide exposure, which we obtain through two or three nearly continuous incisions.

The surgeon should never accept the declaration of a previous operator that the saphenous vein in question is "too small" or "inadequate" to be used as a bypass conduit. Frequently, the problem was failure to identify the true greater saphenous at the bulb. Similarly, the estimation of vein size or adequacy may vary widely from operator to operator. If the vein has not been *physically removed*, it should be regarded as the conduit of choice until direct inspection reveals unsuitability.

For patients with no adequate greater saphenous vein, alternate sources are considered in the general order of preference listed in Table 32.3. The surgeon should not hesitate to use the contralateral greater saphenous vein, regardless of the status of the contralateral leg. If healing of vein harvest sites caused by ischemia in the donor leg becomes a postoperative problem, multiple options for revascularization are available. In actual practice, failure of wound healing in vein harvest incisions has occurred in only 3.4% of patients in whom contralateral saphenous vein was used as a bypass conduit. Healing after revascularization using alternate vein sources has been achieved in all patients.

In the absence of contralateral greater saphenous vein, we prefer to explore the arms for usable cephalic or basilic veins. An undamaged cephalic vein extending from wrist to shoulder is ideal. This vein can provide a

conduit of sufficient length for most infrapopliteal bypasses. Unfortunately, given the frequently hospitalized status of most patients with vascular disease and the well recognized position of the cephalic vein as the "intern's friend," this vein is rarely available in full length. The basilic vein from elbow crease to axilla is rarely injured by venipuncture and is invariably available regardless of external appearance of the arms. This vein segment is of limited length for performance of all but short extension grafts (as in conversion of a femoropopliteal to femoroinfrapopliteal graft). The great utility of the basilic vein segment is that it can be combined with other vein segments of limited length (lesser saphenous, greater saphenous remnant, etc.) to form bypass conduits. Regardless of the harvest site, arm veins function admirably as lower extremity bypasses, being usually of greater diameter than the corresponding leg veins, although more thin-walled, delicate, and difficult to handle. Controversy has surrounded the use of arm veins as bypass conduits. Some have condemned their use.[7] However, we and others who use arm veins frequently have found them to be eminently satisfactory conduits.[8,9]

If neither greater saphenous nor good quality arm vein is present at the beginning of the procedure, we begin the vein harvest with a search for usable lesser saphenous veins. For these operations, the patient is initially placed in the prone position and the posterior surface of the legs prepared and draped. The lesser saphenous veins are removed, the wounds closed, and the patient then placed in the supine position for the balance of the procedure. Although this step requires additional operative time, lesser saphenous harvest with the patient supine is awkward and frequently results in venous injury. Lesser saphenous veins have been of sufficient size and quality to be useful as bypass conduits in about 50% of our patients. The vein is more likely to be of suitable quality in patients in whom the ipsilateral greater saphenous vein has been absent for some time than in patients in whom greater saphenous removal was recent. A single lesser saphenous vein of normal length is sufficiently long to form a bypass to the above-knee popliteal artery. This conduit is useful for below-knee popliteal or infrapopliteal bypass only in patients in whom origin of the bypass distal to the common femoral artery is possible. Two lesser saphenous veins will form a conduit of adequate length to form any commonly performed lower extremity bypass.

Significant branches of the greater saphenous vein, including large parallel segments, exist in many persons. Depending on size, these branches may serve well as bypass conduits. Their size and location should be described in detail in the operative note at time of primary GSV harvest to permit their later use should additional vein be required for secondary procedures. We have extremely limited experience with use of the superficial femoral vein, although this conduit is preferred by some.[10] On those few occasions when we have used it this vein has proven satisfactory, especially as a short extension to another vein of limited length. Venous mapping by

duplex ultrasound[11] may be useful in identifying short segments of remaining, patent vein suitable for bypass after previous vein harvest. We have not relied upon pre-op duplex vein mapping and use the procedure infrequently.

Vein Conduit Preparation

Immediately upon removal the vein is placed on a specially equipped sterile table with an adequate work surface, seats, sufficient lighting, and good-quality miniature instruments (Fig. 32.2). One operating team, consisting

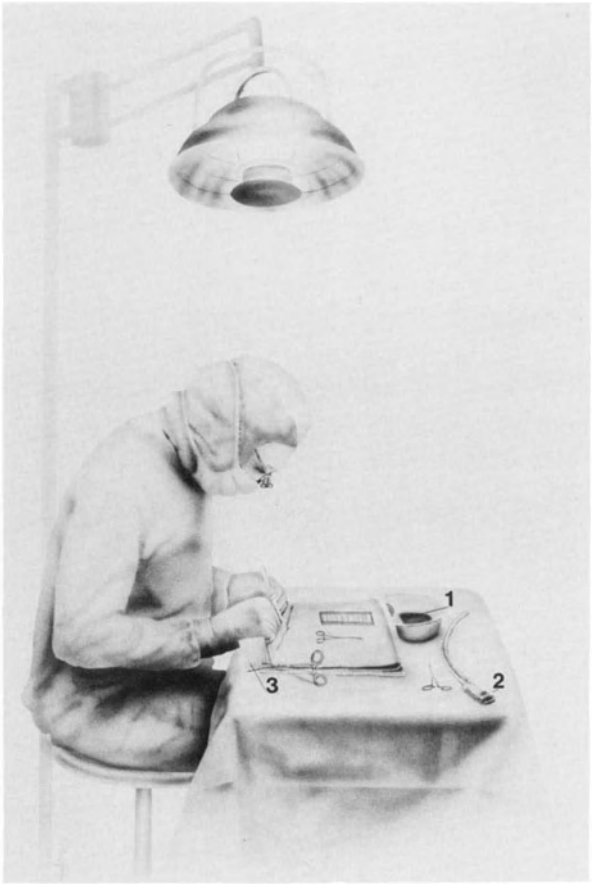


FIGURE 32.2. Preparation of the vein graft is completed at a separate "work bench" with comfortable seating and adequate lighting: **1**) chilled autologous blood; **2**) tunneling device; **3**) 5-0 silk ties for small branch ligation.

of senior surgeon and resident trainee, devotes full attention to preparation of the venous conduit while the other team prepares arterial sites for anastomosis.

We use chilled (4°C) autologous blood with added heparin (30 units/ml) and papaverine (0.5 mg/ml) to distend the vein, insure ligation of all side branches, and confirm patency. Distension is achieved manually using a Titus® needle on a small syringe. We make no effort to monitor intraluminal distending pressure as in our experience this has not been an important variable. Size has been emphasized as an important criterion of vein suitability. In our opinion vein size is far less important than quality, although we do not routinely use veins smaller than 3 mm in diameter. The vein (or veins) must be carefully inspected for sites of thrombosis and/or sclerosis. Vein grafts containing thick-walled, albeit patent, sclerotic segments that are not easily distensible have not functioned satisfactorily in our experience. The absence of normal distension may be the only evidence of multiple intraluminal synechiae and/or sclerotic valves that, if not removed, doom the vein to early thrombosis.

Performance of the maximum number of autogenous vein bypasses mandates that the surgeon be prepared to fashion composite vein grafts using

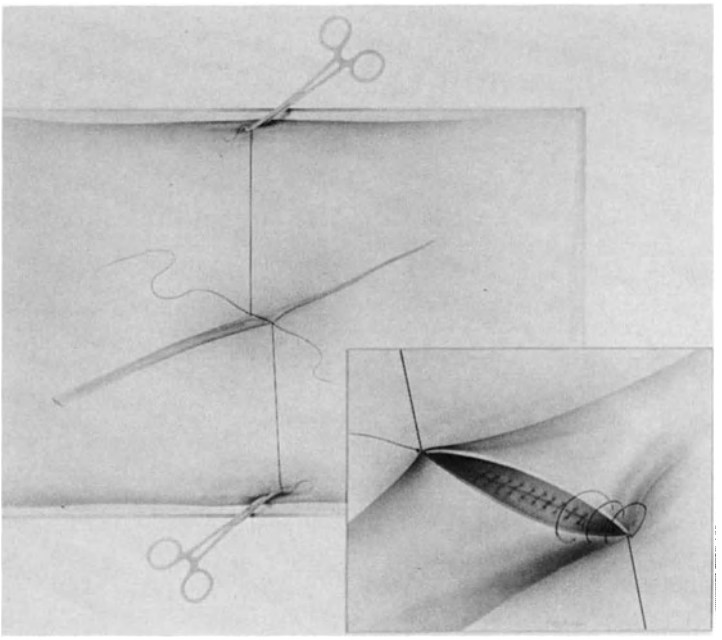


FIGURE 32.3. Technique of venovenous anastomosis. Note guy sutures anchored to operating tray for traction and stability. Inset: The anastomosis is beveled to accommodate components of varying diameter.

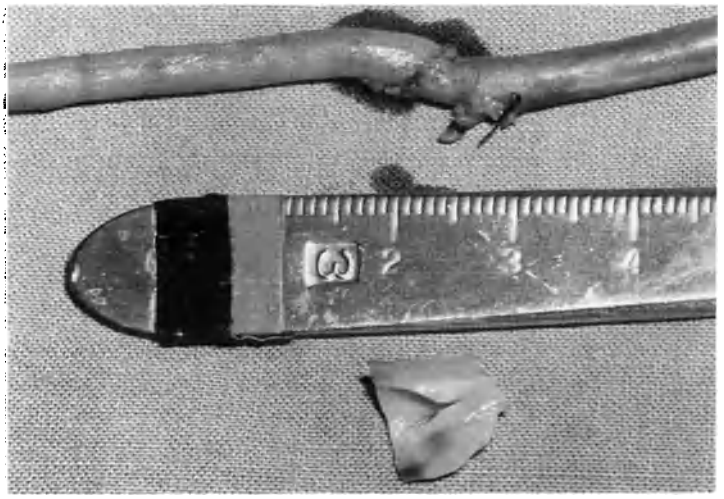


FIGURE 32.4. Completed venovenous anastomosis. This vein was of good quality throughout its length except for the excised sclerotic segment seen below the knife handle. This was discerned by palpation and complete lack of distensibility. Reprinted with permission from Taylor LM Jr, Porter JM: Reversed vs in situ. Persp Vasc Surg 1988;1:35–58.

vein from many different sites. Although this simple technical step was early advocated by Linton and Darling,[12] few have since emphasized its use. Many otherwise aggressive vascular surgeons regard simple end-to-end venovenous anastomoses as difficult and doomed to early failure. We have not found this to be the case. We construct such anastomoses by spatulating the ends of the venous segments to a length approximately two times the diameter of the larger of the two segments and completing closure with running sutures of 7-0 or 8-0 polytetraflouroethylene (PTFE) (Fig. 32.3). The anastomosis is tested, but only gross leaks are repaired by suture, as the need for such "leak stoppers" can only be accurately assessed when the graft is filled with blood at arterial pressure (Fig. 32.4).

Bypass Tunneling

Bypass grafts are placed along the anatomic course of the arteries they replace. Subcutaneous tunneling is used if the anatomic route is excessively scarred. A special tunneler designed by us and constructed in our own instrument shop is used to permit passage without the need for counter incisions (Fig. 32.5).

Grafts composed of a single length of saphenous vein are passed through the tunneler lumen while distended with chilled autologous heparinized blood, following which, two operating teams perform the proximal and

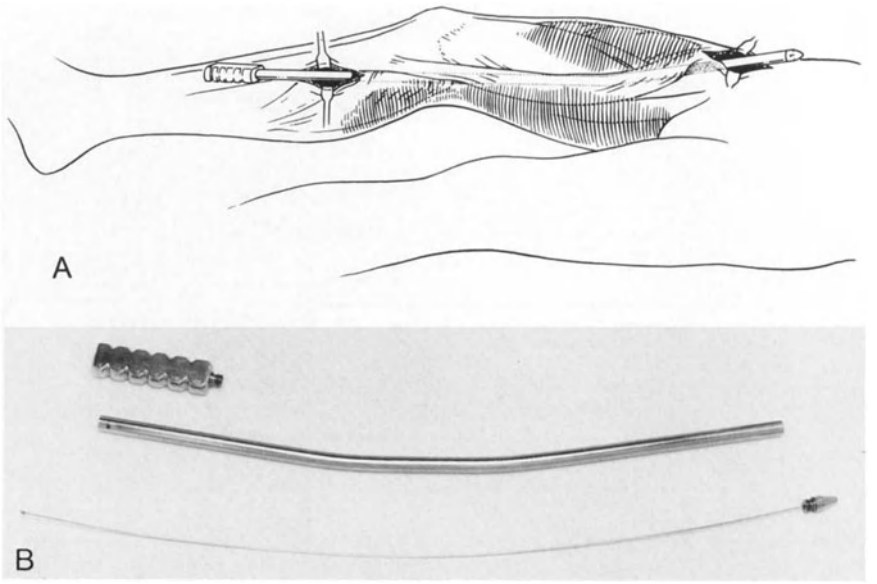


FIGURE 32.5. **A** and **B**: Tunneling device permits passage of grafts without counter incisions. Reprinted with permission from Taylor LM Jr, Porter JM: Current status of the reversed saphenous vein graft. Chapter in Bergan JJ, Yao JST (eds), *Arterial Surgery*, 1988. Orlando, Grune and Stratton, pp. 483–506.

distal anastomoses simultaneously. For grafts with one or more veno-venous anastomoses, the proximal anastomosis is performed first and the graft allowed to fill with blood under arterial pressure. The venovenous anastomoses are then inspected for hemostasis. Once this is assured, the graft is passed through the tunnel and the distal anastomosis performed (Fig. 32.6).

Proximal Anastomosis

The graft origin is chosen based upon the single requirement that there is no obstruction to arterial inflow. We do not preferentially use the common femoral artery for proximal anastomosis, using instead the most distal possible inflow site, both to conserve usable vein and to allow the hemodynamic advantage that may be associated with short grafts.[13] Many diabetic patients have widely patent common femoral and proximal superficial femoral arteries, the latter of which is frequently used by us as the origin for distal bypass grafts. Other patients have severely diseased common femoral and proximal profunda femoris arteries. The deep femoral artery is routinely revascularized concurrently with distal bypass if stenosis is

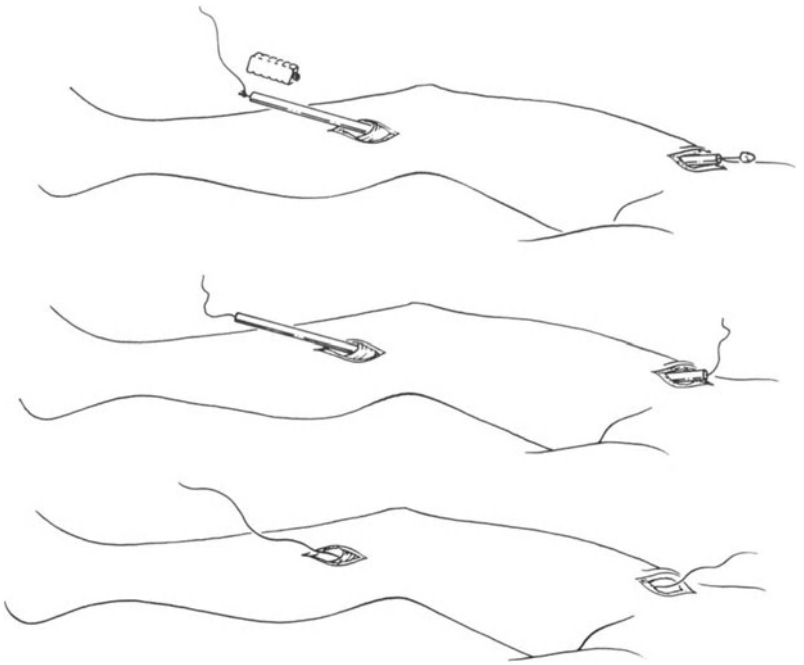


FIGURE 32.6. Use of tunneler. Specially constructed removable handle and stylet permit passage of grafts through metal tube.

present in this vessel. This is typically accomplished by endarterectomy. Deep femoral revascularization is performed concomitant with infrainguinal bypasses in 30% of our patients. The deep femoral artery may conveniently serve as the origin for the bypass, either by using the proximal anastomosis as a full length patch closure of the endarterectomy, or, if vein length is limited, by forming the proximal anastomosis from the distal portion of the revascularized deep femoral (Fig. 32.7).

The reversed vein technique requires anastomosis of the smallest portion of the vein to the proximal arterial site. We incorporate a side branch of the vein into the proximal anastomosis, as illustrated in Figure 32.8. We routinely tailor our vein graft harvest to permit use of these side branches, using this technique for graft origin in over 90% of our bypass procedures. Meticulous technique is essential. Great disparity in vessel size and wall thickness between artery and vein may be best solved by preliminary application of a vein patch to the artery to which the vein graft is subsequently anastomosed. In practice we use this technique infrequently. Both these maneuvers to avoid compromise of the proximal anastomosis were described by Linton.[12] We routinely use 6-0 or 7-0 PTFE sutures for proximal anastomosis.

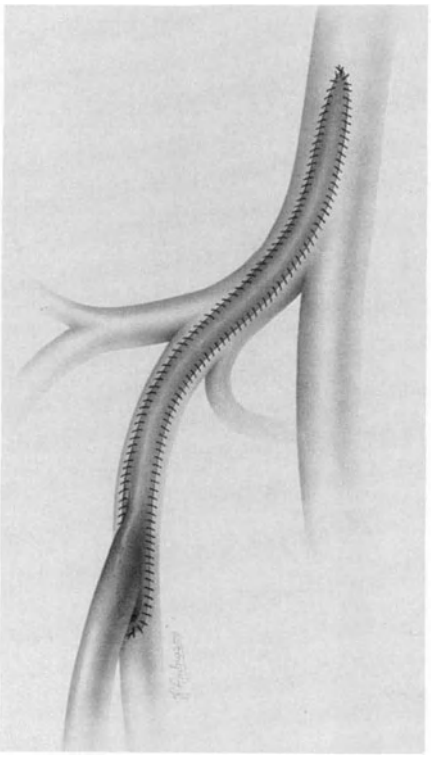


FIGURE 32.7. Use of vein graft origin as patch graft for considerable length of deep femoral artery.

Distal Anastomosis

The site for distal anastomosis is chosen solely on the basis of the preoperative arteriogram. Properly performed studies, described in the section on angiography, provide the most reliable indication of relative size and lack of disease of the recipient artery. The surgeon must resist the temptation to change the operative plan based upon findings of inspection and palpation at operation. External physical evaluation of distal arteries is notoriously misleading. Thus, nearly normal arteries may appear unusably small because of normal spasm, and widely patent arteries may seem unusable because of extensive calcification which, in fact, is generally inconsequential. The angiographic image of the vessel *lumen* is the most important determinant of suitability for anastomosis. The popliteal artery is normally approached from its medial side. Alternate lateral exposure is quite acceptable if the medial route is contraindicated by infection or other factors.

The posterior tibial and peroneal arteries are most conveniently approached through a medial calf incision and the anterior tibial through

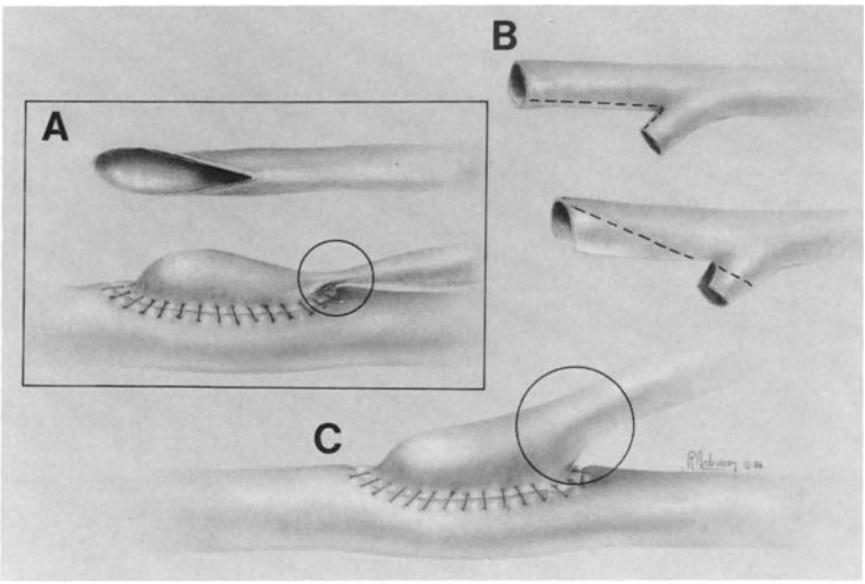


FIGURE 32.8. Technique of proximal anastomosis. **A**: Conventional anastomosis is prone to narrowing in proximal portion of graft. **B**: Incorporation of side branch into anastomosis. **C**: Narrowing prevented. Reprinted with permission from Taylor LM Jr, Edwards JM, Phinney ES, Porter JM. Reversed vein bypass to infrapopliteal arteries: Ann Surg 1987;205:90–97.

an incision lateral to the tibia. The distal posterior tibial, plantar arteries, and dorsal pedal arteries are exposed through longitudinal incisions directly over their course on the ankle or foot. Alternately, the anterior tibial artery can be exposed through a medial calf incision by dividing the interosseous membrane. We rarely use this exposure because of frequently encountered troublesome venous bleeding. The distal popliteal and entire posterior tibial and peroneal arteries can be widely exposed through a midline posterior calf incision. We have found little use for this technique. Although some have advocated fibulectomy for exposure of the peroneal artery, we have never perceived the need for this step, as in our experience the artery is easily and conveniently exposed from the medial side.

Whichever approach is chosen, dissection of distal arteries, especially those below the popliteal level, must be accomplished in a meticulous, unhurried fashion with adequate exposure, lighting, magnification, and appropriately delicate instruments. Multiple large adherent veins with interlocking branches surround each of the infrapopliteal arteries. Troublesome bleeding occasioned by entry into these vessels is best avoided.

We choose to expose completely 3–4 cm of artery in preparation for anastomosis. Rubber or plastic vessel loops are used to stabilize and elevate the artery and control backbleeding in vessels sufficiently soft to allow

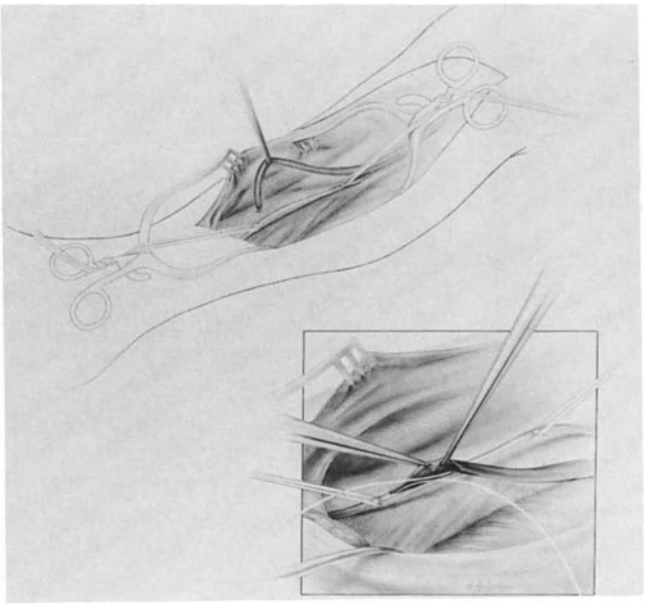


FIGURE 32.9. Exposure of infrapopliteal arteries. Note generous incision, dissection of sufficient length to facilitate anastomosis, and use of Silastic® loops for control and stabilization. Inset: Use of intraluminal balloon catheters for control of hemorrhage in calcified arteries.

compression (Fig. 32.9). Side branches (which may be minute) are carefully preserved and controlled with slings of fine silk. Once the vessel is controlled, an arteriotomy is created, which in length is 10–20 times the diameter of the vessel for infrapopliteal arteries and 2–3 times the diameter of the popliteal artery. Thus, most anastomoses are 20–30 mm in length. The vein is cut at an angle and carefully tapered to provide a flat angle of entry as well as a gradual transition in diameter from vein graft to artery (Fig. 32.10). This technique obviates much of the distal size mismatch otherwise inherent in reversed vein bypass, produces an acceptable angiographic appearance, and has been associated with a high success rate (Fig. 32.11). Continuous suture technique with 7-0 or 8-0 PTFE is used routinely.

Many infrapopliteal arteries are heavily calcified, especially in diabetics. The consistency of these vessels may appear formidable, especially to the uninitiated. Given a widely patent artery by angiography, we have to the present never encountered an infrapopliteal artery unsuitable for anastomosis. Heavily calcified vessels are opened with a longitudinal arteriotomy without any effort at clamping or other vascular control. Gently inflated balloon catheters are used to control backbleeding. In especially rigid vessels, a small ellipse of tissue about the arteriotomy is removed to insure a patent anastomosis. Special needles have been developed to aid in penetra-

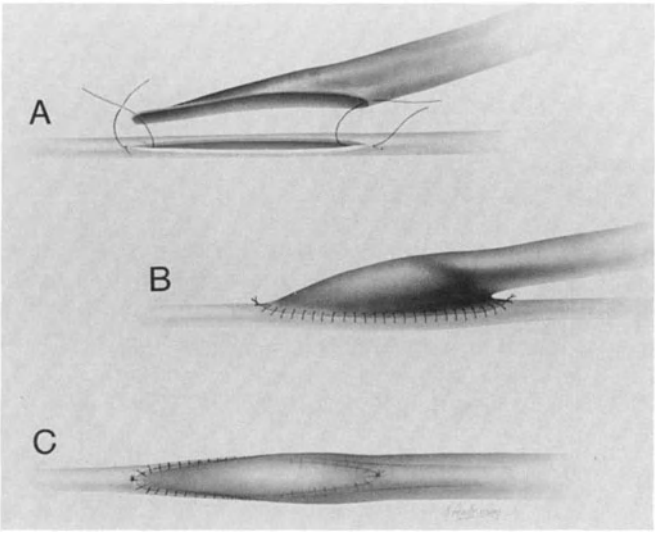


FIGURE 32.10. Technique of anastomosis to infrapopliteal arteries. **A**: Note angle of vein transection and generous length of anastomosis. **B**: Note "flat" angle between vein and artery. **C**: Tip of vein is carefully tapered to gradual transition from vein to artery diameter.

tion of calcified arteries and are a material improvement over those previously available (Piercing Point®, W.L. Gore). Fragmentation of bits of calcified artery wall is common, and these pieces must be irrigated away to prevent distal embolization. In recent years we have come to use PTFE sutures, 6-0, 7-0 and 8-0, almost exclusively for vein bypass procedures.

Once distal anastomosis is completed and flushed, forward flow is established and hemostasis obtained. Pressure and patience frequently achieve more effective hemostasis than multiple sutures, any of which may compromise the anastomosis. Topical thrombin spray (Thrombostat®, Parke-Davis) has been especially useful in our recent experience.

Assessment of Repair Adequacy

Direct measurement of graft flow, Doppler assessment of distal signals, and completion arteriography are all used to document satisfactory graft function. The electromagnetic flowmeter probe (usually 4 mm) is applied directly to the graft. Most infrapopliteal bypasses have initial mean flow from 60 ml/min to 150 ml/min. Values lower than 60 ml/min should raise suspicion of technical problems. Once basal flow readings are recorded, papaverine (30 mg) or tolazoline (25 mg) is injected into the proximal graft. Measured flow should at least double within 60–90 seconds following

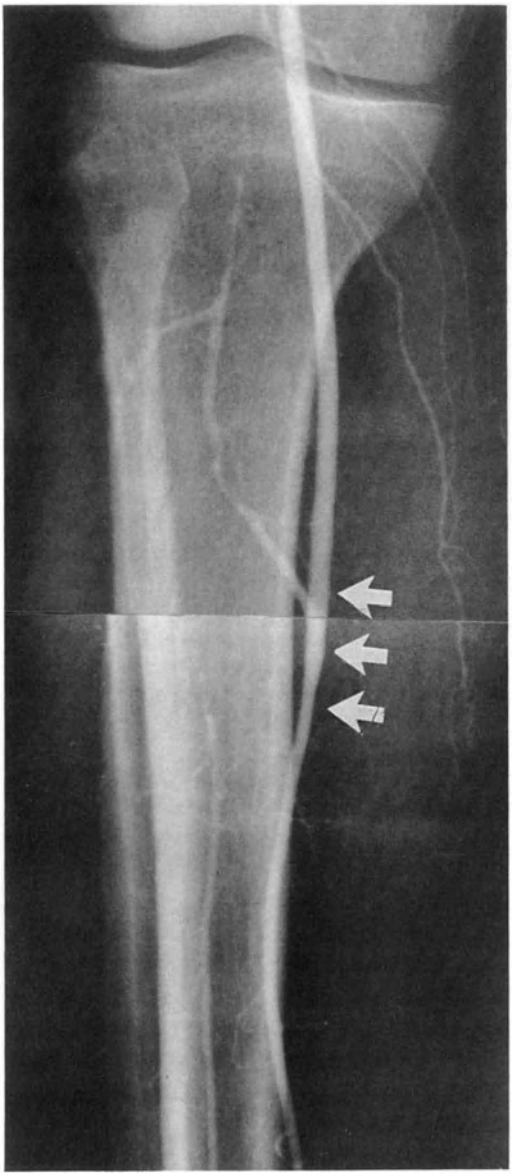


FIGURE 32.11. Angiographic appearance of properly constructed distal anastomosis. This graft has been patent for four years.

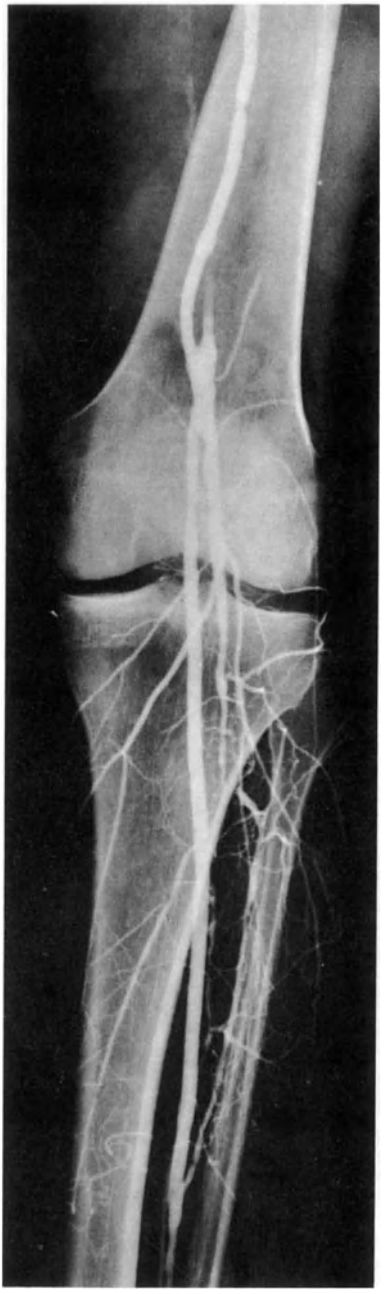


FIGURE 32.12. Operative completion arteriogram demonstrating widely patent sequential femoral-popliteal-anterior tibial bypass with excellent contrast density.

this step. Absence of this response indicates a potential mechanical problem and mandates completion arteriography.

A sterile Doppler probe is used to directly insonate the graft and the distal vessels at the ankle and foot levels. Absence of crisp arterial signals at all expected locations should never be attributed to spasm and is an absolute indication for completion arteriography.

Completion arteriograms are performed by cannulating the proximal vein graft with a 20- or 22-gauge polyethylene catheter, occluding the inflow, and hand injecting 15 cc 60% Conray®. A single exposure is made with the film positioned directly beneath the extremity (not within the operating table). This inflow occlusion technique produces reliable high quality images with good contrast density (Fig. 32.12). *Any* irregularity, stenosis, or intraluminal defect revealed by this technique must be explained and corrected. Spasm, which occurs surprisingly often in these heavily diseased arteries, is easily recognized and differentiated from technical flaws.

Given a satisfactory flow measurement, good quality distal Doppler signal, we have not recognized early graft failure. In recent years we have used completion arteriography much less frequently than previously.

Wound Management

Incisions are closed with absorbable sutures for subcutaneous tissue. Avoiding both fat necrosis on the one hand and hematoma/seroma accumulation on the other is best accomplished in our hands by using a single layer of subcutaneous sutures in all incisions except the groin, where two layers are used. The skin is closed with continuous subcuticular absorbable suture, which we believe has decreased the incidence of skin edge necrosis seen with other techniques used in the past. Suction drains are used liberally, both in distal leg incisions, where postoperative oozing from transected revascularized calf muscle is frequent, and in other incisions as needed, especially previously operated sites.

Postoperative Management

Patients are monitored in an intensive care setting until stable. Ambulation is begun as soon as tolerated, never later than 48 hours postoperatively. Edema is inevitable in the operated legs and is especially severe in reoperative patients. Control is achieved through elastic stockings. An initial light compression stocking is applied the first postoperative morning and is replaced with a below-knee 30 mm gradient stocking as soon as allowed by incisional pain.

Pharmacologic Management

Aspirin, 325 mg orally each day, is begun the day prior to surgery and continued indefinitely. Sufficient heparin is given prior to arterial clamping to prolong the activated clotting time to 2-1/2 to 3 times normal. Additional heparin is given as needed to maintain this level. Once the bypass is concluded, sufficient protamine is given to completely reverse the heparin. Dextran 70, 6% in normal saline, 500 cc/day × 2–3 days, is given to patients with low flow grafts (<75 cc/min) and patients with extensive proximal endarterectomies. Topical papaverine is used liberally to combat visible spasm in infrapopliteal arteries.

Overview and Conclusions

In our opinion the most essential contribution to successful infrainguinal bypass is the surgeon's attitude. Mental commitment to spend the necessary time and expend the necessary effort is mandatory if autogenous vein bypass is to be achieved in a maximum of patients. The putative advantages of decreased operating time, decreased blood loss, and less tissue trauma quickly fade to insignificance when compared to the grim outlook for continued patency of a prosthetic bypass, including to the above-knee popliteal artery.

It is our hope that the technical steps described and illustrated in this report will allow the reader to achieve autogenous vein bypass in all patients in whom it is required without regard to availability of ipsilateral greater saphenous vein. Using the techniques described herein we have been able consistently to achieve an all-autogenous reconstruction in 93.5% of patients and have achieved a five-year life table primary graft patency of 68–89%, depending upon the site of distal anastomosis.

References

1. Edwards JM, Taylor LM Jr, Porter JM. Limb salvage in end-stage renal disease (ESRD). Arch Surg 1988;123:1164–1168.
2. Porter JM, Baur GM, Taylor LM Jr. Lower extremity amputation for ischemia. Arch Surg 1981;193:453–461.
3. Ouriel K, Fiore WM, Geary JE. Limb-threatening ischemia in the medically compromised patient: Amputation or revascularization? Surgery 1988;104:667–672.
4. Kozak BE, Bedell JE, Rosch J. Small vessel leg angiography for distal vessel bypass grafts. J Vasc Surg 1988;8:711–715.
5. Scarpato R, Gembarowicz R, Farber S, et al. Intraoperative prereconstruction arteriography. Arch Surg 1981;116:1053–1055.
6. Taylor LM Jr, Hamre D, Dalman RL, Porter JM. Limb salvage vs. amputation for critical ischemia: the role of vascular surgery. Arch Surg, in press.

7. Schulman ML, Badhey MR. Late results and angiographic evaluation of arm veins as long bypass grafts. Surgery 1982;92:1032–1041.
8. Taylor LM Jr, Edwards JM, Brant B, Phinney ES, Porter JM. Autogenous reversed vein bypass for lower extremity ischemia in patients with absent or inadequate greater saphenous vein. Am J Surg 1987;153:505–510.
9. Harris RW, Andros G, Dulawa LB. Successful long-term limb salvage using cephalic vein bypass grafts. Ann Surg 1984;200:785–792.
10. Schulman ML, Badhey MR, Yatio R, Pillari G. An 11-year experience with deep leg veins as femoropopliteal bypass grafts. Arch Surg 1986;121:1010–1015.
11. Ruoff BA, Cranley JJ, Hannan LA, et al. Real time duplex ultrasound mapping of the greater saphenous vein before in-situ infrainguinal revascularization. J Vasc Surg 1987;6:107–113.
12. Linton RR, Darling RC. Autogenous saphenous vein bypass grafts in femoropopliteal obliterative arterial disease. Surgery 1962;51:62–73.
13. Ascer E, Veith FJ, Gupta SK, White SA, Curtis MA, Bakal CW, Wengerter K, Sprayregen S. Short vein grafts: A superior option for arterial reconstructions to poor or compromised outflow tracts? J Vasc Surg 1988;7:370–378.

33
Air Embolism Associated with Changing an Introducer over a Wire

THOMAS BARNETT, JAMES REILLY, and KEUK YUM

Abstract

Two cases of air embolism associated with changing a right internal jugular introducer to a triple lumen catheter over a wire are presented. Both cases involved thin patients who were in the supine position for the procedure. The introducer catheter's large diameter leaves a large opening in the skin upon its withdrawal. In a thin patient, there may not be enough tissue to collapse around the catheter to prevent air from entering the venous system. We recommend avoidance of guidewire changes with introducers in the neck veins unless the Trendelenberg is used.

Air Embolism Associated with Changing an Introducer over a Wire

Venous air emboli are known to cause sudden deterioration and possibly death when massive.[1] Recently there has been a trend at our institution to change lines over a wire rather than perform a second percutaneous puncture. This is done as long as the site looks good and it is the first line change.

Case Reports

Case 1

A thin, elderly white male was two days status post an exploratory laparotomy. He was doing well clinically and, therefore, his arterial line and pulmonary artery catheters were removed. An introducer is a wide diameter catheter through which a pulmonary artery catheter is threaded. The 8 Fr. introducer was then to be changed over a wire to a triple lumen catheter. The introducer was in the right internal jugular vein. With the

patient supine, a wire was inserted into the introducer and removed over the wire. The resident performing the procedure heard a hissing sound and then witnessed an air bubble through a small amount of blood over the site. Within seconds, the patient stopped breathing, became asystolic and promptly died despite all resuscitative efforts. The family refused autopsy permission.

Case 2

A thin, 70-year-old white male was doing well five days status post exploratory laparotomy. He suddenly decompensated while having his right internal jugular introducer changed over a wire. Resuscitative efforts, including immediate intubation and inotropic support, were successful. Air embolism was suspected but never proved.

Discussion

It has become acceptable at our institution to change lines over a wire given no signs of infection in a clinically stable patient. It is time saving and the risks of an additional percutaneous puncture are avoided.

Based on the experience presented above, we believe it may be wise not to change an internal jugular introducer over a wire. The proximity of the internal jugular to the skin, especially in thin patients, along with the large diameter of the introducer, makes for a dangerous situation. The potential for an opening from outside the body to the internal jugular vein makes air embolism a distinct risk. During inspiration, the negative intrathoracic pressure that occurs is transmitted to the large veins leading to the heart.[2] Air embolism is more likely to occur in a hypovolemic patient who takes a deep breath. The likelihood of an air embolism occurring in a ventilated patient is greatly reduced because of the positive intrathoracic pressure. It is probable that we have avoided air emboli in other patients when we have changed the introducer over a wire only because they were being ventilated.

When changing a triple or single lumen, smaller diameter catheter over a wire, we believe air embolism has been avoided because the skin and subcutaneous tissue are able to close down around the wire upon withdrawal of the catheter.

In both cases presented above, the patients were thin. Perhaps if they had been heavy, they would have had enough subcutaneous tissue to close down around the wire preventing passage of air.

Perhaps both cases of air emboli presented above would have been avoided if the Trendelenberg position had been used for the procedure as recommended by other authors.[2,3] A review of the literature produced no other reports of air embolism associated with change of an introducer over

a wire. Despite the significant morbidity and 50% mortality associated with air embolism, there is a paucity of information in the literature.[4] The diagnosis of air embolism is usually clinical.[5] We suspect that some cases of air embolism are erroneously attributed to other causes. The other possible explanation for the lack of information may be explained by the fact that none of us are too quick to admit, no less publish, our complications.

We recommend avoidance of guidewire changes for introducer catheters in the internal jugular veins, especially in thin patients. If one must persist with an internal jugular introducer wire change, we recommend deep Trendelenberg and constant pressure at the site.

Summary

Changing lines over a wire has become more and more acceptable over the years. Two cases of dreaded air emboli occurred at our institution with changing of an introducer over a wire to a triple lumen catheter. The large hole left by the introducer upon withdrawal provides a possible entrance for air to the venous system. We recommend avoidance of quick wire changes for introducers in the neck. The Trendelenburg position should always be used when changing a line over a wire.

References

1. Takeshita J. et al. Masui 38(8):1000:4, 1989 (Eng. Abstract).
2. Peters JL. Air embolism occurring as a complication of central venous catheterization. Ann Surg April:375–377, 1978.
3. Levinsky WJ. Fatal air embolism during insertion of CVP monitoring apparatus. JAMA 209:966, 1969.
4. Kashuk J. Air Embolism After Central Venous Catheterization. SG&O 159:249–252, 1984.
5. Adar R, and Mozes M. Fatal complications of central venous catheters. Br Med J 3:746, 1971.

34
Comparative Study of the Healing of Precoated Vascular Dacron Prostheses

U. HAKE, H. GABBERT, S. IVERSEN, H. JAKOB, W. SCHMIEDT, and H. OELERT

Abstract

Knitted and woven Dacron grafts commercially coated with bovine collagen, gelatin and human albumin were implanted end-to-side between the infrarenal aorta and the bifurcation in 41 growing pigs. Grafts were explanted after 4, 8 and 12 weeks and evaluated for neointimal healing, peeling of the inner and outer capsule, thrombus formation, extent of neoendothelialization, absorption of the sealant, interstitial cellular infiltration and perigraft inflammation.

Six uncoated knitted grafts preclotted with blood served as a control and rapidly developed a firmly attached neointima lined with complete endothelium. Compared with coated grafts, the thrombus-free surface of uncoated grafts was significantly greater ($p < 0.05$). The impeded resorption of human albumin resulted in a delayed and incomplete neointimal healing and peeling of the outer capsule. Although the bovine collagen was only minimally crosslinked by formaldehyde, healing of the neointima was compromised in a thin woven graft that demonstrated peeling of the inner capsule even after 12 weeks. The identical collagen as well as bovine gelatin were quickly degraded in knitted grafts, and both prostheses showed transprosthetic infiltration at 4 weeks. However, all knitted grafts, sealed with either collagen or gelatin, were occluded after 8 and 12 weeks, respectively. Light microscopy revealed hyperplasia of smooth-muscle cells within the thickened distal anastomotic region.

These results provide evidence that a timely return of porosity is mandatory for development and maintenance of an intact neointima. Both the fabric structure as well as the method of preparing the coating are crucial variables to determine the rate of biodegradation. Knitted grafts coated with collagen or gelatin demonstrate superior incorporation, although late occlusions occurred in both types.

This chapter previously appeared as "Evaluation of the healing of precoated vascular dacron prostheses" in *Langenbecks Archiv für Chirurgie*, volume 376, number 6: pages 323–329, 1991. Used with permission.

Introduction

Clinical studies have widely documented the superior healing properties and good function of porous knitted Dacron grafts.[1,2] Their major technical intraoperative disadvantage, however, is the necessity for a frequently time-consuming preclotting maneuver with autologous blood to render the porous graft impervious at the time of implantation.[3] Emergency surgical revascularization often requires rapid restoration of blood flow; in case of full heparinization and cardiopulmonary bypass for proximal aortic reconstruction or intervention of extracardiac conduits, standard preclotting techniques may be totally impossible.

In these situations tightly woven Dacron protheses with a markedly lower porosity have been considered as an alternative.[4,5] Various complications of the internal capsule, however, like accelerated pseudointimal thickening or dissection of the pseudointima, have been demonstrated in these grafts.[6,7]

The concept of impregnating a knitted porous graft with a mammalian sealant to achieve a zero-implantation porosity was introduced in the early sixties.[8–10] Following degradation of the sealing agent, the knitted prosthesis should regain the initial porosity, thus allowing transinterstitial tissue ingrowth.[10] Technical problems, mainly associated with the application and preservation of the agent, however, resulted in an inhomogenous distribution of the sealant or cracking of the sealed surface and inhibited the widespread use of these prostheses.[11] Recently, the standardized fabrication of coated Dacron grafts has been accomplished and several types are now available.[12–15]

The present study was designed to investigate rate of development of the inner and outer capsule in four Dacron grafts commercially treated with different sealants compared with the healing parameters of a standard knitted Dacron prosthesis.

Material and Methods

Animals and Graft Selection

Forty-one female pigs weighing 16 to 20 kg were included in the study and cared for in accordance with the instructions of "Principles of Laboratory Animal Care" (formulated by the National Society for Medical Research) and "The Guide for the Care and Use of Laboratory Animals" (NIH Publication No. 80-23, revised 1985).

Animals were randomly assigned to one of the five groups: 1) knitted grafts coated with glutaraldehydetreated human albumin—USCI DeBakey® Vasculour® II, C.R. Bard Inc., Massachusetts (Group I, n = 9); 2) knitted grafts without sealing—control group, Meadox Microvel®,

Meadox Medicals, Inc., Newark, NJ (Group II, n = 6); 3) woven grafts coated with bovine collagen crosslinked by formaldehyde—Meadox Hemashield®, (Group III, n = 10); 4) knitted grafts coated with collagen crosslinked by formaldehyde—Meadox Hemashield® (Group IV, n = 8); 5) knitted grafts coated with gelatin crosslinked by formaledehyde, Gelsoft®, Vascuteck Ltd., Inchinnan, Scotland, U.K. (Group V, n = 8). All grafts were 8 mm in diameter and implanted as tubes. Apart from the albuminated graft, packaged in a sterile 0.9 % saline solution, all prostheses are supplied in a dry and sterile state. For each animal the time of stay in the study was determined prior to operation.

Operative Procedures

After sedation with 8 mg/kg azaperone (Stresnil®, Janssen Ltd, Neuss, Germany), animals were anesthetized with 10 mg/kg Metomidat (Hypnodil®, Janssen, Germany) intravenously. Prior to operation all animals received 2.0 g of cefuroxim (Zinacef®, Glaxo) intravenously.

The infrarenal aorta and the bifurcation were exposed by a left retroperitoneal approach. Coated grafts were beveled, and the animals were systemically heparinized (100 units/kg) prior to clamping. The proximal and distal end-to-side anastomoses were performed with a 5-0 monofil polypropylene suture. Interrupted sutures were used at the toe and heel of the anastomosis to prevent anastomotic narrowing. Uncoated prostheses were preclotted with autologous blood prior to application of heparin. After completion of the proximal anastomosis, several clamping and declamping maneuvers were performed to render the graft impervious. The intervening aorta was ligated after completion of the distal anastomosis. The mean length of all grafts was 4.6 cm (±0.6 cm).

Evaluation of surgical handling qualities at time of implantation included fraying of cut edges, suturability, conformability and suture retention. Postoperatively, the animals were returned to their cages and fed ad libidum. No anticoagulants or aspirin were administered.

Graft Harvesting

At prescheduled periods (4, 8 and 12 weeks) animals were anesthetized and received heparin (200 U/kg IV). The suprarenal aorta and one iliac limb were clamped. Animals were now sacrificed by an intravenous lethal dose of Metomidat. The left iliac limb was transsected, and the aorta was flushed clear with chilled phosphate-buffered solution (PBS) to remove any blood from the graft. The entire graft, with the adjacent infrarenal aorta and iliac limbs, was quickly removed and freed from adherent retroperitoneal tissue. Specimens were opened longitudinally, immersed in PBS, pinned out on Teflon sheets and photographed. Sagittal sections of both anastomoses with at least 5 mm of the adjacent aortic wall and cross-

sectional as well as longitudinal sections of midportion of the graft were carefully excised and evaluated separately. Specimens for light microscopy (LM) were fixed in a 4% buffered formaldehyde solution, embedded in paraffin, and stained with hematoxylin and eosin, Masson's trichrome and van Gieson's. Prostheses with collagenous coating were stained with Sirius red F3BA in saturated picric acid and studied with a polarization microscope to distinguish between the subject's own collagen and residual bovine collagen.[16] Various neointimal cell types were identified by immunohistochemical staining. Commercially available antibodies to factor-VIII related antigen (F-VIII: r-Ag) reacted with vascular endothelial cells of the outer capsule and with luminal flattened cells. Smooth-muscle cells were characterized by staining with mouse monoclonal antibodies against smooth muscle-specific alpha-actin. The detection system was biotin-avidin with horseradish peroxidase as a marker.[17,18]

For scanning electron microscopy (SEM), specimens were fixed with 2.5% glutaraldehyde in 0.1 molar phosphate buffer, pH 7.2, followed by fixation in 2% osmium tetroxide at 37°C for 2 hours. After careful rinsing with phosphate buffer, the fixed specimens were dehydrated in a graded series of acetone to the critical-point of carbon dioxide, mounted on studs, spatter-coated with gold palladium and examined with a Philips SEM 500 scanning electron microscope.

The specimens for transmission electron microscopy (TEM) were post-fixed with cold 1% osmium tetroxide, dehydrated in a graded series of ethanol, immersed in propylene oxide and embedded in epoxy resin. Ultrathin sections were cut and stained with methylene blue and examined by light microscopy. Representative sections were stained with uranyl acetate and lead citrate and examined with a Philips 410 electron microscope. In addition, specimens of all grafts were examined by LM and SEM prior to implantation. For SEM evaluation, the albuminated grafts and uncoated grafts preclotted with blood were carefully pinned out on Teflon sheets, treated with 2.5% glutaraldehyde and postfixed with osmium tetroxide. Further processing was identical to that described for ex vivo grafts. Grafts supplied in a dry state were immediately mounted on studs, spatter-coated with gold and examined.

End pieces and midportions of grafts underwent chemical analysis after mincing and shaking for 7 days in 0.9% saline. Residual aldehydes were evaluated by adding 2 ml of supernatant to 8 ml of a solution consisting of 50 ml chromotropic acid (1%) and 200 ml sulphuric acid (12.5 mol/L). The combination was placed in a dry cabinet at 120°C for 30 minutes, allowed to cool down, and extinction was measured at 570 nm with a photometer.

Plasticizer emission was analyzed by twice extracting 40 ml of supernatant with 2 ml of 1,2,2,Trifluorotrichloroethane. Following exsiccation, extracts underwent infrared spectrometry using a Perkin Elmer PE 1420 spectrometer, followed by evaporation and gravimetrical analysis.

Data Analysis

Macroscopic Evaluation

Macroscopic recording included patency, thrombus formation and attachment of the inner and outer capsule. The percentage of thrombus-free surface area was determined from magnified photographs of longitudinally opened specimens using a digital planimeter.

Extent of neointima was scored as follows:

no neointimal glistening	0
glistening confined to suture line	1
island of glistening within the midportion	2
complete neointima	3

Degree of attachment of the neointima or the external capsule to the prosthetic wall was determined during preparation of the graft and evaluated as follows:

firm attachment	0
easy blunt dissection by forceps	1
spontaneous dissection	2
periprosthetic bleeding or formation of seroma	3

Histologic Evaluation

Thickness of the neointima was determined with an ocular micrometer by calculating the mean of at least four measurements between the luminal surface and peaks and valleys of the crimps.

Rate of resorption of the sealant was scored as follows:

no residual sealant	0
sealant <50%	1
sealant >50%	2
unchanged sealant	3

Degree of prosthetic infiltration was evaluated in longitudinal sections:

no infiltration	0
infiltration <50%	1
infiltration >50%	2
transprosthetic infiltration	3

Perigraft inflammation within the external capsule was assessed according to the ratio between mononuclear cells and connective tissue:

fibrosis	0
infiltration perivascular	1
infiltration focally and dense	2
tapelike and confluent	3

Statistical Analysis

Continuous values were compared with the U-test. A statistical significance was accepted when the p value exceeded 0.05.

Results

In Vitro Studies

Light Microscopy of the Prosthetic Wall

In contrast to all knitted prostheses that demonstrated an interfibrillar penetration of the coating within the Dacron bundles, bovine collagen of the woven grafts was mainly distributed on the inner and outer surface.

SEM of the Prosthetic Surface

All prostheses, coated either by collagen or gelatin, showed a smooth and homogenous surface of the inner velour. The albumin-coated grafts revealed a fragile inner surface that appeared to be only loosely attached to the prosthesis.

Following a threefold preclotting maneuver with autogenous blood and intervening rinsing by heparinized saline, the uncoated knitted prostheses demonstrated an incomplete coverage of the surface by a thin fibrin mesh with various entrapped blood cells.

Aldehydes and Plasticizers

Apart from the gelatin-coated prostheses that showed more than 25 g/g prosthesis, only traces of aldehydes below 5 μg/g prosthesis were detected among coated and uncoated grafts.

Infrared spectrometry identified no classes of emitted plasticizers. Gravimetrical analysis revealed the least quantity of emitted plasticizers for the albuminated graft, while concentrations of 0.4 mg ($\pm$0.18 mg) per g prostheses were found for uncoated grafts and prostheses coated by gelatin.

Handling Characteristics

Ease of handling and suturability was worst for the albuminated graft. Fraying of the cut edges and inferior suture retention were the major disadvantages of the woven graft and required tailoring with an electrocautor. Surgical handling of grafts impregnated by collagen or gelatin was identical to the handing of the uncoated prosthesis after immersion in saline.

In Vivo Studies

Major perioperative complications included acute bilateral lower limb ischemia in one animal (group III) and intractable marasmus in a second one (group I). The perioperative course of the remaining animals was uneventful.

Transprosthetic Bleeding

No transprosthetic blood loss occurred in coated prostheses. The uncoated knitted graft required 4 preclotting maneuvers in 3 and 4 in 2 animals.

Neointimal Healing

Gross differences of neointimal development existed among coated and uncoated prostheses (Table 34.1).

The preclotted knitted standard prosthesis (group II) demonstrated a rapid and complete neointimal healing within 4 weeks. LM revealed a layered neointima that was lined by endothelium. Circumferentially orientated cells beneath the endothelium were identified as smooth-muscle cells by alpha-actin staining. Apart from flimsy fibrinous filaments, SEM showed a nearly complete coverage of the grafts midportion with endothelial-like cells. No significant neointimal changes occurred after 8 or 12 weeks, and the mean thrombus-free area was 98% (±1.9%).

The albumin-coated knitted grafts (group I) displayed islands of firmly attached glistening neointima within the midportion after 4 weeks. Light microscopy revealed a thin and inhomogenous neointima. Clusters of endothelium, separated by large areas covered with fibrillar structures, were observed by SEM. After 8 and 12 weeks, grafts were completely covered by an easily detachable neointima with thrombus formation (Table 34.2).

The thin neointima of woven collagenated grafts (group III) confined to the suture lines after 4 weeks. Apart from the anastomotic region, no endothelial lining could be identified by immunohistochemical staining. After 8 and 12 weeks, areas of intact neointima with positive immunohistochemical evidence of endothelium close to severe ulcerative alterations were present). Prosthetic crimp valleys were filled with an acellular and homogenously stained mass. Peeling of the inner capsule was easily provoked by tailoring the specimens for histologic evaluation. SEM demonstrated alternately a large island of intact endothelium and only endothelial clusters separated by areas consisting of collagen. Distribution of luminal clots was similar for albumin-coated or woven collagen-coated grafts, and no significant differences of the mean thrombus-free area were found, (83.7% (±16.4%) vs. 81.6% (±12.6%).

All knitted grafts coated with collagen (group IV) were occluded after 8 weeks following an initially uncompromised neointimal development.

TABLE 34.1. Median score values of the neointimal configuration for group I to V at 4, 8 and 12 weeks.

	I			II			III			IV			V		
	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12
Patency (%)	100	100	100	100	100	100	100	100	100	50	0	0	100	100	0
Neointimal extension	2	3	3	3	3	3	1	3	3	3	*	*	2	3	*
Neointimal peeling	0	2	2	0	0	0	0	1–2	2	0	*	*	0	0	*

* Organized thrombotic occlusion

TABLE 34.2. Median score values depicting the morphology of prosthetic wall and outer capsule for group I to V at 4, 8 and 12 weeks.

	I			II			III			IV			V		
	4	8	12	4	8	12	4	8	12	4	8	12	4	8	12
Sealant	3	2	1	—	—	—	3	1	1	0	0	0	0	0	0
Prosthetic infiltration	1	1	2	3	3	3	0–1	1	1	3	3	3	3	3	3
Neoadventitial peeling	2–3	2	2	0	0	0	2	2	1	0	0	0	0	0	0
Periprosthetic inflammation	2–3	2	2	1–2	1	1	1	1	1	1	1	0–1	1–2	1	1

Cross-sections showed an organized thrombotic occlusion originating from the distal anastomosis that had a mean thickness of 1878 μm ($\pm$133 μm) compared with 1500 μm ($\pm$86 μm) for uncoated prostheses ($p < 0.01$). LM of the subendothelial neointima of the distal anastomosis revealed numerous spindle-shaped cells that gave a positive staining for alpha-actin antibodies. TEM identified these cells as myofibroblasts. SEM of the distal anastomosis showed severely altered endothelium with numerous defects. Among grafts coated with gelatin (group V), a glistening neointima was scattered within the midportion of the graft, and endothelial lining was confined to the suture line after 4 weeks. Apart from minor thrombus formation, an intact neointima was found after 8 weeks. LM showed a regularily layered neointima in most parts, and SEM confirmed an almost homogenous endothelial coverage of the grafts. After 12 weeks, the macroscopic and histologic features of the occluded graft were identical to those of the collagen-coated knitted graft.

Infiltration of the Prosthetic Matrix

Transprosthetic infiltration of uncoated grafts (group II) by macrophages, multinucleated giant cells and connective tissue was completed after 4 weeks.

Apart from the anastomotic region, cellular infiltration of the albumin-coated (group I) or the collagen-coated (group III) woven prosthesis remained incomplete at 4 weeks (Table 34.2).

At three months, spots of albumin, scattered within the grafts midportion, were still present. Only now, foreign body cells were detected in the fabric. Independent from time of explantation, the outer capsule of the albuminated graft was easily detached from the graft and LM revealed a chronic inflammatory response.

The collagen-coated woven graft showed a partial degradation of the sealant on the luminal surface between 4 and 8 weeks. The tapelike collagen coating remained visible on the luminal surface of the graft up to 12 weeks. Again, only a minimal tissue ingrowth of the prosthetic wall was detectable.

In knitted grafts coated with collagen or gelatin, no sealant was found after 4 weeks and transprosthetic infiltration was already completed. Both grafts showed an abundant invasion of macrophages and giant foreign-body cells. The outer capsule consisted of fibrous tissue and was firmly adherent to the graft.

Discussion

The critical importance of porosity to determine long-term performance of vascular synthetic grafts has been repeatedly emphasized. Consequently,

timely degradation of the sealant is the most relevant factor to guarantee tissue ingrowth and function of the graft. Wesolowski (10) proposed an ideal resorption of a sealant at the rate of fibrin. On the other hand, a premature degradation may provoke transinterstitial bleeding.

Zero porosity at the time of implantation is evidently warranted by all coated grafts. Scanning electron studies demonstrated a plain and homogenous surface for grafts coated with collagen or gelatin. Only grafts coated with albumin showed a cracked and fragile surface of the luminal albumin layer, although this graft proved to be impervious to blood as well. The globular structure of albumin molecules is blamed for the inferior attachment of this sealant to the graft.[19]

The deterioration of surgical handling compared to a standard knitted prosthesis was most distinct for the stiff albuminated graft. In fact, the quantity of emitted plasticizers was least for these grafts. On the other hand, evaluation of plasticizers is difficult. One of the potential hazards of a plasticizer is migration into the surrounding tissue. Induction of cancer among rats and mice, as well as hemolysis, have been reported for Di-(2)-ethylhexylphthalate, widely used to soften PVC.[20,21]

Great differences exist between the rate of resorption for various sealants. The mechanism of degradation is partially known for collagen-coated prostheses. Based on ample experience gained with various orthopedic sponges and implants, breakdown of insoluble collagen is performed by collagenase carried by the lysosomes of macrophages.[22] Both insoluble collagen fibers as well as soluble collagen subunits may promote invasion of cells.[23,24] As for collagen the process of degradation is dependent on the extent of crosslinking.[25] Jonas et al.[26] emphasized the role of crosslinking in a clinical trial where pediatric ventriculopulmonary conduits, sealed with strongly crosslinked collagen, behaved like impervious prostheses and demonstrated a thickened and only loosely attached inner capsule. In our study, both the knitted and the woven Dacron graft were coated by minimally crosslinked collagen. In fact, all collagen was absorbed in knitted grafts within 4 weeks and a transprosthetic bridging was complete. In woven grafts treated with the identical bovine collagen, the relevance of the prosthetic structure to determine both tissue ingrowth as well as for consecutive resorption of the sealant was clearly outlined. Tapeline traces of the sealant remained visible up to 3 months on the luminal side of the prostheses. Apart from the anastomotic region, no transprosthetic bridging between the inner and outer capsule was detected. Similar observations are reported by Scott at al.,[27] who found residual bovine collagen up to 90 days.

Compared to the woven collagenated grafts, healing of the inner capsule was accelerated for the albuminated graft; compared, however, to the uncoated standard prosthesis, healing of the inner capsule and tissue incorporation was markedly delayed, although the albumin-treated graft should

offer excellent healing conditions because of its knitted structure and a water porosity of 1.200 ml/min/120 cm H_2O. These results are counter to data obtained by other authors.[28] In contrast to Guidoin et al. (12), who reported a rapid resorption of albumin in dogs within 4 weeks, the fast-healing pig in our model showed spots of albumin even after 12 weeks of implantation. The indispensable foreign-body cells that evidently represent the host's response to exposed Dacron fibers were only focally detectable after 3 months.

Degradation of albumin is mainly achieved by hydrolysis.[29] The probable reason for delayed resorption of albumin is the process of crosslinking the sealant with glutaraldehyde.[30] The porous structure of this fabric should have been an excellent prerequiste for a fast resorption of sealant.

A unique morphologic feature of albuminated grafts was the lack of perigraft adherence, leaving the graft nearly totally free within a connective sheet. A moderate to severe chronic inflammatory response was present within the periprosthetic tissue in all albuminated grafts, representing the failure of graft incorporation.

The high incidence of graft occlusion in knitted grafts pretreated with collagen or gelatin is unclear. Considering the fact that pigs are fast-healing subjects, anastomotic neointimal hyperplasia may play a certain role within the period of 12 weeks.[31] In fact, the occlusion level of the distal anastomosis had reached the level of the native aorta, and collaterals between the supra- and infrarenal aorta gave evidence of a slowly progressive flow obstruction. Light microscopy revealed numerous cells with a positive staining for alpha-actin antibodies, and transmission electron microscopy identified these cells as myofibroblasts.[32] Apart from the configuration of the distal anastomosis that may contribute to the development of anastomotic neointimal hyperplasia, the question arises why knitted prostheses coated by bovine collagen or gelatin are exclusively afflicted.[33,34]

In conclusion, no ideal off-the-shelf coated prosthesis is commercially available. Corresponding to the slow resorption of the sealant, neointimal development and incorporation were delayed in knitted grafts coated with albumin or in woven grafts sealed with weakly crosslinked collagen. While the treatment with glutaraldehyde is obviously responsible for the delayed degradation of the albumin, the tight woven structure compromises resorption of collagen and contributes to the inferior healing pattern of this prosthesis. Early healing of knitted grafts coated either with collagen or gelatin is satisfactory, although the excellent healing data evaluated for knitted porous Dacron prostheses have not been achieved. These grafts, implanted as tubes however, may offer an alternative to untreated prostheses. The high incidence of late graft occlusion in our experiments requires further investigation prior to general application as a bifurcation graft.

References

1. Szilagyi DE, Elliott JP, Smith RF, Reddy DJ, McPharlin M (1986). A thirty-year survey of the reconstructive surgical treatment of aortoiliac disease. J Vasc Surg 3:422–436.
2. Wesolowski SA, Fries CC, Karlson KE, DeBakey ME, Sawyer PN (1961). Porosity: Primary determinant of ultimate fate of synthetic grafts. Surgery 50:91–96.
3. Yates SG, Barros D'Sa AA, Berger K, Fernandez LG, Wood SJ, Rittenhouse EA, Davis CC, Mansfield PB, Sauvage LR (1978). The preclotting of porous arterial prosthesis. Ann Surg 188:611–622.
4. Scott SM, Hoffman H, Gaddy LR, Sethi GK, Bridman AH (1985). A new woven double velour vascular prosthesis. J Cardiovasc Surg 26:175–181.
5. Haverich A, Maatz W, Stegmann T, Oelert H, Borst HG (1986). Experimental and clinical experiences with double-velour woven dacron prostheses. Thorac Cardiovasc Surgeon 34:52–53.
6. Agarwal KC, Edwards WD, Feldt RH, Danielson GK, Puga FJ, McGoon DW (1982). Pathogenesis of nonobstructive fibrous peel in right sided porcine valved extracardiac conduits. J Thorac Cardiovasc Surg 83:584–589.
7. Ben-Shachar G, Nicoloff DM, Edwards JE (1981). Separation of neointima from dacron graft causing obstruction. J Thorac Cardiovasc Surg 82:268–271.
8. Humphries AW, Hawk WA, Cuthbertson AM (1961). Arterial prosthesis of collagen-impregnated Dacron tulle. Surgery 50:947–954.
9. Bascom JU (1961). Gelatin sealing to prevent blood loss from knitted arterial grafts. Surgery 50:504–512.
10. Wesolowski SA, Fries CC, Domingo RT, Liebig WJ, Sawyer PN (1963). The compound prosthetic graft: a pathologic survey. Surgery 53:19–44.
11. Domurado D, Guidoin R, Marois M, Martin L, Gosselin C, Awad J (1978). Albuminated Dacron prostheses as improved blood vessel substitutes. J Bioeng 2:79–91.
12. Guidoin R, Marois Y, Rao TJ, Torche D, Marceau D, Tenney B, Duhamel RC, Lebowitz E, Walcott C (1988). An albumin-coated polyester arterial prosthesis made ready to anastomose: in vivo evaluation in dogs. Clinical Materials 3:119–131.
13. Jonas RA, Ziemer G, Schoen FJ, Britton L, Castaneda AR (1988). A new sealant for knitted Dacron prostheses: minimally cross-linked gelatin. J Vasc Surg 7:414–419.
14. Stegmann T, Haverich A, Borst HG (1986): Clinical experience with a new collagen-coated dacron double-velour prosthesis. Thorac Cardiovasc Surgeon 34:54–56.
15. Quinones-Baldrich WJ, Moore WS, Ziomek S, Chvapil M (1986). Development of a "leak-proof" knitted Dacron vascular prosthesis. J Vasc Surg 3:895–903.
16. Puchtler H, Waldrop FW, Valentine LS (1973). Polarization microscopic studies of connective tissue stained with Picro-Sirius Red F3BA. Beitr Path 150:174–187.
17. Gabbiani G, Schmid E, Winter S, Chaponnier C, de Chastonay C, Vanderkerckhove J, Weber K, Franke WW (1981). Vascular smooth muscle cells differ from other smooth muscle cells: Predominance of vimentin filaments and

specific alpha-type actin. Proc Natl Acad Sci USA 78:298–302.
18. Gown AM, Vogel AM (1984). Monoclonal antibodies to human intermediate filament proteins: II. Distribution of filament proteins in normal human tissues. Am J Pathol 114:309–321.
19. Chvapil M, Moore WS, Noishiki Y (1985). Biological testing of collagen-impregnated Dacron vascular prostheses. Angio Arch 9:7–12.
20. Hillman LS, Goodwin SL, Sherman WR (1975). Identification and measurement of plasticizer in neonatal tissues after umbilical catheters and blood products. N Engl J Med 292:381–386.
21. Barry YA, Labow RS, Keon WJ, Tocchi M, Rock G (1989). Perioperative exposure to plasticizers in patients undergoing cardiopulmonary bypass. J Thorac Cardiovasc Surg 97:900–905.
22. Chvapil M (1981). The fate of natural tissue prostheses. In: *Fundamental Aspects of Biocompatibility*. CRC Series in Biocompatibility, Volume 1. Willams DF (ed). CRC Press, Boca Raton, FL. pp. 87–104.
23. Chang C, Houck JC (1970). Demonstration of the chemotactic properties of collagen. Proc Soc Exp Biol Med 134:22–24.
24. Houck J, Chang C (1971). The chemotactic properties of the products of collanolysis. Proc Soc Exp Biol Medi 138:69–75.
25. Chvapil M, Owen JA, Clark DS (1977). Effect of collagen cross-linking on the rate of resorption of implanted collagen tubing in rabbits. J Biomed Mater Res 9:297–303.
26. Jonas RA, Mayer JE, Castaneda AR (1987). Unsatisfactory clinical experience with a collagen-sealed knitted dacron conduit. J Cardiac Surg 2:257–264.
27. Scott SM, Gaddy LR, Sahmel R, Hoffman H (1987). A collagen coated vascular prosthesis. J Cardiovasc Surg 28:498–504.
28. McGee GS, Shuman TA, Atkinson JB, Weaver FA, Edwards WH (1987). Experimental evaluation of a new albumin-impregnated knitted dacron prosthesis. Am Surg 53:695–701.
29. Ben-Slimane S, Guidoin R, Mourad W, Hebert J, King MW, Sigot-Luizard MF (1988). Polyester arterial grafts impregnated with cross-linked albumin: the rate of degradation of the coating in vivo. Eur Surg Res 20:12–17.
30. Zammit H, Hong-De W, Mathisen SR, Sauvage LR (1986). Influence on healing in the canine thoracic aorta of three substances used to close the interstices of macroporous dacron grafts. Am Surg 52:667–669.
31. LoGerfo FW, Quist WC, Nowak MD, Crawshaw HM, Haudenschild CC (1983). Downstream anastomotic hyperplasia. A mechanism of failure in dacron arterial grafts. Ann Surg 197:479–483.
32. Imperato AM, Baumann FG, Pearson J, Kim GE, Davidson T, Ibrahim I, Nathan I (1974). Electron microscopic studies of experimentally produced fibromuscular arterial lesions. Surg Gynecol Obstet 139:497–504.
33. Birinyi LK, Warner SJC, Salomon RN, Callow AD, Libby P (1989). Observations on human smooth muscle cell cultures from hyperplastic lesions of prosthetic bypass grafts: Production of a platelet-derived growth factor-like mitogen and expression of a gene for a platelet-derived growth factor receptor—a preliminary study. J Vasc Surg 10:157–165.
34. Shimokado K, Raines EW, Madtes DK, Barrett TB, Benditt EP, Ross RP (1985). A significant part of macrophage-derived growth factor consists of at least two forms of PDGF. Cell 43:277–286.

35
The Case for Mesocaval Shunts in the Treatment of Portal Hypertension

MARVIN L. GLIEDMAN and RONALD N. KALEYA

Reflecting on the use of portosystemic shunts in the treatment of variceal bleeding associated with portal hypertension, it has become obvious that reduction of portal venous pressure addresses only one facet of the overall problem in patients with severely diseased livers. With limited hepatic reserve, selection of a particular portal decompression procedure for an individual patient may have a significant effect on that patient's survival freedom from variceal hemorrhage, quality of life as determined by the absence of hepatic encephalopathy, and candidacy for subsequent liver transplantation.

While the pros and cons of the many shunt procedures continue to be argued in the medical and surgical literature, it is clear that none of the portal decompression procedures is a panacea for the treatment of portal hypertension. Looking back on the results for the interposition mesocaval shunt (IMCS) reveals it to be a valuable and important adjunct in the surgical armamentarium for decompression of the portal system, especially in view of progress in hepatic transplantation.

The major cause of death in the severely cirrhotic patient is bleeding from esophageal varices. Therefore, the efficacy of a particular shunt procedure must be measured primarily by its ability to ameliorate variceal hemorrhage with minimal operative morbidity and mortality. Additionally, both technical ease and the incidence and degree of postoperative encephalopathy consequent to diversion of portal blood from the liver are critical but secondary considerations. The overall long-term survival following portal decompression is another absolute endpoint. However, length and survival may not be as much a treatment-related factor as it is a marker of the progressive and severe nature of the underlying primary hepatic disease. A review of the results for the IMCS pertaining to these endpoints underscores its historical acceptance and clarifies its continued clinical applications.

Historical Perspectives

Surgical decompression of the portal venous system was introduced with the portacaval shunt in 1877 by Nikolai Eck.[1] Reynolds and Smithwick were the first to interpose a vein graft between the portal vein and vena cava in 1951. However, it was Theron who introduced the term "H graft" after using femoral vein autografts for portal decompression. Because portal vein thrombosis and small-caliber splenic veins prevented the use of these structures for portal decompression in children requiring shunt surgery, Marion[2] and Clatworthy et al.[3] in 1955 developed the direct mesocaval shunt. This J-type mesocaval shunt required anastomosis of the proximal end of the vena cava, divided just above the confluence of the iliac veins to the side of the superior mesenteric vein. This procedure never gained general acceptance owing to concerns regarding the sequelae of chronic lower-extremity venous stasis.

Subsequently, DeResenda-Alves in 1963 and Gliedman[4] in 1967 employed Teflon and Dacron conduits, respectively, for construction of the IMCS. The IMCS is functionally comparable with the side-to-side portacaval shunt and very effectively reduces portal venous hypertension.

In the early 1970s, several authors reported successful use of autologous vein, homologous vein, and heterologous vein as a conduit for the IMCS. Although Lord described the use of 18-mm woven Teflon prosthesis, Drapanas[5] popularized the interposition shunt after demonstrating its efficacy, safety, and technical simplicity.

Postoperative Mortality

The IMCS has been performed extensively since its introduction in 1967. As opposed to selective portal decompression procedures, recommended primarily in the elective setting and only in patients with reasonable hepatic reserve (Child's class A and B), the mesocaval shunt may be utilized as both an emergency and elective shunt as well as in patients with significant hepatic dysfunction. (Child's class C). Despite the unfavorable setting in which it has been used, mortality remains acceptable. When employed electively, the IMCS has a mortality of 4% to 9%, which is comparable with any of the other shunt procedures currently performed (Table 35.1). In several recent prospective studies comparing various shunting procedures,[6] the distal splenorenal shunt (DSRS) has an average mortality of 13%.

The mortality is, as expected, significantly higher in patients treated emergently with an IMCS, ranging from 25% to 100%. Those patients with Child's class A or B liver disease show a consistently better operative survival, especially when the shunt is performed electively. Child's class C

TABLE 35.1. Postoperative mortality of IMCS.

Source, yr.	No.	Elective mortality, %	Emergency mortality, %	Combined mortality, %
Paquet el al. (16) 1987	86	8		
Terpstra et al. (17) 1987	64			27
Sarr et al. (18) 1986	33			22
Rocko et al. (11) 1986	55	4	13	5
Mulcare et al. (12) 1984	49	4	44	22
Vogt et al. (19) 1983	25	6	100	
Jarvinen et al. (20) 1981	45	8	48	
Fletcher et al. (21) 1980	28	9	33	
Smith et al. (22) 1980	79	8	29	
Reichle et al. (23) 1979	28	7	29	

patients fare poorly in all studies, irrespective of the type of shunt or the setting in which the shunt is performed.

Though the IMCS has been used in the emergent situation, it is not recommended until the patient is stabilized. Our poor experience with portal decompression for emergency control of massively bleeding varices in the cirrhotic patient has compelled us to undertake extensive efforts to stop the bleeding nonoperatively, which then allows for an elective or urgent shunt procedure. An emergency transgastric ligation of varices is carried out only after peripheral intravenous vasopressin, endoscopic sclerotherapy, Sengstaken-Blakemore balloon tamponade, or transhepatic embolization of the coronary and short gastric veins fail to control variceal hemorrhage. When performed in the emergency setting, the IMCS, similar to other shunts, has a high mortality. However, IMCS done electively or urgently has been shown to be safe for the majority of patients.

Rebleeding and Graft Thrombosis

Although not all upper-gastrointestinal bleeding following portal decompression is a result of shunt dysfunction, the great majority of recurrent variceal hemorrhages reflect shunt thrombosis or stenosis. Rebleeding or shunt thrombosis must be considered a technical failure irrespective of postulated associated causes, e.g., progressive liver disease or, as in the case of the splenorenal shunt, renal vein hypertension.

In a large collected series reported by Mehigan et al.,[7] the side-to-side portacaval shunt had a 2% thrombosis rate but was associated with an unacceptably high incidence of postoperative encephalopathy. In this collected review, rate of thrombosis of the IMCS was 10% where 36% of the operations were done in Child's class C patients, and 23% were performed

in bleeding patients as emergency procedures. Despite the fact that 90% of the DSRSs reviewed were performed in low-risk patients and electively in 94% of cases, the occlusion rate was 10%. There was no difference in patency between the unselected group undergoing IMCS as compared with the highly selected group undergoing DSRS.

The early patency of mesocaval shunts has exceeded 90% in most series, with early failures attributed to long- or small-caliber grafts as well as operative errors. The thrombosis rate for the IMCS, however, clearly increases with time. Long-term follow-up of patients undergoing interposition grafts has shown that the average rate of thrombosis in several series has been between 5% and 25%, and approximately 5% to 15% of the patients rebled owing to graft occlusion with the IMCS. In five prospective studies[8] comparing DSRS with other shunt procedures, there was an overall 9.4% rate of rebleeding during the follow-up period, averaging approximately 2.5 years.

Not all graft closures necessarily result in rebleeding, nor are all closures the result of pathologic processes. In patients with patent shunts at the time of hospital discharge, we believe that some shunt closures are related to reduction in graft flow due to decreased portal pressure resulting from improved liver function associated with cessation of alcohol consumption or resolution of acute alcoholic hepatitis.

The rate of graft failure in the IMCS has been correlated with length and diameter of the graft. The shorter and wider grafts tend to remain patent longer; however, these grafts are associated with a higher incidence of encephalopathy. Recognition of this trade-off has made it necessary to tailor graft dimensions to the individual patient, taking into account degree of portal hypertension, size of the liver, and size of the superior mesenteric vein.

Diagnosis of graft thrombosis has been simplified enormously with the introduction of magnetic resonance imaging (MRI). MRI provides a noninvasive method to detect thrombosed or failing shunts. Owing to the configuration of the mesocaval graft, the thrombosed shunts often can be reopened using percutaneous methods, either by infusion of urokinase or balloon angioplasty. With this regimen of postoperative evaluation of graft patency and the use of percutaneous methods to salvage the thrombosed or failing shunt, late patency has been increased to approximately 95%.

Encephalopathy

Postoperative and late hepatic encephalopathy is a serious sequel to any portal decompression procedure. Though in recent years the tide of medical and surgical wisdom has swelled in favor of selective shunting procedures, data supporting this current are not overwhelming, particularly in

the alcoholic cirrhotic. There have been at least six prospective and randomized studies performed to evaluate this; three showed a statistically significant decrease in encephalopathy associated with the DSRS, and three others have not been able to demonstrate such a difference.[8]

The IMCS has been associated with hepatic encephalopathy in up to 77% of patients,[9] but on average, hepatic encephalopathy afflicts only 23%[7] of patients. Early in our experience with the interposition shunt, two patients developed severe encephalopathy, which prompted reoperation to tailor the conduit to increase portal pressure and thus divert less blood from the liver. The encephalopathy abated in both these patients. Since that time we have used narrower (14 mm) and longer grafts in older patients and rarely use a graft larger than 16 mm in diameter so as to minimize postoperative encephalopathy.

Additionally in recent studies, at least 50% of the postoperative encephalopathy clears with dietary intervention alone. Our postoperative dietary regimen has been to allow only fish and fowl protein sources, excluding all beef from the diet. With this diet, overt problems that hinder patients' routine activities have been rare. Difficulties with encephalopathy have been generally associated with continued alcohol consumption or ingestion of beef products.

In patients undergoing DSRS, the selective nature of the shunt deteriorates over time with the development of portal-to-splenic collaterals that result in an increased incidence of late encephalopathy. In fact, 98% of the patients studied at Emory developed such collaterals within a year of the original surgery.[10] These collaterals defeat selectivity of the DSRS, causing more hepatofugal blood flow and increased encephalopathy. The collateral beds involved are the transgastric vessels, the transpancreatic collaterals, and those in the transverse mesocolon. As a result of these findings, modifications of the original distal splenorenal shunt have been proposed to address this problem. While the splenic vein disconnection—the present modification—appears sound, it also significantly increases the procedure's technical demands.

Although the DSRS offers both theoretical and early clinical advantages with respect to hepatic encephalopathy, these effects tend to diminish with time, making the DSRS more nearly comparable with other shunt procedures in prevention of late hepatic encephalopathy.

Survival

The older retrospective series did show a decreased long-term survival for patients undergoing IMCS. However, the groups of patients were not comparable. The mesocaval shunt had been used in the worst situations and in those cases where selective shunts were relatively contraindicated. Survival of patients with portal hypertension attributed to alcoholic cirrhosis is the

same irrespective of shunt procedure performed[8] as shown in several prospective randomized trials.

Technical Considerations

The technical ease of the IMCS is well documented.[4,11,12] The IMCS utilizes large-caliber vessels that are easily accessible without significant retroperitoneal dissection for decompression of the portal system. Also, it provides a vent from the portal vein for hepatic decompression to counteract ascites formation.

The use of a prosthetic conduit eliminated the need to work in the liver hilum to expose sufficient vein for the portal caval shunt or the J-type mesocaval shunt. In addition, a large liver or obesity does not make the IMCS more difficult as is the case with other selective and nonselective shunts.

The DSRS, with its increasing modifications, has become a formidable undertaking. The vessels used for this shunt are extremely friable and require meticulous dissection. Injury or avulsion of one of the many pancreatic tributaries to the splenic vein can result in significant bleeding that is difficult to control. The present commitment to dissect and ligate all the minute pancreatic tributaries to the splenic vein makes this procedure increasingly tedious and difficult. Additionally, the distance separating the renal and splenic veins is variable, and this influences the extent of the retroperitoneal dissection.

The expertise to undertake the technically demanding DSRS safely and expeditiously is developed only in centers where large numbers of shunting procedures are performed by a relatively limited number of surgeons. The learning curve for selective decompression procedures is steep. In at least two prospective randomized studies, the mortality was considerably higher in the earlier part of the study than in the latter half. The rate of complications resulting from technical errors was also higher. Langer et al.[13] reported a 19% mortality for the DSRS group in the early half of their study. The mortality fell significantly in the second part of the trial, leading to an overall 13% mortality for the entire DSRS group, whereas there were no deaths in the portacaval group. Similarly, Rigau et al.[14] reported a 16% initial mortality, which fell to 6% in the second part of their study. The 39% incidence of encephalopathy reported in some series of DSRS was attributed, in part, to the steep learning curve associated with the DSRS.[8]

Despite theoretical and potential clinical advantages of the splenorenal shunt, the learning curve may be too great to accommodate the procedure into general and widespread practice. Contrariwise, the technical simplicity of the IMCS permits its use by the general and vascular surgeon, who is only infrequently confronted with the difficult problem of portosystemic decompression.

Future Implications

With the increasing availability of hepatic transplantation as well as the progressive nature of alcoholic cirrhosis, shunt procedures must be considered as an interim solution to the problem of portal hypertension and bleeding esophageal varices. Patients undergoing transplantation following an IMCS had the lowest complication rate, the lowest blood loss, and were technically the least difficult to dismantle at time of transplantation. The shunt can be used for venovenous bypass during donor hepatectomy and subsequently ligated at conclusion of the procedure. The DSRS, however, can exacerbate blood loss because splanchnic portal hypertension persists after division of collaterals during the procedure. The mesocaval shunt is considered the best alternative for patients who may become future candidates for liver transplantation.[15]

It is important to consider the hemodynamic and parenchymal problems of the cirrhotic as separate. Many cirrhotics have more than adequate parenchymal reserve for long life if the hemodynamic problem (portal venous hypertension due to obstruction) is solved by a decompressive procedure. Not all bleeding cirrhotics have need for new parenchyma and hepatocyte function.

Comment

Although many now believe that the DSRS should be the procedure of choice for portal decompression, there are many contraindications to that procedure, and the technical nuances of the DSRS continue to make it an increasingly formidable undertaking. The IMCS, on the other hand, has had more than two decades of clinical application with consistently good control of variceal bleeding, minimal mortality, and low incidence of encephalopathy, without the technical difficulties associated with other shunt procedures. By looking back at the results for this procedure, it is evident that the IMCS continues to be an effective option for the surgical treatment of portal hypertension and variceal bleeding.

References

1. Eck NV. On the question of ligature of the portal vein. Surg Gynecol Obstet 1953;96:375.
2. Marion P. Les obstructiones portales. Semin Hop Paris 1953;29:2781.
3. Clatworthy WH, Wall T, Watman R. A new type of portal to systemic venous shunt for portal decompression. Arch Surg 1955;71:588.
4 . Gliedman ML. The mesocaval shunt for portal hypertension Am J Gstroenterol 1971;56:323.

5. Drapanas T. Interposition mesocaval shunt for the treatment of portal hypertension. Ann Surg 1972;176:435.
6. Mitchell RL, Ignatius JA. Distal splenorenal shunt: Standard procedure for elective and emergency treatment of bleeding esophageal varices. Am J Surg 1988;156:169.
7. Mehigan DG, Zuiderma GD, Cameron JL. The incidence of shunt occlusion following portasystemic decompression. Surg Gynecol Obstet 1980;150:661.
8. Henderson JM. Variceal bleeding, Which shunt? Gastroenterology 1986; 91:1021.
9. McInnes IE, Gibson PR, Rosengarten DS, et al. Long term followup of patients following selective and nonselective portosystemic shunts. Aust NZ J Surg 1985;55:355.
10. Ruff RJ, Chuang VP, Alspaugh JP, et al. Percutaneous vascular intervention after surgical shunting for portal hypertension. Radiology 1987;164:469.
11. Rocko JM, Howard MM, Swan KG. Surgical management of bleeding esophageal varices. Am Surg 1986;52:81.
12. Mulcare RJ, Halleran D, Gardine R. Experience with 49 consecutive Dacron interposition mesocaval shunts. Am J Surg 1984;147:393.
13. Langer B, Taylor BR, Mackenzie DR, et al. Further report of a prospective randomized trial comparing the DSRS with end to side portacaval shunt. Gastroenterology 1985;88:424.
14. Rigau J, Teres J, Visa J, et al. Long term followup of 100 patients treated by a modified splenorenal shunt. Br J Surg 1986;73:708.
15. Brems JJ, Hiatt JR, Klein AS, et al. Effect of a prior portasystemic shunt on subsequent liver transplantation. Ann Surg 1989;209:51.
16. Paquet KJ, Kalk JF, Koussouns P. Prospective evaluation and long term results of mesocaval interposition shunts. Acta Chir Scand 1987;153:423.
17. Terpstra OT, Ausema B, Bruining HA, et al. Late results of mesocaval interposition shunting for bleeding esophageal varices. Br J Surg 1987;74:787.
18. Sarr MG, Herlong HF, Cameron JL. Long term patency of mesocaval C-shunts. Am J Surg 1986;151:98.
19. Vogt DP, Santoscoy T, Cooperman AM, et al. Surgical management of portal hypertension and esophageal varices. Am J Surg 1983;146:274.
20. Jarvinen H, Kallio H, Lempinen M. Interposition mesocaval shunts for bleeding esophageal varices in moderate and poor risk cirrhotics. Ann Chir Gynaecol 1981;70:45.
21. Fletcher M, Dawson J, Williams R. Long term followup of mesocaval shunting in portal hyptertension. Br J Surg 1981;68:485.
22. Smith RB, Warren WD, Salam AA, et al. Dacron interposition shunts for portal hypertension. Ann Surg 1980;192:9.
23. Reichle F, Fahmy W, Golsorkhi M. Prospective comparative clinical trial with distal splenorenal and mesocaval shunts. Am J Surg 1979;137:13.

36
Venous Insufficiency: Acute and Chronic

SESHADRI RAJU and PETER NEGLÈN

Acute Deep Venous Thrombosis

Optimal treatment for acute deep venous thrombosis is in a state of evolution. Conventional treatment is anticoagulation with heparin followed by chronic Coumadin administration. It has been realized for some time that this treatment is not ideal, as postthrombotic malsequelae affecting the lower limbs are quite common despite adequate anticoagulation. In addition, failure of anticoagulation to provide adequate control of thromboembolism occurs in a definite percentage of patients. Killewich and associates' recent longitudinal study of deep venous thrombosis, performed with the aid of a duplex scanner, revealed significant residual obstruction as well as reflux in the affected limbs.[1] Sporadic attempts at surgical thrombectomy in the past, prompted by disenchantment with anticoagulation therapy, had yielded results that were considered no better than those produced by anticoagulation treatment. Eklof and colleagues have more recently re-evaluated surgical thrombectomy, combining it with the creation of a temporary arterial-venous fistula to improve patency.[2] In a carefully controlled trial, this modality was shown to be superior to anticoagulation in achieving and maintaining iliac vein patency and functional integrity of the femoral/popliteal vein segment. Superiority of the surgical option was apparent at 6 months and was maintained long-term at five years.[3] The superior anatomic and functional parameters, noted with surgical thrombectomy and temporary AV fistula, translated to a reduced incidence of postthrombotic syndrome with symptom presentation and clinical manifestations in the affected patients (Figs. 36.1, 36.2, and 36.3).[4]

The recent advent of interventional radiology and widespread use of urokinase therapy, primarily in arterial thrombotic conditions, has prompted interest in its application in the venous system. While prompt dissolution of clot appears feasible, especially when the agent is administered directly to the thrombotic site via transvenously placed catheter, long-term anatomic and functional studies with this technique are awaited. A perusal of the literature, however, indicates that even "systemically"

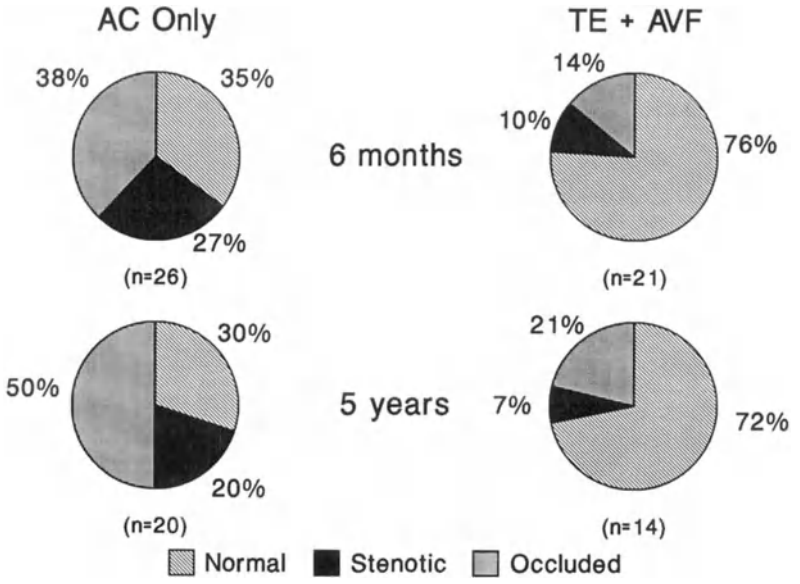


FIGURE 36.1. Comparison of short- and long-term iliac vein patency following treatment with anticoagulation (AC) or surgical thrombectomy with adjuvant AV fistula (TE + AVF). At six months, 65% of patients treated by anticoagulation had demonstrable pelvic collaterals compared to an incidence of only 24% following surgical thrombectomy with adjuvant AV fistula.

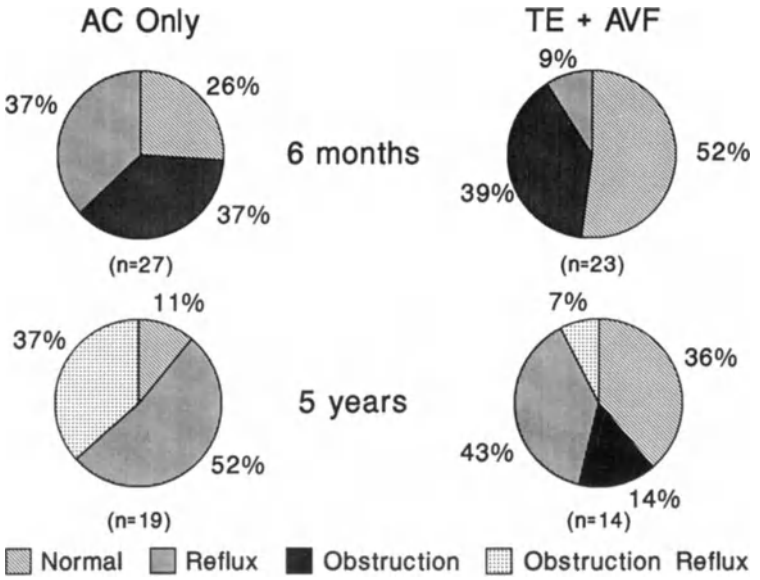


FIGURE 36.2. Status of the femoral popliteal segment following anticoagulation (AC) or surgical thrombectomy with adjuvant AV fistula (TE + AVF). Status of the femoral popliteal segment is superior with surgical thrombectomy, both at six months and at five years.

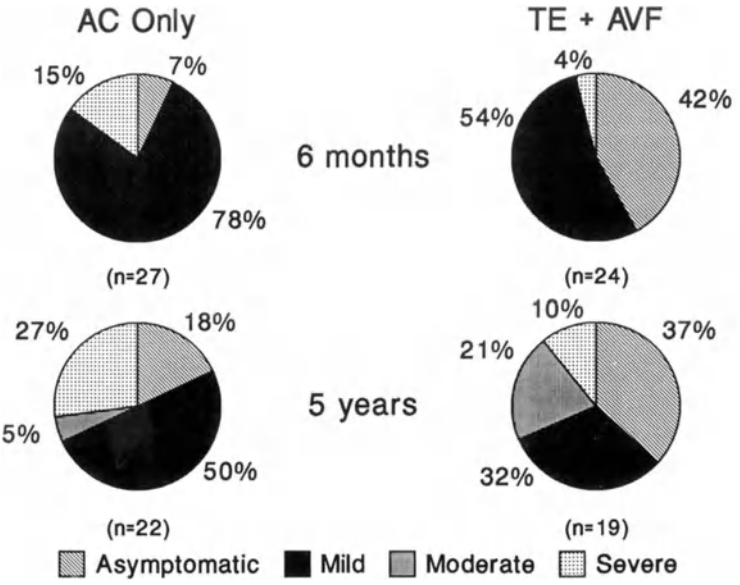


FIGURE 36.3. Symptom status of patients following anticoagulation therapy (AC) or surgical thrombectomy with adjuvant AV fistula (TE + AVF). Clinical results are superior with surgical thrombectomy.

administered lytic therapy with enzymes may be superior to conventional heparin treatment (Table 36.1). Lytic therapy and anticoagulation, in terms of clinical outcome as evaluated by different authors, are compared in Table 36.2. A prospective randomized study comparing catheter-administered lytic therapy and anticoagulation is clearly needed.

Chronic Venous Insufficiency

Chronic venous insufficiency used to be considered synonymous with post-phlebitic syndrome. It is better appreciated now that the term postphlebitic syndrome itself encompasses a variety of pathophysiological mechanisms

TABLE 36.1. Comparison of clot lysis with lytic therapy and with heparin (composite results from 14 published studies).

Degree of lysis	Lytic therapy (n = 322)	Heparin (n = 271)
None/worse	32%	82%
Partial	21%	12%
Significant/complete	47%	6%

TABLE 36.2. Published incidence of postphlebitic syndrome following anticoagulation (AC) or thrombolytic (SK) treatment (See Reference 4).

Author	Year	No. of patients	Follow-up (years)	Postphlebitic syndrome (%)	
				AC	SK
Common et al.	1976	27	0.7	92	60
Elliot et al.	1979	51	1.5	90	40
Arnesen et al.	1982	35	6.5	67	24
Kakkar et al.	1985	100	5.7	no diff	

consisting of venous reflux, obstruction, or a combination. The relative incidence of these different hemodynamic entities following deep venous thrombosis is unknown, as no controlled longitudinal functional studies have been conducted. It is now clearly recognized, however, that a type of venous reflux that is nonthrombotic in origin is a definite clinical entity. The latter condition, variously termed primary venous reflux or cryptogenic reflux, was described as early as half a century ago by Bauer.[5] The entity has become more well recognized following successful surgical correction of the valve deformity by Kistner.[6]

Chronic Venous Obstruction

Chronic venous obstruction has been poorly evaluated and managed mainly due to the lack of reliable diagnostic techniques. Plethysmography is cumbersome and seldom used routinely. Nicolaides has recently standardized air-plethysmography, which appears to be reliable. Venography is notoriously unreliable because it provides no hemodynamic information.[7] A dependable hemodynamic technique developed in our laboratory utilized arm/foot venous pressure differential at rest.[8] In the recumbent position, approximately 50% of patients with venous obstruction will have a significant elevation of foot venous pressure compared to arm pressure. The remainder have developed collaterals with a normal pressure differential. The latter group can be further evaluated by inducing a reactive hyperemia and noting the pressure changes in the venous system. An abnormal venous pressure increase with reactive hyperemia denotes inadequate collaterals. In advanced venous obstruction, venous pressure response may be paradoxically "normal," apparently because the classic reactive hyperemia response is either absent or delayed.[9] This observation suggests that the same mechanism may be responsible for venous claudication. Bypass procedure for venous obstruction is indicated only if Grade III or Grade IV obstruction is documented by the above mentioned hemodynamic tests. Bypass procedures performed in the absence of a significant

gradient based on radiographic appearance alone are not likely to remain patent for long. Bypass size should be adequate for the anatomical location. Iliac vein obstructions usually require a 16 mm prosthetic bypass utilizing stented PTFE grafts. Excellent patency rates can be expected with this material when bypass is performed for nonthrombotic etiology (i.e., tumor, trauma) and for transient hypercoagulable states, such as that in the patient who has used and discontinued birth control pills.[9] Long-term results are poor for both autogenous and prosthetic bypasses when the etiology of venous obstruction is that of a previous thrombotic process. A temporary AV fistula (up to six weeks) may be utilized in conjunction with the bypass to improve perioperative patency rate. A long-term fistula (up to six months) may be utilized to increase the size of the saphenous vein conduit with the Palma procedure.[8] Bacterial infection of such a fistula is a specific hazard. Distal venous hypertension with the AV fistula can be avoided by sizing the fistula with a 4 mm prosthetic sleeve, which also helps in percutaneous obliteration of the fistula later by interventional radiology (Fig. 36.4).

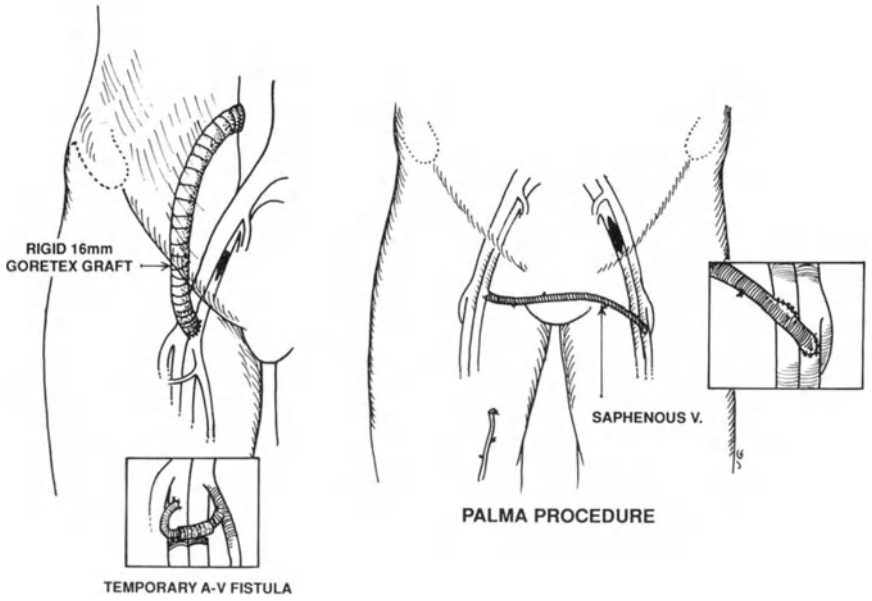


FIGURE 36.4. Bypass for venous obstruction utilizing a stented PTFE graft or the autogenous saphenous vein (Palma procedure). The inset beneath the prosthetic graft shows a branch of the saphenous vein being utilized for creation of an AV fistula. The vein is ensheathed in a 4 mm prosthetic graft for sizing and to help with later percutaneous balloon closure (see text). The inset beside the depiction of Palma procedure shows an end-to-side AV fistula between the saphenous vein and the femoral artery. The saphenous vein was anastomosed side-to-side to the femoral vein beside the fistula.

Venous Valvular Insufficiency

There is increasing realization that cryptogenic valve reflux of nonthrombotic etiology is responsible for a significant number of patients presenting with pure reflux. A deep venous thrombosis usually results in combined lesions involving both obstruction and reflux. While ambulatory venous hypertension has been accepted traditionally as the basis of stasis ulceration, approximately 25% of patients in our experience[10] are noted to have "normal" postexercise pressures. These patients appear to have abnormal Valsalva foot venous pressure, however. The latter technique is particularly useful in following patients with valve reconstruction surgery. This parameter is completely normalized, or nearly so, in patients successfully cured of stasis symptoms by surgery. Patients undergoing valve reconstruction surgery should undergo a complete workup, including pressure studies and ascending and descending venogram. Color-coded Duplex is proving invaluable in analyzing reflux in these patients preoperatively and following their postoperative course. Our preference is for valvuloplasty, as results with the axillary vein valve transfer are inferior probably due to

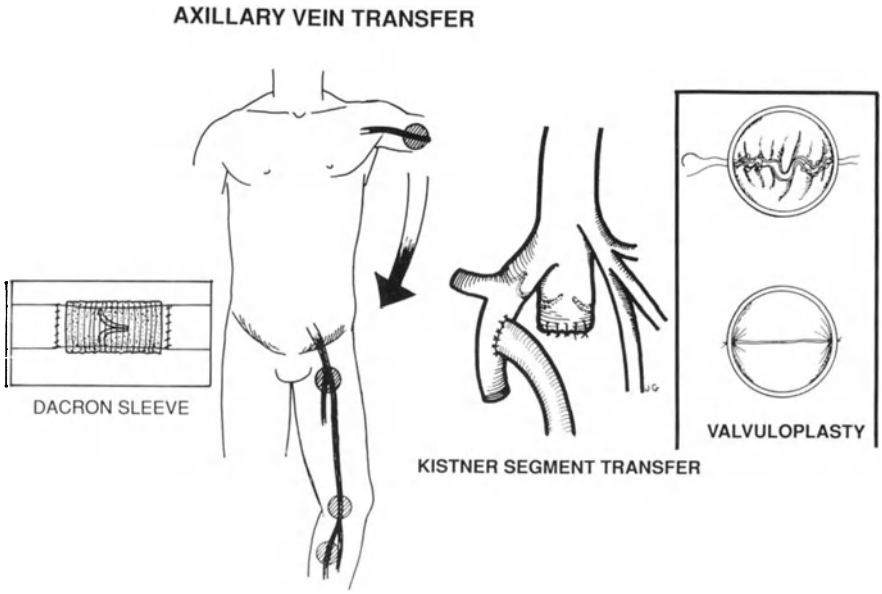


FIGURE 36.5. Surgical procedures for valve reflux. Axillary vein wall transfer with a Dacron sleeve around the transferred valve is shown on the left. The Kistner segment transfer where the superficial femoral vein is divided below an incompetent valve and reanastomosed to the profunda vein below a competent valve is shown in the middle. The internal valvuloplasty procedure, where the redundant valve cusps are tightened, is shown on the extreme right.

late dilatation.[11] Late dilatation can perhaps be prevented by applying a Dacron sleeve jacket around the transferred axillary valve (Fig. 36.5).[12] Internal valvuloplasty technique has now been replaced by an external technique, once again described by Kistner.[13] Since the technique is rapid, multiple valvuloplasties are possible. We routinely attempt repair of the superficial femoral profunda and posterior tibial valves in symptomatic limbs. In approximately 10% of patients, surgical manipulation of the vein results in venoconstriction, which restores competency to the previously leaky valve.[11] In these patients, a restraining Dacron jacket is applied around the vein to maintain competency without formal valve repair. With proper selection, over 80% of patients with stasis symptoms will be cured at two years with valve reconstruction surgery.

References

1. Killewich LA, Bedford GR, Beach KW, Strandness DE. Spontaneous lysis of deep venous thrombi: Rate and outcome. J Vasc Surg 9:89–97, 1989.
2. Plate G, Einarsson E, Ohlin P, Jensen R, Qvarfordt P, Eklöf B. Thrombectomy with temporary arteriovenous fistula in acute iliofemoral venous thrombosis. J Vasc Surg 1:867–876, 1984.
3. Plate G, Akesson H, Einarsson E, Ohlin P, Eklöf B. Long-term results of venous thrombectomy combined with a temporary arteriovenous fistula. Eur J Vasc Surg 4:483–489, 1990.
4. Neglèn P, Eklöf B. Thrombectomy with temporary arteriovenous fistula: the method of choice to treat acute iliofemoral venous thrombosis. In: *Current Critical Problems in Vascular Surgery-III*, (ed.) Frank J. Veith. St. Louis: Quality Medical Publishing, Inc. (in press).
5. Bauer G. The aetiology of leg ulcers and their treatment by resection of the popliteal vein. J Int Chir 8:937, 1948.
6. Kistner R. Surgical repair of the incompetent femoral vein valve. Arch Surg 110:1336, 1975.
7. Raju S. A pressure based technique for the detection of acute and chronic venous obstruction. Phlebology 3:207, 1988.
8. Raju S. New approaches to the diagnosis and treatment of venous obstruction. J Vasc Surg 4:42, 1986.
9. Raju S, Fredericks R. Venous obstruction: diagnosis and treatment. In: *Current Critical Problems in Vascular Surgery-III*, (ed.) Frank J. Veith. St. Louis: Quality Medical Publishing, Inc. (in press).
10. Raju S, Fredericks R. Hemodynamic basis of stasis ulceration—An hypothesis. J Vasc Surg 13:491, 1991.
11. Raju S, Fredericks R. Valve reconstruction procedures for non-obstructive venous insufficiency: Rationale, techniques, and results in 107 procedures with 2–8 year follow-up. J Vasc Surg 7:301, 1988.
12. Raju S. Venous insufficiency of the lower limb and stasis ulceration: Changing concepts and management. Ann Surg 197:688, 1983.
13. Kistner RL. Surgical technique of external venous valve repair. Straub Foundation Proc 55:15–16, 1990.

37
A New Surgical Technique for Venous Reconstruction: The Nonpenetrating Clip

YONG HUA ZHU and WOLFF M. KIRSCH*

Abstract

The surgical principles governing venous reconstruction remain uncertain in contrast to the standardized principles of arterial corrective surgery. The technical difficulties attending venous reconstructions, their potential for complicating thromboembolism, and poor surgical outcome are directly related to the biological response of veins to needle-and-suture. A new method for venous reconstruction, predicated on intimal nonpenetration and flanged eversion, exhibits technical and physiologic advantages over conventional suture.

Venous reconstruction with the nonpenetrating clip is equivalent to or superior to suture by trials in the surgical laboratory and clinic. The clip has been tested with the following venous reconstructions: end-to-end, end-to-side, arterial interpositional venous grafts, vascular access procedures, and free flap transfers. Long- and short-term patency rates, blood flow rate and characteristics of intimal repair, and mechanical properties of the flanged anastomoses (burst and tensile strength) have been determined. Details of this new surgical technique, the histological and physical properties of the flanged venous anastomoses, and clinical experiences are described.

Introduction

Despite vascular surgery's progressive advances, uncertainty still characterizes venous reconstructions. Procedures adequate for arterial anastomosis become difficult and ineffective when transposed to thin-walled veins. Thrombosis regularly complicates suturing of veins, a system marked by low pressure, sluggish flow, relative hypoxemia, and fragile intima. As a consequence, a variety of nonsuture techniques have been applied to veins. These include fixed rings and cuffs, staples, hollow tubes, biological adhe-

*To whom reprint requests should be addressed.

sives, and laser welding.[1–4] None so far have proven practical. Despite inherent flaws, suturing remains the standard to which any new method for venous reconstruction must be compared, even though there is disagreement regarding indications and optimal suturing technique.[5,6] Recognized inadequacies warrant continuing investigation for an improved surgical method for venous reconstruction.

Venous thromboses are related to vein intimal penetration, a problem that becomes intensified when suturing at the microsurgical level.* Small-caliber veins have a more severe inflammatory reaction, slower rates of endothelial and medial repair, and more extensive medial necrosis than small arteries in response to suturing.[7,8] Though needle and suture are clearly accepted in vascular surgery, their use for venous reconstruction sets the stage for a train of deleterious events, largely traceable to intimal damage. Thus, any improvement in venous surgical technique should address the following problems inherent to the use of conventional methods and avoid intimal damage.

1. *Intimal damage*: Ischemia of the entire venous wall segment occurs during the course of application of double approximator clamps. Ischemia leads to a significant slough of fibrinolytic, venous intima with exposure of a potentially thrombogenic subendothelial basement membrane.[7,8] This exposure will be tolerated unless blood flow stagnates or superimposed intimal damage (stretching, needle puncture) triggers the clotting cascade.
2. *Venous wall damage associated with adventitial stripping*: Adventitial stripping is recommended as a preliminary step to sutured anastomoses to prevent capture of thrombogenic material in the suture line.[9] Since perfusion of the venous wall occurs via the adventitia, this dissection promotes intimal sloughing, heightened sensitivity to further trauma, and thrombosis.
3. *A sutured venous anastomosis is never precise*: The joining of thin-walled venous tissue by passage of a suture, followed by tying a knot, produces inevitable wall overlap ("purse-stringing"), secondary medial necrosis, and an irregular anastomotic line (Fig. 37.1). Secondary blood flow turbulence leads to initiation of clot formation at the suture line, particularly after exposure of potentially thrombogenic subendothelial collagens and media matrix.
4. *A sutured venous anastomosis is not "blood-tight"*: Bleeding from needle puncture sites is accepted as an inevitable consequence of suturing. The surgeon counts on "natural" hemostatic mechanisms to stop bleeding. The clotting cascade does not conveniently shut down when leaks are plugged but continues to propagate intravascularly, embolize, and

*Microvascular surgery arbitrarily refers to procedures on arteries or veins below 2.5 mm O.D.

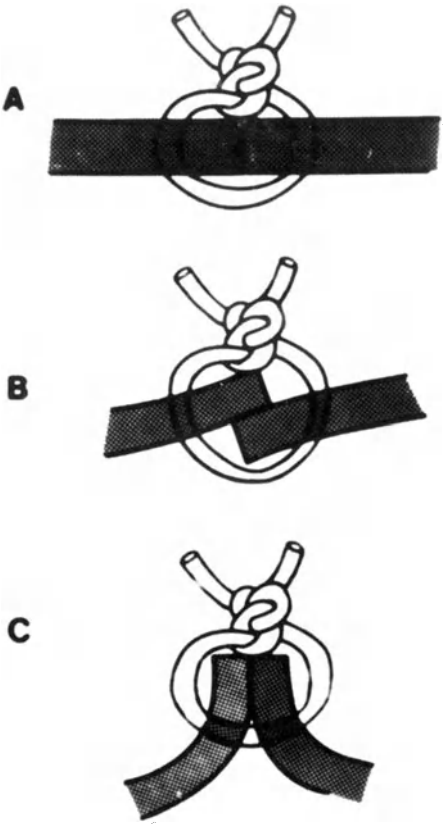


FIGURE 37.1. Models of tissue approximation by suture. **A** represents the idealized "butt" joint that is virtually impossible to attain, **B** is the type of apposition usually attained in practice, and **C** is optimal eversion and intimal approximation.

conceivably leads to total occlusion. Tamponading a bleeding venous anastomosis with manual pressure is potentially harmful, yet is recommended in contemporary vascular surgery texts.[10]

5. *The penetrating needle*: Passage of needle and suture inevitably damages the vascular intima (Figs. 37.2, 37.3) or may, in fact, damage the opposite wall. A commonly used microsurgical needle and suture (10-0 nylon, 22 micron suture on a 75 micron needle) produces a laceration larger than the suture and exposes traumatized subendothelium to blood.
6. *Needle and suture are a potent stimulus for intimal hyperplasia*: Passage of needle and placement of a permanent suture leads to a foreign body reaction. This response is more extensive in veins than in arteries and persists longer.[11] Proliferation of leukocytes, intimal migration of smooth muscle cells, and platelet aggregation result. The cellular reac-

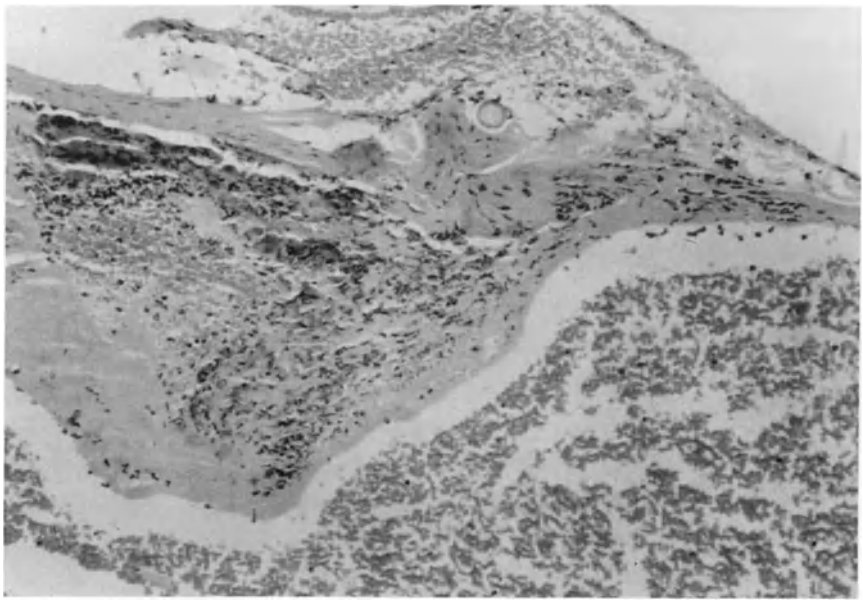


FIGURE 37.2. Typical appearance of microsutured (10-0 nylon) rat femoral vein anastomoses one week postoperative. Relative dimensions can be gauged by the 22-micron diameter 10-0 suture. Vein prepared by method of Acland and Trachtenberg.[17] Note intraluminal fibrin deposition at anastomotic site with interference of endothelialization.

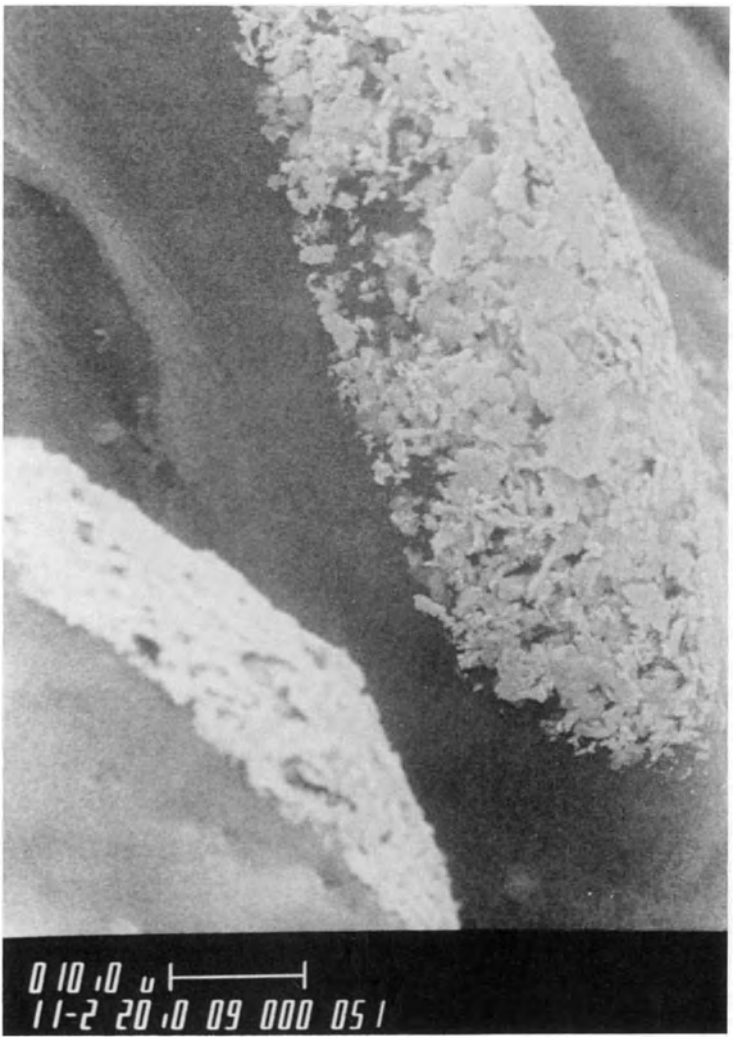


FIGURE 37.4. Scanning microscopic picture of a 10-0 sutured rat femoral artery anastomosis 60 days after surgery. Note myointimal hyperplasia at site of intraluminal sutures with platelet and fibrin aggregation. Reactive hillocks are not covered by a true endothelium. Dimensions on scale.

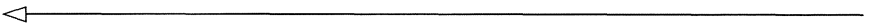


FIGURE 37.3. Scanning microscopic picture of the sutured luminal anastomotic surface of a rat femoral artery. Note the endothelial loss, overriding of tissue planes (as in Fig. 37.1B), bunching of the subendothelium, needle puncture hole, and platelet aggregation about needle puncture. Relative dimensions can be gauged by the 22-micron, 10-0 nylon suture diameter.

tion leads to intimal hyperplasia and platelet aggregation, a cause for late failure of vascular anastomosis or interpositional grafts (Fig. 37.4).

7. *Technical problems with small caliber veins*: Surgical precision is a crucial ingredient for venous repair. Placing sutures too tight narrows the anastomotic line—too loose and the line bleeds. There is little margin for error. These considerations plus the limits of human proprioception are important when repairing veins with wall thicknesses of 50 to 100 microns at the microscopic level.

The realization of the nonpenetrating clip stems from the need to improve the quality of microsurgical venous anastomoses after experiencing the frustrations and doubts that accompany conventional microsuture. A review of nonsuturing methods for microvenous repair did not turn up any reports of previous reconstructions with nonpenetrating microclips. At the macroscopic level, there were reports of successful arterial and venous anastomosis with "toothed" penetrating clips.[12,13] This study commenced at the microlevel to test the feasibility of microvenous and microarterial anastomoses with a nonpenetrating clip. Success at the microlevel has warranted further testing on larger caliber veins. The results of these studies form the basis of this paper.

Description of Clips, Applier, and Surgical Technique

Initial experiments were done with rat femoral veins and arteries. This paper will emphasize results of venous studies with comments on arterial work added for the sake of completeness. The adult rat femoral vein, distal to the lateral circumflex femoral tributaries, is 1.0 to 1.5 mm in outer diameter with a wall thickness of 50 to 100 microns. In order to fabricate a miniature, nonpenetrating microclip with dimensions capable of approximating this delicate tissue, computer-assisted wire corrosion technology was employed. This advance in precision engineering enabled mass production from pure silver of an "H-shaped" clip, 700 microns in length, 100 microns thick, configured like a clothespin. These first generation microclips were manipulated and applied with a fine-bore, suction-fitted microneedle holder using the short arms of the "H" for loading and stabilization.[14] From the outset, these trials proved the worthiness of a microflanged anastomosis for both rat femoral veins and arteries. Short- and long-term patency of microclipped venous and arterial anastomoses (0–7 days, 7–240 days), procedure duration, light scanning and transmission microscopic appearance of the anastomoses (Fig. 37.5), and incidence of false aneurysm at the anastomotic site compared favorably to results obtainable by microsuture. The elastomeric flange, formed by the coaption of intimal venous surfaces, healed with remarkable rapidity and unusual tensile strength. Flange joint strength is a function of coapted surface area

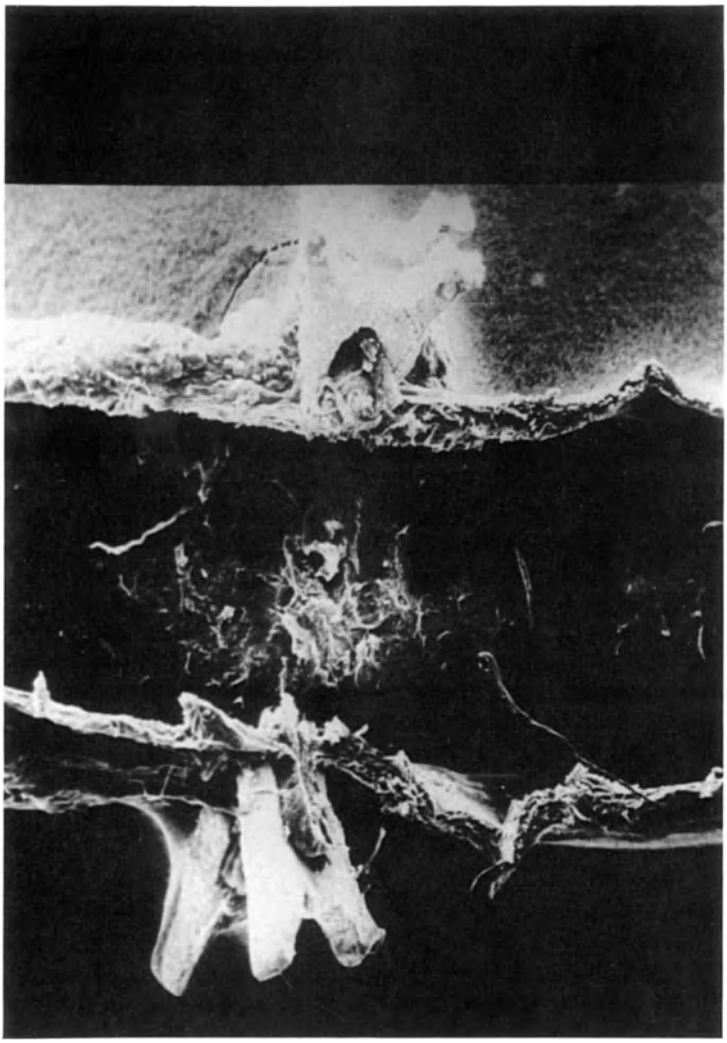


FIGURE 37.5. Scanning microscopic picture of 1.5 mm diameter rat femoral vein anastomosed with the original "H-shaped" microclips 24 hours after anastomosis. Flanged coaption of adventitia is evident. The anastomotic cleft cannot be visualized nor has fibrin accumulated at the site of anastomosis. Relative dimensions and diameter of vein wall thickness can be gauged by the size of 700-micron-long clip (100 microns thick).

and pressurization. A flange joint is different from a stapled or cuffed-ring anastomosis. Staples penetrate and do not form a flanged joint since the everted walls are not coapted. Cuffed rings do not allow a change in anas-

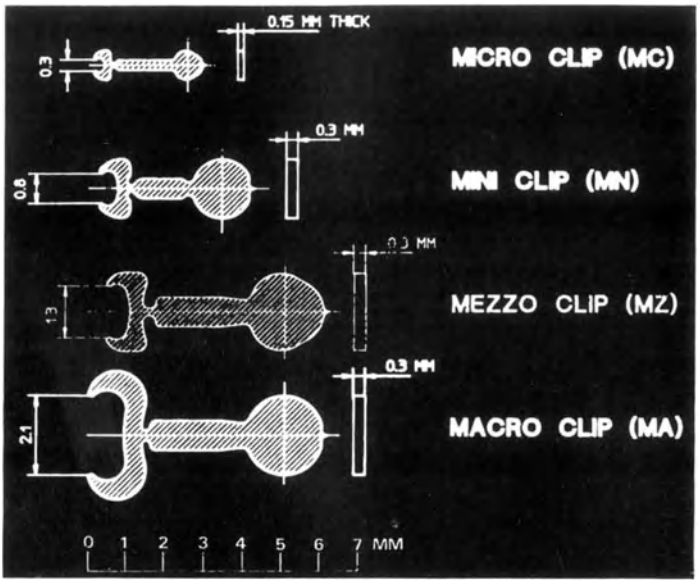


FIGURE 37.6. Dimensions and configurations of the micro, mini, mezzo, and macroclips. The theoretical background for jaw and tang design are given in the text.

tomotic luminal diameter with changes of intraluminal pressures and fail because venous walls are unable to collapse. Stagnating flow contributes to thrombosis.[15] Interrupted clips permit venous collapse and preserve the adventitia. The flange is a minimally thrombogenic anastomotic conformation. Eversion and approximation of venous walls for clip application are readily accomplished with special "everting forceps," jeweler's forceps with blunt tips angled at 45°.

Encouraged by the advantages of the nonpenetrating flanged clip anastomosis at the microvascular level (vein and artery), efforts were directed to the design and fabrication of a less ambiguous clip and applier. A clip applier was needed in which the clip could be conveniently loaded, reproducibly actuated, and readily removed in the event of misapplication.

These needs were fulfilled by design of a nonpenetrating clip having spaced, arcuate arms but mounted on a frangible tang (Fig. 37.6). The tang (measured in mm) enables unambiguous clip loading into a conforming slot, as well as being a component of the clip actuation mechanism. Manual squeezing of the applier by a pincer movement between thumb and forefinger pulls the tang, forcing the shoulders of the clip against stationary jaws. A defined angulation of these jaws in relation to the clip shoulders closes the tips of the clip to a "zero gap" without springback. The conformation of the tang neck on the clip is such that its breaking strength is greater than the tension required to deform by bending the clip bridge.

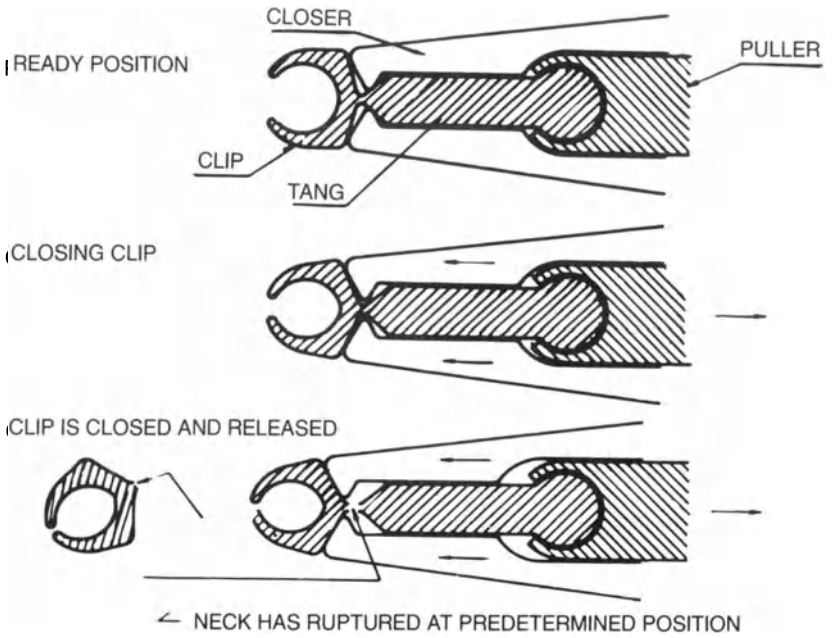


FIGURE 37.7. Mechanism of clip actuation. Pincer movement on forward aspect of applier causes puller to retract clip by the frangible tang. The tang ruptures at preset tensile force after the clip is closed and applied. Tang is then removed by pressure on midpoint of applier and the slot exposed for reloading manually. Microclips are loaded with microscopic control; mini, mezzo and macroclips with naked eye.

The jaw face and clip shoulder relationship is designed to place the points of initial engagement remote from the tang neck. Application of tension (T) to the tang defines a bending moment ($M = TX/2$) at the center of the bridge (X equals the distance from tang axis to either contact point). Bridge deformation and degree of clip closure are determined by clip/applier geometry and are independent of the surgeon's activating force. The tang snaps off after the clip is applied and is easily removed from the applier slot in order to reload (Fig. 37.7).

Four different sized clips are suitable for all laboratory and clinical venous anastomoses so far encountered (Fig. 37.6). The microclip, for example, can be used for veins up to 2.5 mm O.D. with wall thickness <150 microns. The radius of curvature of the tip of each arm of the microclip is 12 microns. Though changes in jaw face geometry or clip shoulder shape can produce any desired change in the degree of clip deformation and closure, all of the venous and arterial surgical studies have been done with clips set to a "zero gap" (jaw faces engaged at an obtuse angle of 150°). Appliers and clips have been calibrated by microscopic control to verify the

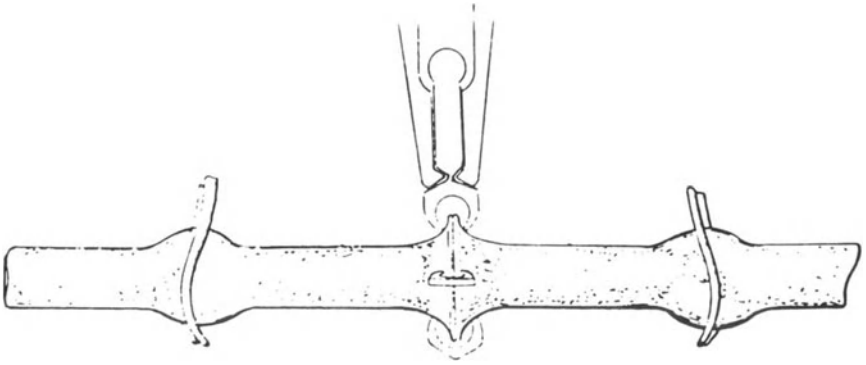


FIGURE 37.8. Drawing taken from U.S. Patent 4,733,664 demonstrating the applier in position to actuate a clip about the everted vessel edges. The clip is detached from the applier by using pressure against the applier sides with thumb and forefinger. This manipulation causes the clip to automatically close about everted tissue and to detatch from the applier, independent of the digital force applied.

gap of the clip points. Clips are made from plastically deformable pure silver, a metal with minimal springback. Loading mode of the applier is set by manual pressure on the midpoint of the handle to expose the slot for clip loading. Clip actuation occurs by a selective pincer movement of thumb and index finger on the forward portion of the handle. This differentiated finger manipulation is highly desirable when working at the microlevel. The clip, applier, and method have been granted United States patents (U.S. Patent Numbers 4,586,503 and 4,733,664) (Fig. 37.8).

Reproducible eversion of vein walls in preparation for clip application is a rapidly learned skill even at the microlevel. In the event of a malaligned clip, removal is accomplished by squeezing the bridge between the clip arms with a fine-pointed needle holder. The intima is not damaged by repeated clip application and removal. We have repeatedly performed a successful microvenous or microarterial anastomosis with clips, then removed the clips immediately, reopened the vessel, and reapplied the clips with restoration of flow. This was repeated eight times in succession with restoration of arterial flow of a rat femoral artery in a demonstration for the Surgery Department of the University of Colorado School of Medicine (courtesy of Dr. Alden Harken, July 1988).

Eight to 10 microclips are required for a blood-tight 1.5 mm rat femoral vein anastomosis, equivalent to the number of interrupted microsutures. A stent is not needed, since clips are nonpenetrating and there is less risk of catching the opposite wall as with microneedle and suture. Clip penetration of the venous intima has never been observed, though misuse of the everting forceps has the potential for this damage. The clips are adaptable

for end-to-side, vessel caliber mismatch, as well as vascular prosthesis anastomosis.

General Experimental Comments

Venous anastomoses performed with nonpenetrating clips have remarkable biological properties. Anastomoses are routinely blood-tight at the outset, have surprising physical strength, and result in prompt reendothelialization and venous wall remodeling. Scanning microscopic studies of microclipped rat femoral vein anastomoses demonstrate restoration of the intima within one week (Fig. 37.9). Dr. Nicholas Wilson (laboratory of Dr. Norman Browse, St. Thomas's and Guy's Hospitals, London) has confirmed these observations in the sectioned canine saphenous vein anastomosed with mezzoclips. Dr. Wilson has reported venous anastomotic healing by both direct venoscopy and histologic preparations within one week using the clips.[16]

The rate of venous endothelial repair is significantly slower with a sutured anastomosis. The formation of a flanged anastomosis from a sectioned vein after application of double occluding approximator clamps opposes two ischemic, intimally depleted surfaces. These subendothelial surfaces bond with surprising strength in a short period of time. Microclips can be removed 30 minutes after the formation of an anastomosis with preservation of anastomotic integrity from either microclipped femoral arteries or veins. This experiment was extended by performing a rat femoral artery-to-vein, end-to-side anastomosis with microclips, waiting 30 minutes, then removing the clips. The anastomosis remained intact and patent, the wound was closed and the animal allowed full activity. One months later, the groin was reopened and the arteriovenous fistula found patent without evidence of anastomotic aneurysm.

Another study compared the extent of anastomotic site fibrin deposition in rat femoral veins anastomosed with either 10-0 nylon microsuture or microclip. Each of 20 rats served as their own control, with one femoral vein anastomosed by microclip and the other by microsuture. At one hour, 4 hours, one day, 3 days, and 1 week, animals were anesthetized, the groins reopened and the femoral veins exposed. Patency testing ("strip and refill") was performed 2.0 cm proximal from the anastomotic site and the veins harvested and processed by the method of Acland and Trachtenberg.[17] The blood-filled specimens were immediately formalin fixed. After one week of fixation, clips were removed from the anastomoses in order to section the blood-filled segments longitudinally. Anastomotic site fibrin deposition was scored as none, present, or totally clotted. Microclipped anastomoses were virtually fibrin free, whereas all of the sutured veins had significant anastomotic fibrin deposition (Fig. 37.2). The microclipped venous anastomoses had in fact endothelialized the anastomotic site (Fig. 37.10).

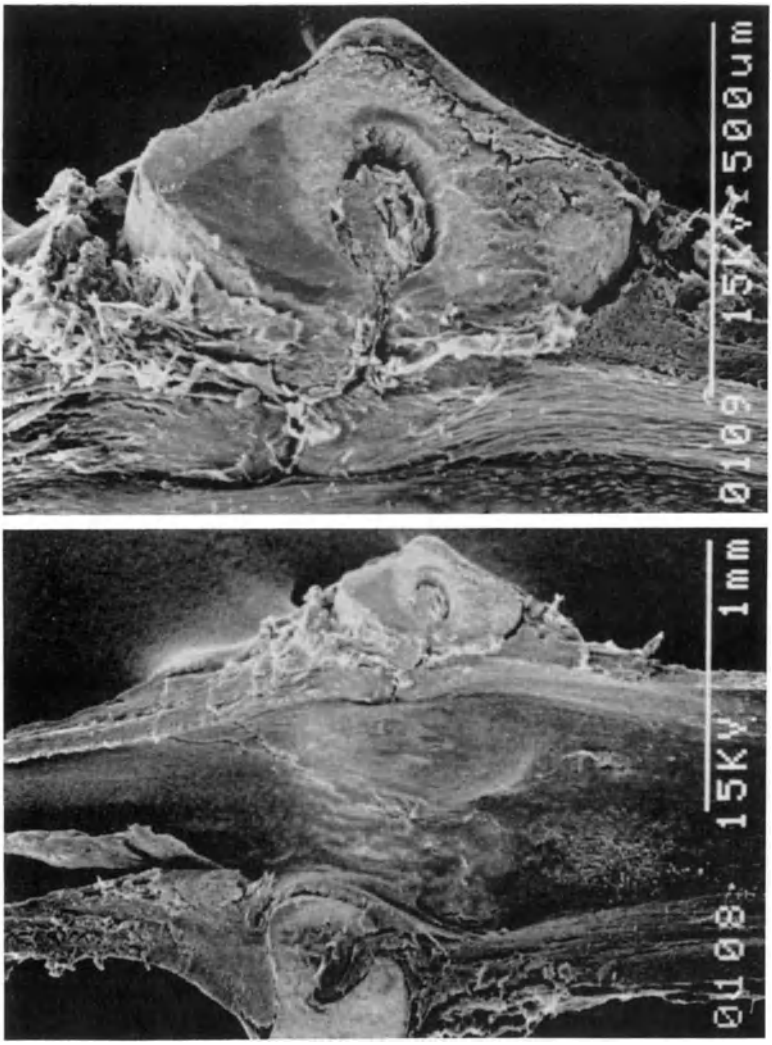


FIGURE 37.9. Scanning microscopy of the rat femoral vein, clipped anastomotic site demonstrating complete endothelialization at one week. Note the smooth and highly reproducible point of rupture of tang from the clip, the "zero gap" enclosing adventitia and media, and endothelialization with remodeling of the venous wall. This series of pictures "zooms" in on the anastomotic line with dimensions on scale.

Encouraged by these results, an expanded series of fully controlled animal studies has been performed in accordance with "Good Laboratory Practice" standards. These are described in detail in the subsequent sections.

FIGURE 37.10. Scanning microscopic picture of microclipped rat femoral vein (1 mm O.D.) one week after anastomosis. Note smooth impression of closed clips on the intima with complete endothelialization of the anastomotic line.

Experimental Studies

All animals received humane care in compliance with the "Principles of Laboratory Animal Care," formulated by the National Society for Medical Research, and the "Guide for the Care and Use of Laboratory Animals," prepared by the National Academy of Sciences and published by the National Institutes of Health (NIH publication No. 85-23, revised 1985).

All animal surgeries were done by three surgeons, Dr. Yong Zhu, Dr. Wolff M. Kirsch and Dr. David Stoloff. These studies were monitored and regularly audited by an independent quality assurance unit. All experimental data was accurately recorded and verified, and the raw data, documentation, protocols, specimens, and final reports are available. Five of the six studies were performed with microclips, one with mezzoclips. All anesthetics were standardized—Innovar i.p. for rats and acepromazine maleate/ketamine HCl for rabbits. Sterile conditions and the operating microscope were used.

Study 1: Comparison of Suture and Clip for Microvascular Anastomoses

The purpose of this study was to compare microsuture to microclip (rat femoral vein and artery) with respect to the following parameters: short- and long-term patency rates, procedure duration, light, scanning, and transmission microscopic appearance of the anastomoses, and incidence of anastomotic aneurysm.

Methods and Materials

Thirty-eight 400 gm Sprague-Dawley rats had surgical exposure of both femoral veins. Femoral veins were isolated immediately below the lateral circumflex vessels (average diameter 1.5 mm), placed between double approximator clamps and sharply sectioned. One side was anastomosed with microsuture (10-0 nylon, 10–12 sutures/anastomosis) and the other with microclip (10–12 clips/anastomosis). The microclips used for this experiment were the original "H-shaped" devices and were applied with the suction-fitted needle holder. All of the remaining studies (see below, 2–6) were performed with the tanged clip and cam-actuated applier. Wounds were then closed and reopened at designated intervals up to 60 days. The same procedure was performed on isolated femoral arteries (38 animals).

Patency and Histological Analysis

One week postoperative, six animals (femoral vein anastomoses study) were anesthetized, their groin wounds reopened, and the anastomoses examined. One sutured and one clipped venous anastomosis had thrombosed by "strip and refill" testing. veins were then harvested according to the method of Acland and Trachtenberg and prepared for histological sectioning.[17] Microclips were removed from the specimens prior to sectioning. This technique enables longitudinal examination of the vessel with examination of the anastomotic site. The remainder of the veins were harvested between seven and 60 days. Some specimens were fixed for scanning and electron microscopy by the methods of Thurston et al.[18]

TABLE 37.1. Patency rate and false aneurysm in sutured and clipped rat femoral veins.*

	Sutures N = 36	Clips N = 37
0–7 days	84%	84%
7–60 days	75%	92%
False aneurysms	0%	0%

*Previously published. Reprinted with permission from Zhu YH, Kirsch WM, Cushman R et al. Comparison of suture and clip for microvascular anastomoses. Surg Forum 1985;36:492–495.

RESULTS

Femoral vein anastomoses by microsuture averaged 30 minutes in duration, microclipping averaged eight minutes. Patency rates are given in Table 37.1. Histological studies of clipped venous anastomoses demonstrated endothelialization within one week, minimal or no fibrin about the anastomotic site, medial necrosis at the site of clip application, but remodeling of the venous wall by 60 days. In contrast, sutured femoral veins at one week had marked fibrin deposition about the intraluminal suture, intense foreign body reaction and necrosis in the media, with no evidence of reendothelialization. Scanning and transmission electron microscopy demonstrated endothelialization of the anastomotic cleft of the clipped anastomoses within 24 hours after surgery (Figs. 37.11, 37.12). In contrast, the microsuture demonstrated tearing and bunching of the intima as well as intimal hyperplasia and platelet aggregation about the suture (Fig. 37.3).

An initial study demonstrated a significant number of false aneurysms in the clipped arterial group (47%) in comparison to the microsutured group (25%). Aneurysm formation was associated with an intense foreign body reaction to the clips. This reaction was traced to contaminating Cu^{++}, Zn^{++}, and hydrocarbons on the silver clips from the brass wire electrocorrosion manufacturing process. As a result of this study, cleaning of clips prior to sterilization was revised. Clips were subsequently cleaned in stepwise fashion with phosphoric acid, nitric acid, and isopropyl alcohol to eliminate contaminating brass and oils. Subsequent to this cleaning procedure, no further anastomotic aneurysms were detected in clipped arteries.

CONCLUSIONS

Microclips are technically superior to microsuture for venous anastomoses despite the cumbersome suction-fitted applier. Anastomotic arterial aneurysms at the microlevel associated with a foreign body reaction secondary to brass and oil clip contamination. These studies resulted in development of a new applier and clip cleaning method that eliminated arterial anastomotic aneurysms.

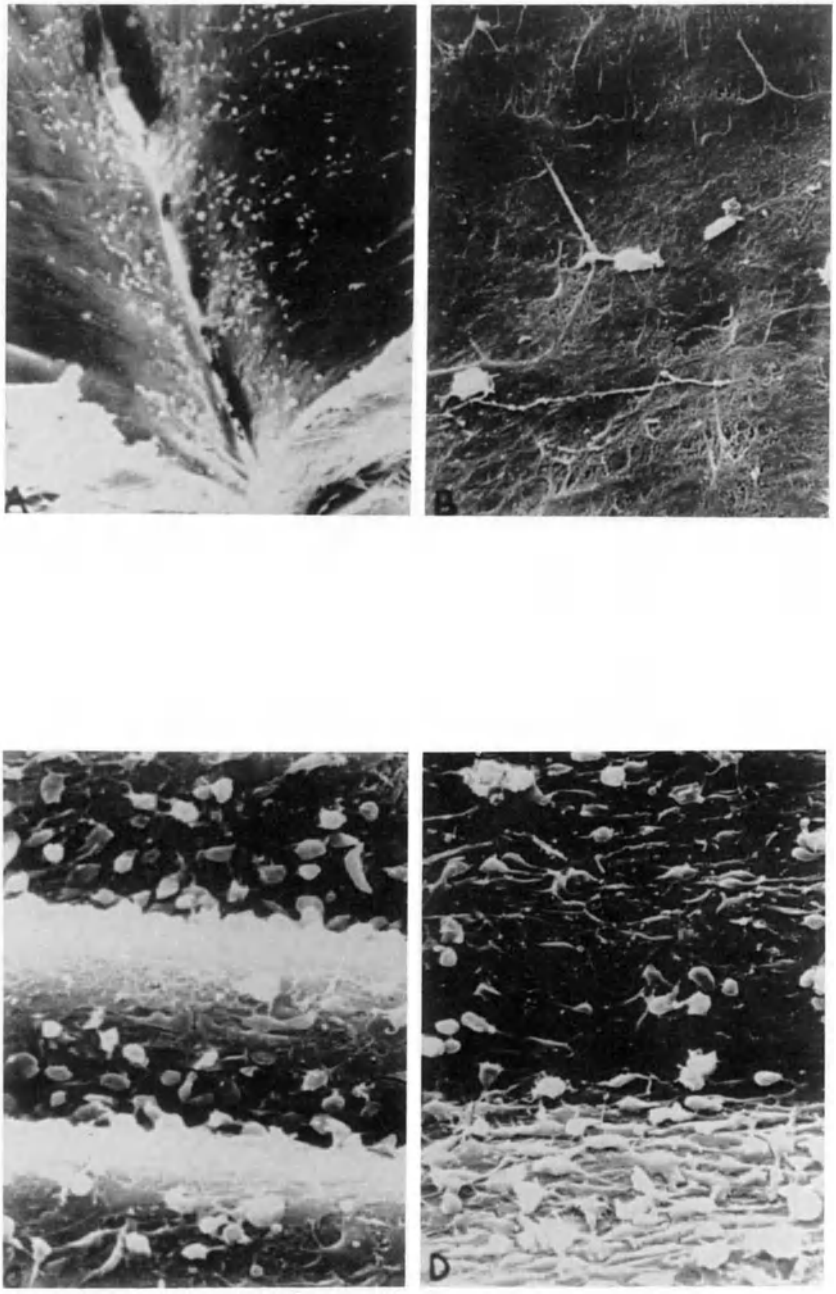


FIGURE 37.11. Scanning microscopic topography of rat femoral vein, clipped anastomotic cleft 24 hours after surgery. Note the selective endothelialization of the anastomotic cleft region. (**B**) 1 mm downstream from the anastomotic cleft with exposed subendothelium and sparse endothelialization. (**C** and **D**) Two different scanning microscopic views of the anastomotic cleft showing dense endothelial cell appearance at 24 hours.

FIGURE 37.12. Transmission electron microscopic montage of the anastomotic cleft of a clipped rat femoral vein demonstrating endothelial cell bridging of both subendothelial surfaces (24 hours postoperative). This cell-mediated adhesion may be responsible in part for the unusual early strength of the flanged anastomoses after clips are removed.

TABLE 37.2. Study 2: Number of anastomoses performed for each evaluation period.

Evaluation parameter	Postoperative period (days)	Arterial and venous paired anastomoses
Patency	7,14	10
	28	4
	70	8
Histology	7,14,28	4
	70	8

Study 2: Comparison of the Patency and Histology of Anastomosed Rabbit Femoral Veins

The purpose of this study was to compare patency and histology of paired "leak free" microclipped rabbit femoral vein anastomoses. All clips were cleaned by a process incorporating successive washes with phosphoric acid, nitric acid, and isopropyl alcohol to remove traces of Cu^{++}, Zn^{++}, and hydrocarbons. The tanged clip and camactuated applier were used in this and all subsequent studies. Thirty-two femoral vein anastomoses were performed on a total of 36 healthy New Zealand white rabbits (1.8 to 2.8 kg, mean weight 2.2 kg). Evaluations (patency, light microscopy) were performed at 7, 14, 28, and 70 days following surgery. The number of anastomoses for each evaluation period is given in Table 37.2.

SURGICAL PROCEDURE

Following standard anesthesia and surgical preparation, the femoral vein was isolated below the lateral circumflex tributaries and a double approximator clamp applied. Background material was placed beneath the vein to improve visualization, the vein transected, and the lumen irrigated with heparinized physiologic saline. The edges of the vein on the anterior surface were everted to achieve intima-to-intima contact, and microclips were placed at appropriate intervals. The approximator clamp was rotated after clipping the anterior wall and the procedure repeated for the posterior wall. The approximator clamp was then removed and the anastomoses examined. Additional microclips were placed if needed to secure a "blood tight" anastomoses. Only technically correct anastomoses were included in the study and the access incision was closed. The contralateral femoral vein was then exposed and anastomosed in the same manner. An analogous study was performed on paired rabbit femoral arteries with microclips cleaned by the method found to eliminate anastomotic aneurysms. A summary of vessel measurements and microclips applied per anastomosis is given in Table 37.3.

TABLE 37.3. Study 2: Summary of vessel measurements and microclips applied per anastomoses (rabbit femoral artery and vein anastomoses, number of animals in text).

	Arteries		Veins	
	Range (mm)	Mean (mm)	Range (mm)	Mean (mm)
Outside vessel diameter	0.8–1.80	1.22	1.2–2.0	1.6
Single vessel wall thickness	0.17–0.35	0.22	0.04–0.10	0.06
Number of clips applied per anastomosis	10–22	16	12–23	15

GROSS EXAMINATION AND PATENCY EVALUATION

At designated postsurgical time periods, rabbits were again anesthetized and the previously anastomosed vessels exposed. Anastomotic sites were grossly examined for evidence of aneurysm, fibrosis, or hemorrhage. Patency was determined by the "empty and refill" test, and if necessary, by incising the vein upstream (artery, downstream) from the anastomotic site and observing flow. Patency tests were performed at least 2.0 cm from the anastomoses to avoid damage to the anastomotic site.

HISTOLOGIC EVALUATION

Following the patency evaluation, a strip of plastic was placed beneath the anastomosed vein and "Ligaclips" (Ethicon) applied 2.0 cm on both sides of the anastomosis, attaching the vessel to the plastic strip. Stab incisions were made in the isolated segment adjacent to both Ligaclips. A cannula was inserted into the vein and flushing accomplished in the direction of blood flow with lactated Ringer's Solution, then with a combination fixative of 4% commercial formaldehyde (10% formalin) and 1% glutaraldehyde in a Millonig phosphate buffer. Each rabbit was then killed with an IV or intracardiac injection of barbiturate.

Vessels (arteries and veins), with microclips in place, were embedded longitudinally in glycol-methacrylate and stained with H. and E. Pontamine turquoise. An elastin stain was also used. Embedding in glycol-methacrylate allows silver clips to be sectioned with a glass knife. Microscopic evaluation was then carried out to determine the presence or absence of pseudoaneurysms, thrombosis, vessel wall inflammation, and appearance of the anastomotic site.

Results: Vessel Patency and Gross Appearance

A patency rate of 100% was achieved; 32 arteries and 32 veins were patent at the end of the designated survival intervals.

Histologic Observations

None of the clipped arteries or veins had any significant fibrin or platelets at the anastomotic line, nor were any aneurysms noted. All of the anastomoses were considered histologically acceptable. The jaws of the microclip compressed adventitia and media of the vessels together causing focal medial necrosis and elastic degeneration. Intimal regeneration was extensive by day 7 in veins and complete by day 28 in both arteries and veins. None of the clips were in contact with the bloodstream. There was a zone of metallic granules 5 to 30 microns thick about each microclip but very little inflammatory reaction. Most of the vascular damage noted histologically (medial atrophy, elastica damage, focal adventitial and medial mineralization, and endothelial sloughing) occurred well away from the microclips. This damage was attributed to the approximator clamps and/or adventitial stripping performed while isolating the vessels. Similar changes have been reported in studies of microvascular anastomoses using suture and other devices.[11,17]

Conclusions

Thirty-two rabbit femoral veins and 32 arteries were patent at the conclusion of a designated survival interval (up to 70 days) resulting in a 100% patency rate. Tissue reaction to the clips was minimal and intimal resurfacing of veins was noted at the anastomotic line within one week. Vascular damage noted histologically was at a distance from the venous anastomoses and attributed to approximator clamps and/or adventitial stripping.

Study 3: Tensiometric and Patency Evaluation of Anastomosed Rabbit Veins and Arteries

The purpose of this study was to compare breaking strength and elongation-to-break of rabbit femoral arteries and veins anastomosed with microclips against that of paired unoperated vessels at 0, 7, and 14 days postoperatively.

Methods and Materials

A total of 21 healthy female New Zealand white rabbits, weighing 2.1 to 2.6 kg, were used in this study. Venous anastomoses in three animals were excluded because of vessel trauma. One arterial anastomosis was excluded because of arterial wall penetration with eversion forceps. Additional rabbits were operated on to replace these animals.

Results: Intraoperative Observation

On one occasion the applier misfired and failed to break the tang during microclip placement.

Postoperative Observations

Fourteen of the 15 rabbits recovered uneventfully from surgery, but one animal developed a small skin dehiscence over an anastomotic site. The dehiscence healed spontaneously and all anastomosed vessels were patent at the end of the designated survival period with no evidence of hemorrhage or aneurysm.

Tensiometric Evaluation

Each rabbit was killed and Ligaclips applied to each vessel no less than 3.5 cm on either side of the anastomosis. Comparable locations were used on the contralateral unoperated femoral artery and vein. Both anastomosed and normal vessel segments were excised, washed in lactated Ringer's solution, and prepared for tensiometry. Ends of the vessel were bonded to a folded piece of emery paper with cyanoacrylate bonding adhesive and stored in damp gauze until testing. After calibration of the universal testing instrument (Instron), the enclosed vessel was gripped in the crosshead by planar rubbershod jaws. Distraction of the crosshead was initiated and maintained until the specimen separated.

Force generated at the instant of failure is denoted as "breaking strength" of the specimen. Vessel elongation to break is determined graphically using a technique that permits resting length to be determined under the same tension for each specimen. The results are expressed as "force," "elongation at break," and "percent elongation." Means and standard deviations for each group of anastomosed and unoperated vessels are given in Table 37.4.

Conclusions from Tensiometric Observations

Two problems were encountered in preparing vessel specimens for testing. One was assuring that bonding of the vessel end to emery paper had occurred, the other was measuring gauge length of the resting vessel if the specimen retracted and was too short. During tensile testing, observations were made to locate the point of tensile separation in each specimen. If a test abnormality was noted, e.g., slippage through the holder or jaw damage to the specimen, data for that trial were discarded. Data for one of 18 anastomosed and one of 18 unoperated arteries, one of 18 anastomosed and two of 18 unoperated veins were discarded. The remainder of the tests were considered valid.

Force-to-break tension and percent vessel elongation values are summarized in Table 37.4. Breaking strengths of anastomosed vessels were slightly less (arteries 7%, veins 20%) than those of paired unoperated

TABLE 37.4. Study 3: Tensiometry of microclipped rabbit femoral arteries veins, and unoperated controls.

	Time Periods					
	0 Days		7 Days		14 Days	
Parameter Tested	Anastomosis	Unoperated control	Anastomosis	Unoperated control	Anastomosis	Unoperated control
Force to break (GMS)*						
Arteries	274 ± 34 215–300;n = 5	295 ± 73 200–375;n = 5	398 ± 86 250–505;n = 6	361 ± 105 200–500;n = 6	399 ± 84 235–480;n = 6	317 ± 69 225–390;n = 6
Veins	171 ± 49 115–225;n = 5	213 ± 58 115–275;n = 6	270 ± 80 150–480;n = 6	211 ± 50 145–280;n = 5	247 ± 53 150–290;n = 6	215 ± 94 115–370;n = 5
Elongation to Break (%)*						
Arteries	183 ± 30 150–230;n = 5	193 ± 64 118–290;n = 5	179 ± 75 180–320;n = 6	266 ± 108 137–375;n = 5	200 ± 49 156–267;n = 5	193 ± 56 144–280;n = 5
Veions	109 ± 26 75–138;n = 4	264 ± 174 83–550;n = 5	97 ± 48 50–175;n = 5	141 ± 45 67–183;n = 5	162 ± 49 100–214;n = 4	191 ± 67 91–275;n = 5

*Expressed as an average ± standard deviation; (range); number of observations (n)
Instron parameters; jaw faces planar, rubbershod; jaw pressure 50 psi, load cell-tensiometric, gram cell, chart speed 1 in/min crosshead distraction rate—1 in/min

control vessels immediately after surgery. Postoperatively, anastomotic breaking strengths were found to increase over control values. The seven-day arterial anastomoses averaged 10% stronger and veins 15% stronger than unoperated control vessels. Since standard deviations for both anastomosed and unoperated groups were relatively large, the conclusion is that breaking strengths of vessels anastomosed with microclips is equivalent to those of normal vessels.

Percent elongation-to-break values between paired anastomosed and unoperated vessels similarly were large. Anastomosed veins tended to be less elastic than their unoperated controls, a physical property that correlates with normal fibrous reaction expected in the healing process. The tensiometric studies demonstrated that the clipped venous and arterial anastomoses were mechanically sound and functionally patent.

Study 4: Comparison of Microsutured and Microclipped End-to-Side Anastomoses Fistulas

An important aspect of clinical venous reconstruction is creation of an arterial-to-vein, end-to-side anastomosis (A-V fistula formation). Fistula formation is the cornerstone of circulatory access surgery for hemodialysis, chemotherapy, and total parenteral nutrition. The experimental fistula model for comparing effectiveness of microsuture vs. microclip was the common carotid to jugular vein, end-to-side anastomosis in the rabbit. Patency of the fistulas was evaluated 14 and 28 days after surgery by digital subtraction angiography in vivo and explant histopathology. The time required to construct a sutured vs. a clipped fistula was compared from the beginning point of anastomosis after mobilization of artery and vein.

Materials and Methods

Twelve anesthetized adult male New Zealand white rabbits, 3.2 to 3.75 kg, had the ventral cervical area depilated. Under sterile conditions and with the operating microscope, the rabbit's cervical region was incised to expose the right common carotid artery and external jugular vein. Animals were then divided randomly into two groups. Six animals had microsutured fistulas and six were microclipped. One group of six (three clipped, three sutured) were studied 14 days after performance of the anastomosis and the second group 28 days later. The jugular vein was prepared first and then the common carotid artery identified, sectioned, and the distal portion tied off. The carotid artery was then mobilized with a temporary microvascular clip, and double approximator clips were applied to the isolated external jugular vein on the right side. A 45′ angle oblique cut and small linear incision was made with microscissors (1.5 mm in length) at the site of anastomosis in the jugular vein. Microclips were applied to one wall and then the other, 10 to 15 microclips being utilized to complete the anastomosis.

Temporary occluding clips were released to establish patency and the wounds closed. The identical fistula was created with microsuture (10-0 nylon) in 10 rabbits, and these animals were studied in an identical manner. Patency was determined by angiography.

Results

Construction of a fistula by clipping averaged 10 minutes, by suture 50 minutes. No correlation could be established between the angiographic appearance of the fistulas and the histologic appearance of the anastomoses. Angiography gives a true picture of the in vivo fistula and explantation may introduce histologic artifacts. Twenty-eight-day fistulas (three sutured, one clipped) had small amounts of fresh fibrin at the anastomotic sites without evidence of endothelial disruption or thrombosis. This was considered to be artifactual in view of the patency visualized by angiography for both suture and clip. During explantation, arteries constrict due to trauma of removal from surrounding dense, fibrotic scars. The histologic dilatation observed at the anastomotic site in respect to the entering artery lumen could be explained by inability of the fixed anastomosis to constrict. The endothelium appeared complete at both sutured and clipped fistulas at 14 and 28 days, but there was slow replacement of necrotic media and compressed connective tissue in the sutured groups. In both the clipped and sutured fistulae there was proliferation of subintimal and intimal smooth muscle cells, whereas smooth muscle cell proliferation in the media was limited. The temporarily clipped carotid arteries demonstrated foci of mineralization in the adventitia and media, probably related to temporary occluding clip pressure.

Conclusions

Microarteriovenous fistulas (end-to-side) can be readily constructed by clips as a less tedious and surgically demanding procedure. Clipping is equivalent to suture with respect to patency as determined by angiography (100%) at 14 and 28 days. The angiographic appearance of the fistulas did not correlate with the histological appearance. The latter is complicated by explantation of fistulas from dense postoperative cervical surgical scars with artifactual trauma to the carotid artery and jugular vein.

Study 5: Comparison of Suture and Clip for Venous Anastomoses: Diameter Mismatch

Since the majority of clinical microvascular anastomoses occur between vessels of unequal diameter, the purpose of this study was to compare the ability of the surgical microclip vs. conventional microsuture for unequal diameter anastomoses. Experimental models consisted of venous and arte-

rial interpositional grafts in the rabbit (femoral vein to femoral artery, carotid artery to femoral artery).

Materials and Methods

Twenty-four adult New Zealand white rabbits (3.0 to 3.5 kg) had mismatched interpositional grafts placed. Two separate groups were prepared and studied.

Group I: Arterial Interpositional Grafts (12 Animals)

Two carotid artery grafts, each 2 cm long, were obtained from one carotid artery of each animal and used as interpositional grafts in the femoral arteries of the same animal. Interposition of one graft was performed with surgical microclips and the other with standard microsurgical suture technique. Ratio of outer diameters of femoral arteries to outer diameter of interpositional carotid grafts averaged 1:1.2.

Group II: Venous Interpositional Grafts (12 Animals)

One femoral vein was taken for two 1-cm interpositional vein grafts to both femoral arteries. Again, one side was sutured, the other microclipped. Ratio of femoral artery outer diameter to interpositional graft averaged 1:1.5.

Explantation of all interpositional grafts took place at 21 days after surgery. Animals were reanesthetized and surgical exposure of the operative femoral sites made to determine patency and explant the specimen. Patencies were determined by the "strip patency test." Specimens were harvested utilizing the glutaraldehyde formaldehyde fixation perfusion method described above. Specimens were embedded in glycol-methacrylate and sectioned with a glass knife with clips in place. Rabbits were killed after explantation by a lethal dose of IV pentobarbital.

Results

All of the clipped, mismatched venous and arterial grafts were patent at 21 days with no evidence of false aneurysm formation or bleeding, as were all of the sutured venous and arterial grafts. Histologic examination of interposed sutured arterial segments at 21 days revealed foreign body granulomas about sutures, various amounts of thrombus formation about the anastomotic line, but apparent endothelialization and patency. Medial necrosis was noted at the sutured anastomotic site with proliferation of the adventitia. Clipped arterial grafts had minimal thrombus but medial necrosis and thinning at the clip site. Medial necrosis was noted randomly in the arterial grafts and could not be correlated with surgical technique.

Venous interpositional grafts were all patent histologically without narrowing of anastomotic lumens. At 21 days the microsutured venous grafts

were marked by severe medial atrophy without evidence of repair through the graft. Microsutures were enveloped by an intense foreign body reaction with collections of macrophages and giant cells as well as intimal proliferation. The inflammatory reaction and medial necrosis were much less prominent in the microclipped anastomoses with smooth endothelialization at the anastomotic site. Histologic examination (longitudinal sectioning) revealed endothelialization of the interposed clipped grafts with connective tissue replacement at the anastomotic line. Sutured anastomoses had healed with moderate inflammatory reaction and organized thrombus about the suture line.

Conclusion

Both suture and clip were equally effective for interposition of mismatched venous and arterial interpositional grafts. Patency rates of 100% at three weeks were noted. These results are equivalent to patency rates obtained by microsuture or glue (97% patency) for rat interpositional veins in the jugular system. Venous anastomotic inflammatory response was less striking in clipped grafts than in sutured specimens.

Study 6

The purpose of this study was to compare the in vitro mechanical characteristics of clipped human saphenous vein anastomosed end-to-side to diseased human coronary arteries with those of the standard 7-0 polypropylene sutured anastomoses. These studies were performed on coronary arteries harvested from cadavers (3–7 days postmortem), rinsed with saline, and frozen for later use. Only vessels with significant atherosclerotic disease were used for the study. All saphenous veins were harvested from the distal lower extremities.

Methods and Materials

In order to optimize mechanical strength comparisons of the two different anastomotic techniques, pairs of anastomoses were performed on segments of the same coronary artery. If the artery branched, an anastomosis was performed on each of the branching segments. By this method, each pair of anastomoses was performed on an artery with equivalent disease. Anastomoses were generally constructed distal to any occlusive vessel disease, although some disease was usually present at the anastomosis site.

Though it could be argued that clip anastomosis should be compared to an interrupted, sutured anastomosis, most coronary anastomoses in adults are performed with a running technique. Properties of the clip were thus compared to conventional suturing methods for a typical coronary artery anastomosis. Anastomoses were analyzed for their abilities to withstand

both a tensile force and excessive intraluminal pressures ("burst strength").

Cadaver coronary arteries, with internal diameters of 2.0 to 3.5 mm, and distal saphenous veins, 3.0 to 4.0 mm, were used for the experiments. All anastomoses were performed by connecting the vein end to the coronary artery side. The angle between the vein and artery at the anastomosis site was analogous to that used in clinical coronary bypass surgery. A continuous suturing technique using 7-0 polypropylene monofilament suture was used. Average number of stitches per anastomosis was approximately 15. The mezzoclip was used for all anastomoses. Average number of clips per anastomosis was approximately 16, slightly exceeding the average number of stitches in the sutured anastomoses. The microclip technique required that the vein and artery be everted and approximated prior to placement of the clip. No internal stents were used.

Results

Coronary artery anastomoses are generally subjected to some tensile force as the heart moves during its contraction cycle. Therefore, it was felt that resistance to failure under tension for both techniques be compared.

Unpressurized Tensile Force

In the unpressurized state, vein–artery specimens were subjected to a linear force along the line of the vein graft, until the anastomosis or one of the vessels failed. Coronary vessels were secured to a mounting board with 18 g needles, and an increasing linear force was applied to the vein graft at the natural angle formed by the vein–artery interface. Linear force was generated by a water balance mechanism. With a constant flow of water into a reservoir, the rate of increase of force could be accurately maintained (240 grams/min). At the point of disruption of the anastomosis or vessel, the flow of water was halted. By measuring the amount of water in the reservoir, the force of failure under tension could be determined. Any disruption of a suture line or a clip was noted as a failure of the anastomosis; linear tears in a vessel extending from the anastomosis were also noted as a failure of the anastomosis.

The force of failure under tension in the unpressurized state is given in Table 37.5 for each pair of specimens tested. Specific information about the vessels, the anastomoses, and the mode of failure are given. Average force of failure under tension for the sutured vessels was 710 ± 93 grams; for the clipped vessels, the average was 479 ± 46 grams.

If one divides failure force by the cross-sectional area of the coronary artery, one can derive a quasi-tensile force for each anastomotic technique. This assumes that the strength of a coronary artery is proportional to its cross-sectional area, and the artery is the weak link in the anastomosis (no

TABLE 37.5. Study 6: Unpressurized tensile failure; clip vs. suture.

Technique	Number of stitches/clips	Vessel size (mm)	Failure mode	Tensile force (grams)‡
7-0 Polyprop*	18	2.5	Artery	405
Microclips†	20	2.5	Artery	345
7-0 Polyprop	18	2.0	Artery	775
Microclips	22	2.0	Anastomosis	415
7-0 Polyprop	18	2.0	Artery	600
Microclips	17	2.0	Artery	435
7-0 Polyprop	15	2.5	Artery	733
Microclips	14	2.5	Anastomosis	590
7-0 Polyprop	15	3.0	Artery	1037
Microclips	12	3.0	Anastomosis	610

*Surgilene, Davis and Geck—Lot No. 844362J
†Mezzo sized clips
‡Rate of Increase in Force 240 grams/min

failure of an anastomosis was caused by failure of the vein graft). Tensile force of the continuous polypropylene anastomosis was 167 ± 28 grams/mm^2 and 110 ± 12 grams/mm^2 for the interrupted clipped anastomosis.

Pressurized Tensile Force

Coronary artery anastomoses are not only subjected to tensile forces but also to the forces of transmural pressures. Failure force under tension was therefore measured for each anastomotic technique with the vein–artery system under a nonpulsatile pressure of 200 mm Hg. The identical water balance was used to apply a linear force to the vein graft.

A cannula was placed in the distal limb of the coronary artery so that a flow of normal saline could be used to pressurize the system. A second cannula was placed in the proximal limb of the artery for the purpose of pressure monitoring. This second cannula was connected to a Gould DTX pressure transducer. Output from the pressure transducer was connected to a PC computer via an analog-to-digital converter.

After stabilizing intraluminal pressure of the vein–artery specimen, an increasing tensile force was applied to the vein graft (240 grams/min). Individual failure forces and modes of failure are given for each pair of specimens tested in Table 37.6. Average force of failure under tension in the pressurized state was $631 + 70$ grams for sutured anastomoses and microclip anastomoses, respectively. Tensile force (failure force/cross-sectional area of the artery) was 108 ± 21 grams/mm^2 for sutured anastomoses and 114 ± 16 grams/mm^2 for clipped anastomoses.

Burst Pressure

Resistance to excessive intraluminal pressures was determined for each anastomosis technique. Pressure was applied to a cannula inserted into the lumen of the vein graft. A second cannula was placed in the distal limb of the artery and connected to a mechanical pressure gauge (USG 0–40 psi). The proximal limb of the artery was cannulated with a plugged cannula. The pressure gauge was calibrated using a 25-foot water manometer; accuracy was determined to be ± 0.5 psi. A limitation to the amount of pressure that can be applied to the vein-artery system is the leak rate of the anastomosis. Although inlet pressure can be increased to compensate for leakage, eventually the inlet cannula's internal diameter becomes the limiting factor to the amount of pressure that can be delivered to the system.

In burst pressure tests, very few actual disruptions of anastomoses occurred, even at very high intraluminal pressures (many times the maximum pressures encountered clinically). Only a single pair of anastomoses failed before limits of applied intraluminal pressure were reached. In this pair of specimens, the sutured anastomosis failed at 10 psi, and the microclip anastomosis failed at 8 psi. Coronary arteries used for this particular test pair had extensive disease and required an endarterectomy before

TABLE 37.6. Study 6: Pressurized tensile failure (200 mm Hg); clip vs. suture.

Technique	Number of stitches/clips	Vessel size (mm)	Failure mode	Tensile force (grams)‡
7-0 Polyprop*	13	2.0	Anastomosis	368
Microclips†	14	2.0	Anastomosis	525
7-0 Polyprop	13	3.0	Anastomosis	1232
Microclips	14	3.0	Anastomosis	500
7-0 Polyprop	12	3.5	Anastomosis	600
Microclips	15	3.0	Anastomosis	665
7-0 Polyprop	14	2.5	Anastomosis	665
Microclips	14	2.5	Anastomosis	590
7-0 Polyprop	17	3.0	Anastomosis	290
Microclips	19	3.0	Anastomosis	935

*Surgilene, Davis and Geck—Lot No. 8496195J
†Mezzo sized clips
‡Rate of Increase in Force 240 grams/min

TABLE 37.7. Study 6: Anastomosis burst pressures; clip vs. suture.

Technique	Number of stitches/clips	Vessel size (mm)	Failure mode	Luminal force (grams)‡
7-0 Polyprop*	17	2.5	None	9.5
Microclips†	16	2.5	None	9.5
7-0 Polyprop	18	2.5	Artery	10.0
Microclips	17	2.5	Anastomosis	8.0
7-0 Polyprop	13	2.5	None	16.0
Microclips	14	2.5	None	16.0
7-0 Polyprop	15	2.5	None	22.0
Microclips	14	2.0	None	23.0
7-0 Polyprop	14	2.0	None	20.0
Microclips	16	2.0	None	21.0

*Surgilene, Davis and Geck—Lot No. 8496195J
†Mezzo sized clips
‡1 psi = 51.7 mm Hg

preparing the anastomoses. Despite this fact, burst pressures of the anastomoses far exceeded clinical systolic blood pressures (200 mmHg = 3.86 psi) (Table 37.7).

If one assumes that the maximum pressure that could be delivered to a particular set of vein–artery specimens was the actual anastomosis burst pressure, average burst pressure for each technique was virtually equivalent (polypropylene continuous suture 15.5 ± 2.3 psi, interrupted mezzoclips 15.5 ± 2.7 psi).

Conclusions

Tensile strength of the interrupted clipped anastomosis in an unpressurized vessel is significantly less than that of a running polypropylene sutured anastomosis. The sutured anastomosis has approximately 50% greater tensile force than the mezzoclipped anastomosis.

When intraluminal pressure is applied to the vein–artery system, the microclip anastomosis gains strength as compared to the unpressurized state. There was no significant difference in tensile strength of the two anastomosing techniques in the pressurized state. Pressurizing the vein–artery system allows a more even distribution of tensile force around the entire circumference of vein artery anastomoses. More clips contribute to the anastomosis strength. No increase in tensile strength was noted in the polypropylene anastomosis.

Resistance to failure due to excessive intraluminal pressures was identical for both techniques. In most cases, the pressure needed to disrupt the anastomosis could not be achieved because of high resistance to flow within the vessels and high leak rates of anastomoses. Nevertheless, both anastomotic techniques have a high resistance to failure under pressure. Typical systolic arterial blood pressures are less than 4 psi. The only mezzoclipped anastomosis that failed did so twice at normal systolic blood pressure (>400 mmHg). These in vitro studies provide clear evidence that the interrupted mezzoclip application does not result in a significant reduction of mechanical anastomotic strength in comparison to conventional suture.

Clinical Application of the Clip

Access Surgery

The first systematic clinical application of the clip was for construction of autologous arteriovenous fistulas for hemodialysis. This venous reconstruction was selected because of the relatively high early failure due to thrombosis (16–20% during the first year),[19] minimal threat to life or limb, and the potential for noninvasive monitoring of fistula dimensions and flow rate. In order to design a clip for human arteriovenous fistulas, multiple

TABLE 37.8. Types of macroclipped subcutaneous A-V fistulas n = 20 (August 1988–December 1989).

Brescia-Cimino (radial artery to cephalic vein) (end-to-side)	5
Brachial	
Brachial artery to basilic vein	12
Gore-Tex prosthesis	3

measurements of human brachial arteries and cephalic veins were made. These measurements were made at surgery with calipers, and the results compared to dimensions obtained from study of formalin-fixed, stained surgical specimens. Wall thickness measurement of the brachial artery at the elbow ranged between 1.4 to 0.8 mm, cephalic veins from 0.11 to o.64 mm. Accordingly, the "macroclip" was designed (Fig. 37.6) with a space between the clip tips of 2.0 mm. It should be noted that measurements with micrometer oculars on formalin-fixed materia averaged 9–22% greater than gross measurements. This observation is consistent with measurements of wall thickness of unfixed rabbit femoral arteries in which histologic measurements after fixation average 16% greater.

This clinical study was initiated on August 2, 1988, at the Nephroligisches Zentrum Niedersachsen, Werra-Fulda-Klinic, Hann-Munden, West Germany (Prof. Dr. E. Quellhorst, Director) and has continued for the past 17 months. Drs. Winfred Schott and Kurt Rohwer performed all the procedures after being trained in the use of the clip. All the procedures were performed on patients whose A-V access procedures had failed on at least one occasion with conventional suturing methods and who had given informed consent. In most cases, there had been more than one thrombotic failure. Between August 1, 1988, and December 1989 a total of 77 arteriovenous fistulas were performed on 64 patients. Twenty patients with previous suture failures have had macroclipped arteriovenous fistulas (Table 37.8).

All the clipped fistulas have thus far remained patent and in steady use for hemodialysis with the exception of one patient. This man died four months after the procedure of myocardial infarction. The fistula was patent up to death. In one case (basilic-vein to a brachial artery), a postoperative subcutaneous hematoma required draining 24 hours after surgery. The fistula was noted to be blood-tight at reexploration. The only complication of note was slow and progressive growth of two clipped fistula sites that increased the minute shunt volume to the point of creating a peripheral "steal" syndrome. In these two cases it was necessary to reexpose the fistula and narrow the anastomoses with suture. Spontaneous narrowing of a clipped anastomosis has not yet been observed. This phenomena of progressive fistula growth observed with clips may be beneficial when utilizing clips for anastomoses in the distal forearm or creating angioaccess in

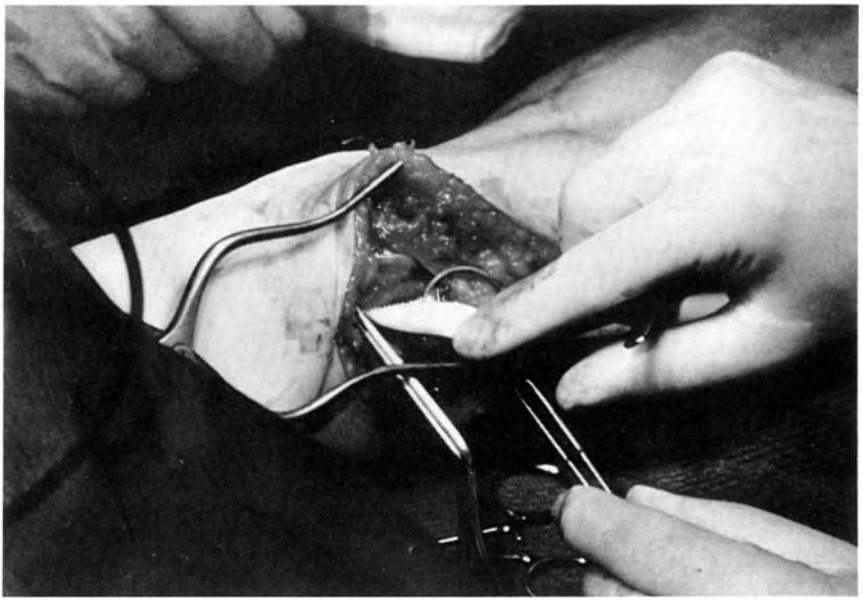


FIGURE 37.13. Construction of a Gore-Tex subcutaneous prosthetic basilic vein to brachial artery arteriovenous access fistula with clips. An 8 mm thin wall PTFE bioprosthesis is being macroclipped end-to-side to the basilic vein.

small diameter (1 mm) vessels of children. Operative photographs of the creation of a basilic artery to basilic vein Gore-Tex end-to-side prosthetic fistula with macroclips is given in Fig. 37.13.

In the same period of time (August 1988–December 1989), 57 arteriovenous fistulas were created by conventional suture, 42 at the wrist (Brescia-Cimino) and 15 at the brachial level. Early thrombosis (within six months) was noted in nine patients or 11%. This study remains in progress and the results are under continuous review. Provisional conclusions are that clipped fistulas have a lower early thrombosis rate than those made with conventional sutures and can exhibit progressive enlargement rather than stenosis.

Free Flap Reconstructions

The transposition of large composite tissue blocks to fill extensive body defects is dependant on the successful reconstruction of microarterial and microvenous anastomoses. The clip has been used with great success in 15 cases and enables the rapid assembly of venous and arterial vascular connections, thus reducing the warm ischemia time of the transpoised tissue block. The first successful application of the "Kirsch clip" for a free-flap transfer was conducted by Dr. Martyn Webster at the Canniesburn Hos-

FIGURE 37.14. End-to-end free flap venous anastomoses with mezzoclips (free flap Case 1 from text). Twelve mezzoclips were utilized and a "blood-tight" anastomosis accomplished in eight minutes. The veins were 3.0 mm in diameter (venae comitantes) and 2.5 mm O.D. (posterior tibial vein).

pital, Glasgow, Scotland. This procedure, a breast reconstruction, was performed on closed circuit television for the participants of the Microsurgical Society Meeting (January 1991). Dr. Webster's experiences with the clip have been uniformly positive with eight free-flap transfers. Dr. Webster feels that the clip offer significant advantages in terms of speed and precision of the anastomotic portion of the free flap transfer. The remainder of the other clip mediated free flap transfers have been accomplished at the University of California Irvine (Dr. David Furnas) and Loma Linda University (Dr. Robert Hardesty).

Details of Hardesty's (Cases 1, 2) cases are given below to illustrate other clinical experiences.

Clinical Applications of the Non-penetrating "Kirsch" Clip

CASE 1: FIGURE 37.14

A 68-year-old diabetic woman with an 8 cm by 7 cm ulcerated osteomyelitic lesion of the left tibia and fibula had transfer of a rectus abdominous muscle flap to cover the defect. The anterior tibial artery and two vena

comitantes were isolated in the leg, and joined end-to-side to the epigastric artery and to the vena comitantes of the rectus abdominous muscle. Warm ischemia time for the flap was 98 minutes: suturing time: tibial to epigastric artery, 30 minutes; one posterior tibial vena comitante to one rectus vena comitante, 25 minutes; the third anastomoses of the post-tibial and rectus vena comitante was done with mezzoclips, eight minutes. The muscles were covered with split skin. The flap remains viable.

Comment

This case illustrates the advantage offered by the clips in shortening the duration of the anastomotic procedure, particularly for the venous anastomoses. This reduction of warm ischemia time can contribute to flap survival, but more importantly the troublesome venous anastomosis is simplified.

Case 2

A 72-year-old male with metastatic squamous carcinoma of the right buccal cavity with dissemination into the mandible had been previously treated with a right radical neck dissection, mandibulectomy, and 5000 rads of local irradiation. The extensive ulcerating lesion was debrided with a right radical neck dissection. Reconstruction of the oral cavity and neck was attempted with a transverse rectus abdominus myocutaneous flap. The right common, internal, and external carotid artery were profoundly atherosclerotic and diseased secondary to irradiation. The superior thyroid artery was opened and found to be totally occluded. The flap was placed into position and a venous anastomoses between the rectus vena comitantes was promptly established by mezzoclips end-to-side to the right internal jugular vein. The end-to-side clip anastomoses of veins required 6 minutes. The markedly atheromatous, and almost totally occluded, external carotid stump was then clipped end-to-end to the rectus arterial supply. Flow through the flap was adequate for 15 minutes and then stopped. The arterial anastomotic site was occluded with fresh thrombus, particularly proximally. The arterial anastomoses was then restructured with 8-0 nylon and blood flow was temporarily restored to the flap with excellent venous drainage. The arterial anastomoses thrombosed again after a ten minute interval. A third attempt to establish an arterial supply to the free flap was made by attempting a microsutured anastomoses end-to-side to the right common carotid artery. The artery was severely atherosclerotic and a limited endarterectomy was performed before establishing a microsutured anastomoses. Perfusion was restored to the flap for about ten minutes with excellent venous drainage, but again the arterial anastomoses thrombosed. The free flap was then abandoned and coverage was accomplished by a pectoral flap.

COMMENT

Case 2 is the only failure to restore circulation with either clips or sutures in a free flap transfer. The case demonstrates the futility of attempting to access an exceedingly diseased arterial supply to a free flap. In retrospect, the external carotid artery should have been endarterectomized at its origin or an interpositional venous graft should have been placed. The remarkable fact is that the clipped venous anastomoses did not occlude and reopen with arterial perfusion. This event would be unusual in a sutured venous anastomoses.

Venous Hypertension and Valve Reconstruction

Drs. Nicholas Wilson and Derek Rutt of the St. Thomas's and Guy's Hospital, London, England (laboratory of Dr. Norman Browse), have been introduced to the clip concept and trained in its use. This group of investigators have devised a new procedure for creation of valves in incompetent lower extremity veins based on use of the nonpenetrating mezzoclip. Their experience with this new procedure for venous hypertension of the lower extremities was presented in January 1990 at a meeting of the British Surgical Society and details are in press.[16] This procedure fails with sutures because of intimal damage.

Conclusions

In experimental models of venous reconstruction encompassed by this study (rat, rabbit), the nonpenetrating clipped anastomosis proved to be both technically and biologically superior to conventional needle and suture. The advantages of the clip for venous reconstructions can be summarized as follows:

1. The anastomosed vein wall is rapidly restored morphologically to its preoperative state—with microsutures the wall is never fully restored.
2. Endothelialization of the anastomotic site occurs within one week without evidence of intimal hyperplasia. This time course is consistent with previous reports of the time for vein anastomotic site endothelialization with the Nakayama ring.[15] With suture, and particularly suture error, endothelialization of the anastomotic site may be delayed two to three weeks or may never occur when associated with myointimal overgrowth and platelet adherence.
3. With microclips, endothelialization begins at the anastomotic cleft. This observation fits with the hypothesis that endothelium is regenerated from multipotential cells derived from the blood stream as well as from in-growth replacement.[20]
4. Mechanical properties of the clipped anastomosis, including patency, tensile strength, and burst strength, are equivalent to, or superior to, results with microsuture.

5. The technique is adaptable to vessel caliber and wall thickness mismatch, end-to-side anastomoses, and bioprosthetic vessel insertion. The technique is more rapid and reproducible than suture.

Biological advantages of the clip appear to reside in the fact that intima is not penetrated and there is no foreign body within the vessel lumen. The remarkable adhesive properties of coapted, flanged intima (arterial and venous) provide an immediate "blood-tight" anastomosis in contrast to suture. Sutures result in an immediate intraluminal fibrin-platelet coagulation at the point of intimal injury that can readily propagate into a thrombus. The clipped anastomosis is devoid of this response, a platelet-mediated simulation of smooth-muscle cells and so-called "myointimal" proliferation.[21,22] There is now evidence that endothelial regrowth in injured rat and rabbit arteries is inhibited by the presence of luminal smooth-muscle cells,[23] a mechanism conceivably responsible for progressive compensatory myointimal proliferation and subsequent vein occlusion.

An important question raised by the nonpenetrating clip's effectiveness for venous repair is definition of the mechanism for the observed rapid healing and vein wall remodeling. What molecular factors govern the functional reorganization of endothelial lined surfaces when held in close contact but not traumatized by penetration? This wound healing takes place without cellular proliferation and with the apparent maintenance of cellular function. Thus, the nonpenetrating clip provides an interesting model for study of in vivo wound healing—particularly the venous and arterial endothelium. Our findings confirm previous observations of Lidman and Ostrop, who noted rapid venous endothelialization after intima-to-intima coaption without penetration.[15]

The vascular endothelium is a large expanse of tissue, in the order of 6×10^{13} cells or approximately 1000 grams in weight, about the number of cells in a small organ.[24] The vascular endothelium, and particularly venous endothelium, must exert moment-to-moment control of two diametrically opposed functions: preventing thrombosis during sluggish, hypoxic blood flow conditions and initiating thrombosis of blood in the event of a leak. Only recently have macromolecular products of subendothelial collagens and endothelial cells themselves been identified and their physiologic functions defined.[25] The nonpenetrating clip may provide new experimental avenues to define the role of these molecules in healing of venous and arterial wounds.

References

1. Obora Y, Tamaki N, Matsumoto S. Nonsuture microvascular anastomoses using magnet rings: Preliminary Report. Surg Neurol 1978;9:117–120.
2. Daniel RK, Olding M. An absorbable anastomotic device for microvascular surgery: Experimental Studies. Plast Reconstr Surg 1984;74:329–342.
3. Gottlob R, Blumel G. Anastomoses of small arteries and veins by means of

bushings and adhesive. J Cardiovasc Surg 1968;9:337–341.
4. Quigley MR, Bailes JE, Kwaan HC, et al. Microvascular anastomoses using the milliwatt CO2 laser. Lasers Surg Med 1985;5:357–365 .
5. Hobson RW, Rich NM, Wright CB. *Complications in Vascular Surgery*. Barnhard VM, and Towne JB, eds. New York: Grune and Stratton, 1980:325–340.
6. Haimovici H. *Vascular Surgery. Principles and Techniques*. New York: McGraw-Hill Book Company, 1976.
7. Ramos JR, Berger K, Mansfield PB, et al. Histologic fate and endothelial changes of distended and nondistended vein grafts. Ann Surg 1976;183:205–228.
8. Zhong-Wei C, ed., *Microsurgery*. Springer-Verlag, Berlin, Heidelberg, New York: Shanghai Scientific and Technical Publishers, 1982:72.
9. Coblett JR. Microvascular surgery. Surg Clin North Am 1967;20:16–20.
10. Rutherford R. *Vascular Surgery*. WB Saunders Co., Philadelphia, 2nd ed., 1984:339.
11. Lidman D, Daniel RK. The normal healing process of microvascular anastomoses. Scand J Plast Reconstr Surg 1981;15;103–110.
12. Bikfalvi A, Dubecz S. Observations in animal experiments with mechanized vessel suture. J Int Chir 1953;5:481–496.
13. Samuels PB. Method of blood vessel anastomoses by means of metal clips. AMA Arch Surg 1955;70:29–38.
14. Zhu YH, Kirsch WM, Cushman R, et al. Comparison of suture and clip for microvascular anastomoses. Surgical Forum 1985;36:492–495.
15. Lidman D, Ostrup LT. Blood flow and scanning electron microscopic study on venous microvascular anastomoses with Nakayama's apparatus and manual suture. Scand J Plast Reconstr Surg 1981;15:97–101.
16. Wilson N. Bri J Surg, in press.
17. Acland RD, Trachtenberg LS. A method for accurately orienting microsurgical blood vessel specimans for longitudinal sectioning. Stain Technol 1977;52:114–116.
18. Thurston JB, Buncke HJ, Chapter NL, et al. A scanning electron microscopy study of micro-arterial damage and repair. Plas Reconstr Surg 1976;57:197–203.
19. Kootstra G, Jorning PJG, eds., *Access Surgery*. Lancaster. MTP Press Ltd., 1982:21–29.
20. Stump M, O'Neal R, DeBakey M, et al. Growth potential of circulating cells in the peripheral blood. Surg Forum, 1963;14:301–303.
21. Rosenbaum TJ, Sundt TM Jr. Thrombus formation and endothelial alterations in microarterial anastomoses. J Neurosurg 1977;47:430–441.
22. Ross R, Glomseth J, Kariya B, et al. A platelet dependent serum factor that stimulates the proliferation of arterial smooth muscle cells in vivo. Proc Natl Acad Sci 1974;71:1207–1212.
23. Reichy MA, Clowes AW, Schwartz SM. Endothelial regeneration. Inhibition of endothelial regrowth in arteries of rat and rabbit. Lab Invest 1983;49:569–575.
24. Wolinsky H. A proposal linking clearance of circulating lipoproteins to tissue metabolic activity as a basis for understanding atherogenesis. Circ Res 1980;47:301–311.
25. Martin GR, Timpt R. Laminin and other basement membrane components. Ann Rev Cell Biol 1987;3:57–85.

38
Budd-Chiari Syndrome (How I Treat It) Personal Experience of 250 Cases

ZHONGGAO WANG

Introduction

Budd-Chiari syndrome (BCS) is a disease of hepatic venous outflow disturbance caused by occlusion of the hepatic veins (HVs) and/or the retrohepatic segment of the inferior vena cava (IVC) proximal to the hepatic venous ostiae. BCS is characterized by clinical manifestation of portal hypertension either with or without IVC hypertension. It was first described by Budd in 1845 and by Chiari in 1899, and it was thought to be a rare disease. Hepatophlebo-vena caval occlusive syndrome might be a better name for this disease entity. Due to a lack of knowledge of the etiology and epidemiology of this disorder, preventative measures are not available. Methods of treatment have been far from uniformly satisfactory. Prognosis is poor if no proper corrective means is instituted. Increasing awareness of this syndrome and rapid development of vascular imaging technology during the last decade have resulted in an increased rate of discovery of this disease in some countries. This paper presents our clinical experience in the management of 250 such patients along with our work on the experimental and epidemiological aspects of this disease and reviews of the related world literature.

Material and Methods

Epidemiological Study

The distribution of 250 cases treated by the author was analyzed geographically. A formal epidemiological study was carried out in 1988 in Dongping County, Shandong Province, China, which has some 680,000 inhabitants. The county was chosen as an example of typical Chinese demographic characteristics. Three towns (total population 100,680) were selected as geographically representative samples of the target county. Every inhabitant of these towns was screened by the author's team for symptoms and

signs relevant to BCS. The team was composed of 15 medical workers using a questionnaire and standardized examination format. Firm diagnosis was made by means of a hierarchical series of investigations consisting of ultrasonography, venography, and laparotomy. At the county level, practitioners from 10 hospitals received specific diagnostic training from the author's team and were issued the standardized questionnaire and examination format. These were employed as the method for discovering BCS patients within the catchment of each hospital. All discovered cases were treated by the author's team.

Animal Experiment

An animal model of BCS was created in 4 male mongrel dogs. Under intraabdominal anesthesia of pentobarbital (20 mg/kg), a right thoracotomy through the 7th intercostal space was performed, and the supradiaphragmatic IVC was partially ligated to about 30% of the original diameter. All animals developed marked hepatomegaly, varicose veins on the abdominal wall, and ascites appeared on the third postoperative day. Pressure of the IVC increased 8.3 cm water column, from 7.90 ± 1.33 to 16.2 ± 5.76 ($p < 0.05$), and that of the portal vein 12.4 cm water column, from 16.8 ± 5.56 to 30.0 ± 1.87 ($p < 0.01$). All the dogs were cured of the disease by either balloon dilatation or cavoatrial shunt.

Another 21 dogs were used to investigate suitable IVC shunting grafts. Partial IVC was resected and replaced by Meadox Double Velour, knitted Dacron graft 5 cm in length and 10 mm in diameter. Among them, 12 grafts were seeded with Factor VIII antigen-positive, autogenous endothelial cells (ECs) derived enzymatically (collagenase) from the omentum (density was $7.0 \pm 2.1 \times 10^5$ ECs per square centimeter of graft surface) before implantation. These served as an experimental group in which 3 samples of graft were harvested at 7 days and 9 samples at 100 days following surgery. The other 9 grafts received only a serum preclotting (sham seeding) and served as a control group. A distal arteriovenous fistula (AVF) was created, and it functioned for 1 week in all animals.

Additionally, 42 male mongrel dogs were used for investigation of endothelial seeding effect on different materials and configurations of the grafts.

Clinical Experience

Two hundred and fifty cases of BCS were admitted to the Department of Vascular Surgery in the Peking Union Medical College Hospital, the Beijing Heart Lung Blood Vessel Medical Center Anzhen Hospital, and another 13 hospitals between December 1982 and March 1991, all treated by the author himself. There were 165 males and 85 females, with a male to female ratio of 1.94:1. Ages ranged from 2.5 years to 65 years with an

average of 33.3 years. Symptoms had been present for periods ranging from 3 days to 26 years. All cases were confirmed by B-ultrasonography, venacavography, or hepatovenography through the femoral venous catheterization or the percutaneous transhepatic approach. Ten patients underwent splenopneumopexy, omentopneumopexy, azygoatrial shunt, cavoatrial shunt, mesoatrial shunt, membranotomy, splenectomy, balloon dilatation, etc., in other hospitals prior to admission.

Various operative methods, from symptomatic to radical treatment, were employed in this study. Graft materials used for the shunting procedures were woven Dacron (14 mm to 22 mm in diameter, made in China), Gore-Tex graft (external ring enforced, 16 mm in diameter, made in USA), Vascuteck external ring, supported knitted graft (made in Scotland), polytetrafluoroethylene (PTFE, made in Shanghai) graft with external ring (14 mm or 17 mm in diameter), composite spiral saphenous vein graft, and saphenous vein. Stainless steel spiral external support, combined with Chinese-made Dacron and Meadox Double Velour, knitted Dacron grafts, were also used. Among them, 8 Chinese woven Dacron grafts, 1 Meadox graft, and 1 Vascuteck knitted product were pretreated with EC seeding technique prior to implantation.

Conservative management was mainly composed of symptomatic treatment, urokinase, or Chinese herbal medicine and was reserved for those cases who refused surgery, presented at an acute stage, or who belonged to the endstage cases incapable of tolerating surgical intervention.

Follow-up study was regular review of all patients, including venography in 50 patients, superior mesenteric arteriography in 3, and ultrasonography in all shunting cases.

Results

Distribution of the 250 cases is shown in Fig. 38.1, in which each point represents a patient. It was clear that the most concentrated prevalence of BCS is along the lower reach of the Yellow River and is somewhat correlated to a high-incidence area of esophageal carcinoma. In addition to the author's series of 250 cases in this high-prevalence area, there were at least a dozen hospitals that discovered or treated from 20 to 100 such cases each in the recent 3-year period. In one village, with a population of less than 900, 4 cases were screened out. In the author's series, 2 IVC web patients are brothers.

The epidemiological survey in Dongping County found a total of 44 BCS patients, for a prevalence or 6.5 per 100,000 population, and in the 3 towns, the prevalence was 12 per 100,000. There were 29 males and 15 females. Their ages ranged from 21 years to 57 years with an average of 38.5 years. Symptoms had been present between 3 months and 15 years,

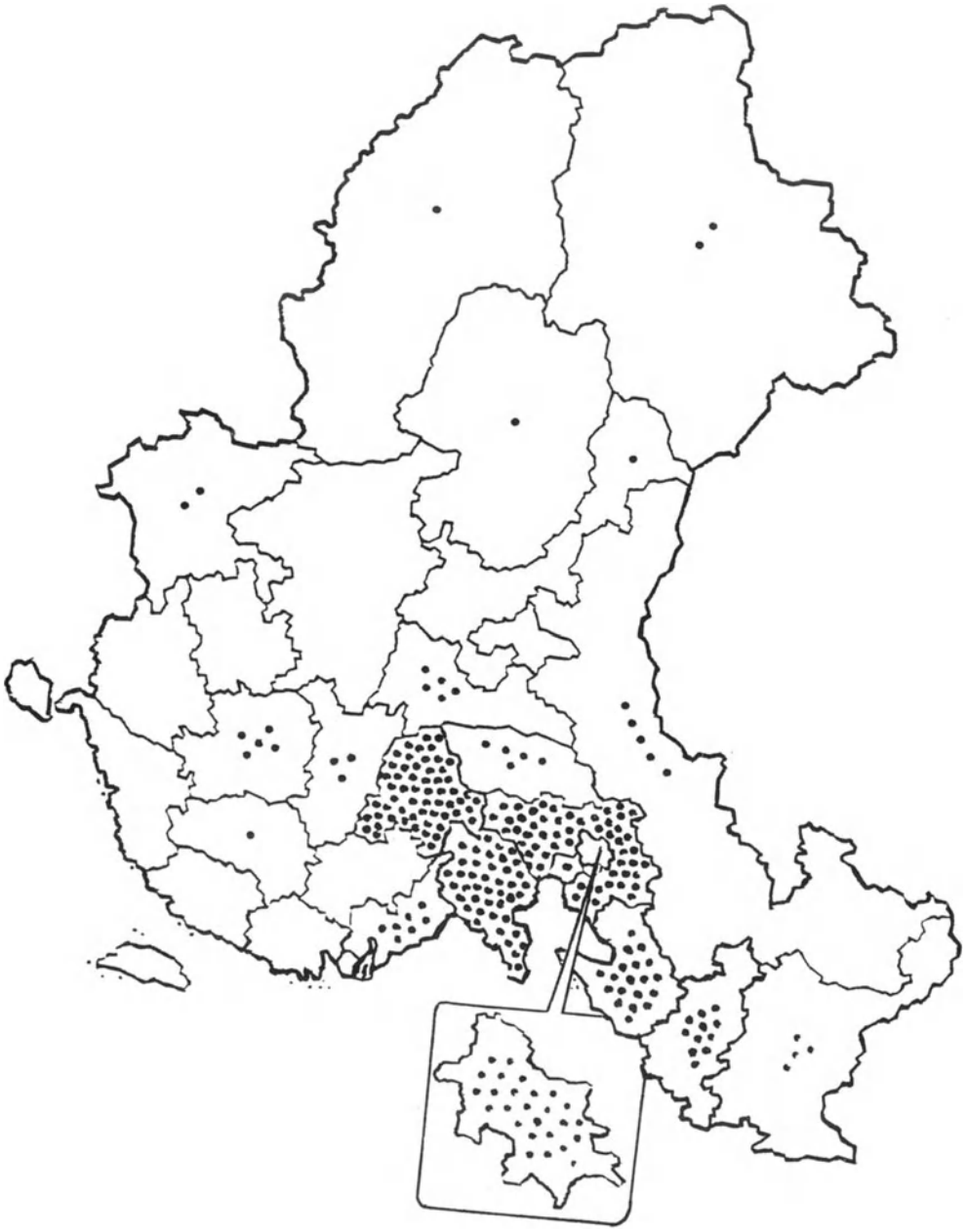


FIGURE 38.1. Distribution of the author's 250 cases in China. Each point represents a patient.

with a mean of 3.1 years. Membranous obstructions were confirmed by surgery in 5 cases (11%).

All dogs in the animal experiments recovered following corrective management. Elevated IVC and superior mesenteric vein (SMV) pressures were reduced to 7.15 ± 0.87 and 15.75 ± 5.12 cm water column, respectively, which are within the normal range, and clinical signs disappeared within a week. For the EC seeding study, 3 seeded grafts were harvested at 7 days and 9 grafts at 100 days following implantation; 9 control grafts were all procured at 100 days. A completely confluent endothelial lining had formed and was confirmed in all grafts tested at 7 days following implantation. All seeded grafts (9/9) and only 2 control ones (2/9) were patent ($p < 0.001$), and the neointimal thickness was significantly thinner in the former (302.0 ± 106.1 microns) than in the latter (783.1 ± 84.2 microns) ($p < 0.01$). Thus an increasing patency and inhibition of neointimal hyperplasia of vena caval, knitted Dacron prosthesis was achieved by high density seeding of the omentum-derived autogenous Factor VIII positive ECs. It was also found that an early endothelialization at 10 days after insertion of EC-seeded implants was established in the high porosity, knitted Dacron (12/12) and PTFE (4/4) grafts, but not in the silk (0/4) and woven Dacron (0/4) grafts.

Clinical findings and underlying pathology are listed in Tables 38.1 and 38.2. The superior vena cava (SVC) occlusive syndrome accompanied 10 cases (4%) in this series.

Eight pathological categories and the number of patients in each of them are shown in Fig. 38.2. These 8 are: 1) incomplete IVC web, 2) complete

TABLE 38.1. Clinical fiindings (N = 250).

Classification	Cases	Percentage (%)
Thoraco-abdominal varicose veins	231	92.4
Edema of lower extremities	197	78.8
Labial/scrotal edema	19	7.6
Hepatosplenomegaly	235	94.6
Ascites	164	65.6
Esophageal varices	125	50.0
Hematemesis	64	25.6
Exertion palpitation	102	40.8
Hepatic coma	12	4.8
Oliguria	124	49.6
Abdominal wall hernias	23	9.2
Gynecomastia	16	6.4
Jugular enlargement	12	4.8
Pleural effusion	34	13.6
Cachectic appearance	72	28.8
Albumin/globulin reversal	49	19.6
Abnormal liver function tests	42	16.8

TABLE 38.2. Etiology (N = 250).

Classification	Cases	Percentage (%)
IVC web	102	40.8
"Thrombosis"	73	29.2
"Vasculitis"	5	2.0
Liver cirrhosis	4	1.6
Malignancy	3	1.2
External compression	3	1.2
Behcet's syndrome	2	0.8
Pregnancy	1	0.4
Polycystic disease of liver	1	0.4
Inflammatory constriction of IVC	1	0.4
Chronic myeloid leukemia	1	0.4
Polycythemia	1	0.4
Malconnection of IVC	1	0.4
Cause unknown	52	20.2
Total	250	100.0

IVC web, 3) localized IVC stenosis, 4) localized IVC occlusion, 5) long segment IVC constriction, 6) long segment IVC occlusion, 7) occlusion of HVs, and 8) occlusion of hepatic venules.

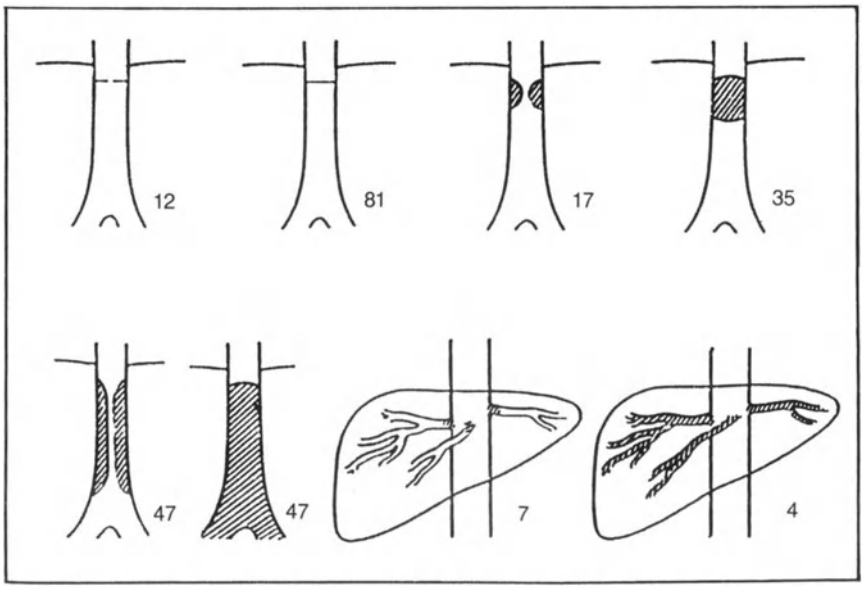


FIGURE 38.2. Pathologic classifications showing Types I to VIII, from left to right, together with the numbers of cases.

TABLE 38.3. Gradation of severity of BCS.

Stage	I	II	III	IV
Life quality	Good	Fair	Poor	Worse
Ascites	None or slight	Moderate	Severe	Extreme and uncontrollable
Esophageal varices	None or slight	Moderate, no bleeding	Severe or bleeding episodes	Acute hematemesis
Serum albumin (g%)	>3.5	3.4–3.0	2.9–2.5	<2.4
Bilirubin (mg%)	<1.2	1.3–2.4	2.5–2.9	>3.0
Nutritional status	Good	Fair	Poor	Malignant appearance
Operative risk	Good	Moderate	Poor	Very poor

Gradation of Lesions

In order to judge the severity level of patients and to approximately predict prognosis and surgical outcome, 4 stages were defined, as described in Table 38.3. There were 29 (11.6%) Stage I patients who had basically no symptoms or barely noticeable ones; 79 (31.6%) Stage II cases had mild to moderate ascites or esophageal varices; 72 (28.8%) Stage III cases had either distinct ascites, marked esophageal varices, or poor general condition; 70 (28.0%) Stage IV cases were of very poor constitution, suffered attacks of hepatic coma or acute massive hematemesis, or had albumin levels below 2.4 g% or bilirubin above 3 mg%.

Conservative Therapy

In 40 nonoperative cases, conservative therapy was used. The only positive result was marked relief of ascites in one patient; 13 patients died of late complications, such as hepatorenal failure, hematemesis, or septicemia of *Pseudomonas aeruginosa*. All 13 cases who died within 6 months after final diagnosis were in the Stage IV lesion group. Mortality for Stage IV nonsurgical patients was thus 87% (13/15) in this series.

Surgical Treatment

The remaining group of 210 cases had surgical treatment. Table 38.4 indicates the outcome of the various operative procedures. A good result was defined as complete resolution of ascites, edema of the lower extremities, and esophageal varices. A fair result indicates relief but incomplete resolution of symptoms and signs. A poor result represents little change in patient condition.

TABLE 38.4. Therapeutic approaches and results (N = 210).

Classification	Good	Fair	Poor	Rec*	Died		Total
					Op**	Late	
Mesoatrial shunt	35	4	—	5	6	1	51
Splenoatrial shunt	1	—	—	—	—	—	1
Membranotomy	25	6	3	7	1	2	44
Cavoatrial shunt	29	2	1	4	2	1	39
Balloon catheter dilatation	12	5	4	1	—	—	22
Radical resection	14	3	—	1	1	1	20
Mesocaval shunt	6	1	1	—	1	2	11
Mesojugular shunt	4	—	—	—	—	1	5
Mesocavoatrial shunt	—	—	—	—	3	—	3
Mesoatrial & innominate-atrial shunt	2	—	—	—	—	—	2
Membranotomy & innominate-atrial shunt	1	—	—	—	—	—	1
Splenorenal shunt	2	1	—	—	—	—	3
Cavoazygous shunt	—	1	—	—	—	—	1
Splenectomy & embolization of left gastric vein	—	1	—	—	—	—	1
Thoracic duct-internal jugular vein reanastomosis	—	1	—	—	—	—	1
Cephalofemoral shunt	—	1	—	—	—	—	1
Releasing external compression (+ dilatation)	2	1	—	—	—	—	3
Mesoinnominate shunt	—	—	—	—	1	—	1
Total	133	27	9	18	15	8	210
Percent	63.3	12.9	4.3	8.6	7.1	3.8	100

*Recurrent.
**Operative death.

Postoperative Complications

Fifteen patients died within 30 days following surgery, an operative mortality of 7.1%. Postoperative complications are listed in Table 38.5. Chylous hydrothorax was resolved by paracentesis of the thoracic cavity and infusion of tetracycline or "fever" therapy induced by intramuscular injection of sterilized fresh milk. For chylous ascites, parenteral nutrition of approximately 1 month was given. Other complications were resolved uneventfully with appropriate therapies.

Continuing follow-up of operative survivals has been conducted by letter, ultrasonography (Fig. 38.27, 38.28), cavography (Fig. 38.19), portovenography (Fig. 38.24), or superior mesentery arteriography (Fig. 38.21). To date, the mean follow-up period is 32 months, with the longest more than 8 years. Good results have been achieved in 63.3% of the pa-

TABLE 38.5. Postoperative complications (N = 210).

Classifications	Number of cases	%
Cardiac insufficiency	46	22.0
Encephalopathy	11	5.3
Pleural effusion	10	4.8
Chylous ascites	7	3.3
Hemothorax	6	2.9
Chylous thorax	3	1.5
Hydromediasternum	2	1.0
Atrial premature	2	1.0
Stress ulcer	2	1.0
Pulmonary abscess	1	0.5
Hepatitis B	1	0.5
Cardiac tamponade	1	0.5

tients and fair results in 12.9%. Thus, the operative outcome was effective in 76.2%. In one female patient, graft patency was maintained through pregnancy and the child is now 6 years old. Among 8 late deaths, 4 were due to other causes. Of these 4, 2 shunting grafts and 1 membranotomized IVC were patent; the 4th graft was compressed by an expanding liver carcinoma. The other 4 late deaths were due to graft occlusion or hepatorenal failure. Seven shunting grafts were revised because of their occlusion, resulting in 6 successes and 1 death. Five-year patency of the mesoatrial shunt (37/39, 29/33, 19/25, 15/21, 10/14), the cavoatrial shunt (26/30, 16/22, 8/14, 4/8, 2/4), and membranotomy (25/34, 22/31, 16/25, 8/12, 6/9) is shown in Fig. 38.3. Patency was confirmed by ultrasonography in most cases.

Discussion

Historical Review

Since Budd described a case of hepatic venous thrombosis due to liver abscess in 1845 and Chiari described three cases of hepatic venular thrombosis or obliterative thrombophlebitis of unknown cause in 1899,[1,2] the initial concept of Budd-Chiari syndrome was established. It represented mainly the status of thrombotic obstruction of the intrahepatic veins and was claimed as a very rare disease for many decades. With increasing awareness of this disease and the development of medical imaging facilities, more and more cases have been discovered in many countries. The spectrum of this disorder has been expanding to include any clinical entity that may cause an outflow blockage of the liver sinusoidal bed, resulting in portal hypertension with or without IVC hypertension. Thus, in addition to pure obstruction of hepatic veins (HVs) or hepatic venules, obstructions of the hepatic portion of the IVC and the right atrial orifice of the IVC, as

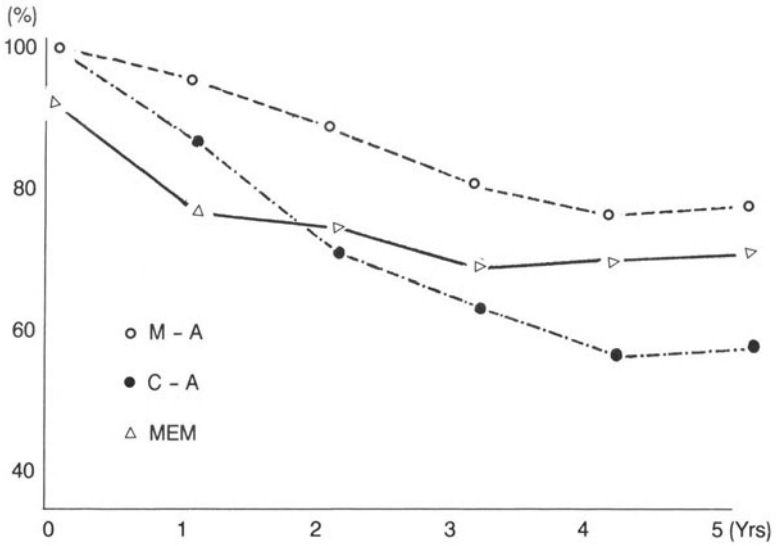


FIGURE 38.3. Five-year patency curves of mesoatrial shunt (M-A), cavoatrial shunt (C-A), and membranotomy (MEM).

well as multiple-positioned obstructions of the vena cava, and HVs were added to this disease entity. Those derivative types involving IVC became predominant in Eastern countries and South Africa. The disease even involves the SVC in a small number of patients, such as the 10 (4%) in this series of patients. Thus a new name, hepatophlebo-vena caval occlusive syndrome, has been suggested or advocated.[3,4]

In order to resolve this entity, Kimura first reported a membranotomy through the right atrium by finger fracture in 1963,[5] and O'Hara the cavoatrial shunt, also in 1963.[6] Hirooka and Kimura suggested 7 pathological classifications in 1970.[7] Victor introduced a retrohepatic cavoatrial bypass to treat BCS in 1974.[8] Yamamoto used the azygocaval anastomosis as a means for relieving BCS in 1969.[9] Putnam reported treating BCS with liver transplantation in 1976.[10] Akita first employed splenopneumopexy in 1980.[11] Cameron first created a mesoatrial shunt in 1983.[12] Senning reported a transcaval posteriocranial resection of the liver as treatment of BCS in 1983.[13] Eguchi and Yamada reported transluminal balloon dilatation to treat BCS in 1974 and 1983, respectively.[14,15] Franco reported a portoatrial shunt for treating BCS in 1986.[16]

In Japan, there were 192 reported cases before 1988.[17] In China, the first case was reported in 1957. Sun reported cavoatrial shunt in 1982.[18] Liu performed splenopneumopexy in 1982. Wang carried out finger membranotomy in 1982, mesoatrial shunt in 1983, transluminal balloon catheter membranotomy in 1984, splenoatrial shunt in 1985, radical resection in 1986, and mesojugular shunt in 1990, all being the first in China.[19]

Epidemiology

BCS is relatively more common in East Asia than in the Western world. There were 239 cases reported in China before early 1988. The first International Symposium on Budd-Chiari Syndrome was held in September 1988 in Jinan, China, and 25 Chinese hospitals presented a total of 566 cases during the symposium. It is estimated that 500 new cases have been discovered since then. The highest prevalence has been in the area of the lower reach of the Yellow River. Reasons for the high incidence of BCS in this area of China and in East Asia in general are worthy of further investigation. The study conducted in Dongping County, China, seems to be the only epidemiological study available to date. It might be a good idea to conduct an internal cooperative study on this aspect. Perhaps that will be one outcome of the Second International Symposium on Budd-Chiari Syndrome in Kyoto, Japan, October 6–9, 1991.

Etiology

Etiology of BCS in Western countries is usually thrombosis or hypercoagulable status. In South Africa, an alarming incidence of 47% (48 out of 101 cases) of hepatic cellular carcinoma was reported in patients with Budd-Chiari syndrome.[20] In Eastern countries, however, the predominant cause is webs or membranes of the IVC, especially noted in Japan, China, and India.[6,8,21–23] This author's series reported 40.8% with membranous

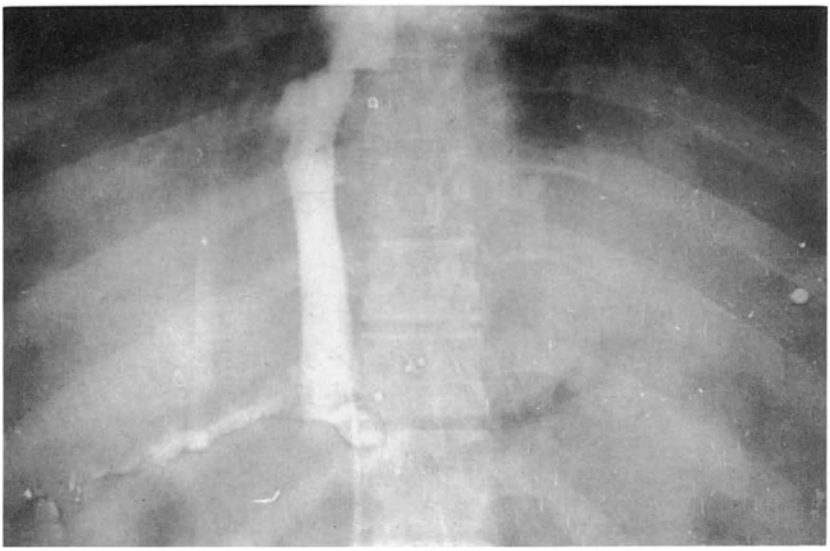


FIGURE 38.4. Cavography showing a malalignment of the IVC.

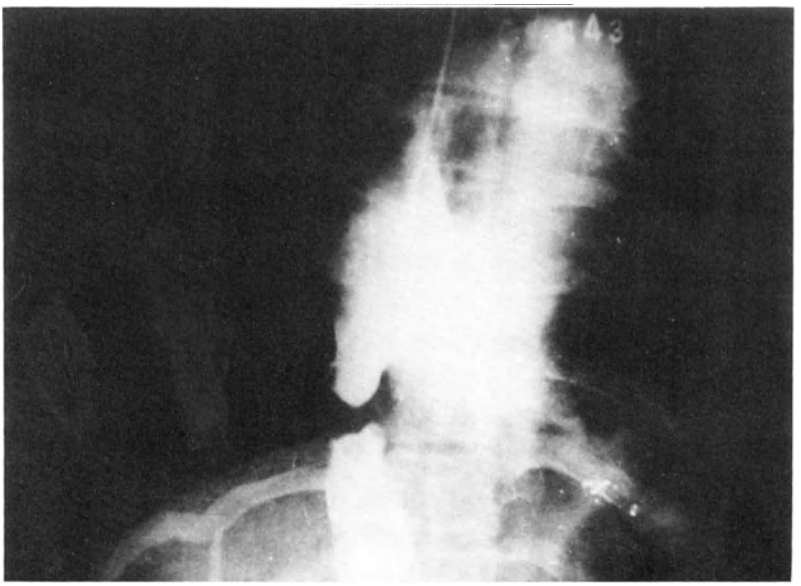


FIGURE 38.5. Bidirectional vena cavography showing a membranous obstruction of the IVC.

obstruction of the IVC. The majority of these membranes were located at the level between the 8th and 9th thoracic vertebrae. Fig. 38.4 shows a malalignment and Fig. 38.5 and 38.6 show a thick membrane and a dome-like membrane with striking dilatation of the IVC in which filling defect (thrombus) is revealed. Fig. 38.7 shows an autopsy sample in which a diffuse thrombus occupies the entire lumen of the IVC and a membrane is located just on the top of it. Figure 38.8 indicates different appearances and corresponding numbers of cases of IVC webs found in the author's series. One or more holes were present in 12 cases. Two brothers in the same family suffered from IVC webs. Evidence from all these cases support the congenital etiology of IVC webs.

Pathogenesis of IVC membranes may originate from: 1) a previous sieve-like obstruction of the IVC, in which the holes gradually become sealed as the body grows and develops; 2) failure of amalgamation of the right subcardial vein with the common HVs during the embryonic stage;[24,25] 3) malalignment of embryonic IVC blocks; 4) malformation of the Eustachian valve;[21,24] 5) the possible similarity in accordance with formation of the congenital atresia ani, biliary tract atresia, hymenal atresia, and other congenital lesions;[26] or 6) a process of obliteration of the ductus venous may extend into the IVC, producing obstruction of the IVC.[7] Needless to say, in addition to IVC malformation, any other pathological entities that may cause hepatic outflow disturbance, whether in the

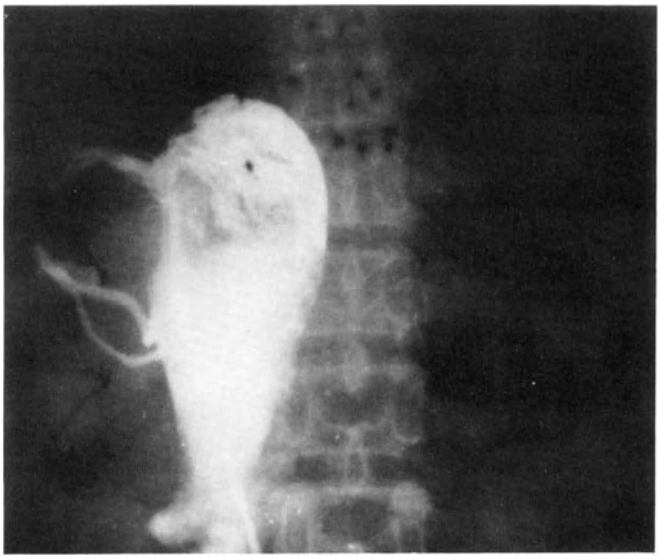


FIGURE 38.6. Cavography showing a dome-like IVC web with thrombus formation in the strikingly dilated distal IVC.

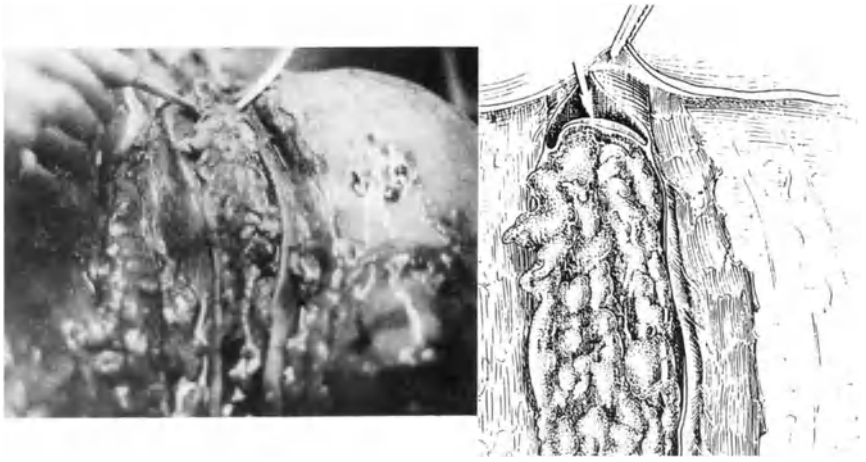


FIGURE 38.7. Autopsy specimen (L) and artist's drawing (R) showing a diffuse thrombus formation in the IVC. A web is seated on the top of it.

vessel lumen, the wall, or from external compression, may also cause BCS,[26–33] such as neoplasms, hypertrophied caudate lobe of the liver, amoebic abscesses, sarcoid granulomas, nonspecific vasculitis, contraceptive-induced hypercoagulable state, pregnancy, myeloproliferative dis-

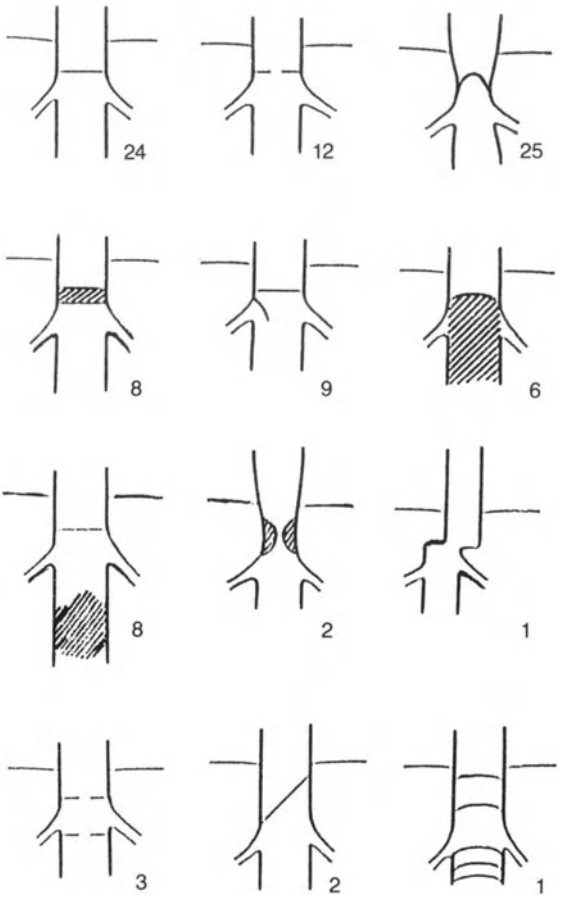


FIGURE 38.8. Diagram showing 12 types of webs or malformations of the IVC.

ease, polycythemia rubra vera, paroxysmal nocturnal hemoglobinuria (PNH), antithrombin III deficiency, lupus anticoagulant, certain plant alkaloids,[34] and even parasites such as filariasis,[26] etc.

Animal Study

The successfully created animal model of BCS and its smooth relief either by cavoatrial shunt or balloon dilatation[35] gave confidence in handling such a bizarre and awkward disorder. Achievement of rapid endothelialization,[36] improvement of patency, and inhibition of neointimal hyperplasia of the IVC shunting Dacron graft by high density seeding of omentum-derived, autogenous Factor VIII, antigen positive ECs in dogs[37] may bring about a remarkable improvement in venous reconstruction, especially

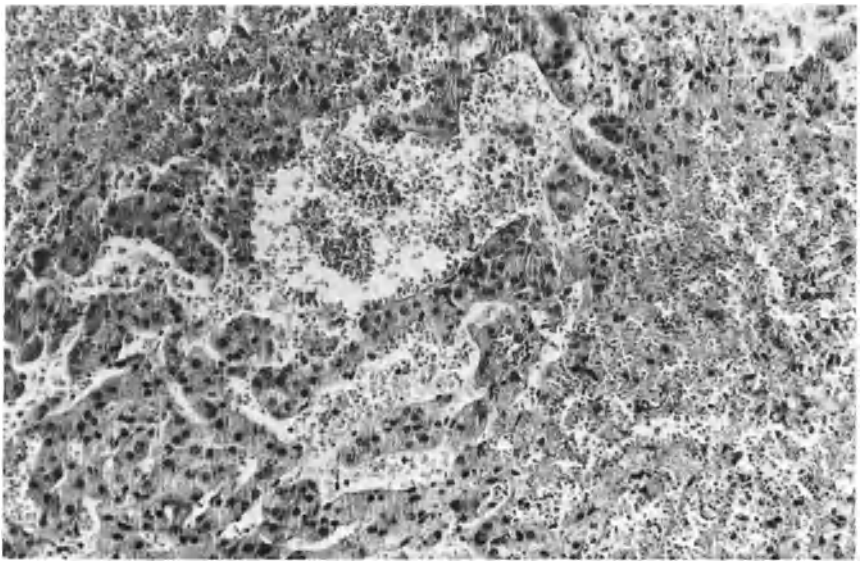


FIGURE 38.9. Micrograph of a liver biopsy showing distinctly dilated and erythrocyte-congested central venules and sinusoids of the liver, characteristics of BCS (×60).

when supplemented by the external ring device and anastomotic retainer. The venous prostheses most suitable for endothelial cell seeding are the high porosity Dacron (including gel impregnated ones) and the PTFE grafts.[38]

Pathophysiology

The main pathophysiological factor causing BCS is an outflow blockage of the sinusoidal beds of the liver with characteristic dilatation and congestion of the central veins and sinusoidal areas of the liver (Fig. 38.9), resulting in a progressive portal hypertension and IVC hypertension if the latter is also involved. When the blood flow is still constantly entering the liver through the hepatic artery and portal vein but the main hepatic venous outflow blocks, highly hydrostatically pressured hepatic sinusoids are created, so that plasma pours into the Disse's Space and then into the lymphatics. Excessive formation of lymphatic fluid, usually beyond the capacity of accommodation, by increasing lymph flow via the hepatic lymphatics, causes leakage of fluid from the overburdened hepatic lymphatics into the peritoneal cavity through Glisson's capsule. Fig. 38.10 demonstrates the mechanism of a progressive liver congestion and transudation or oozing of lymphatic fluid from Glisson's capsule. This "weeping liver" can often be

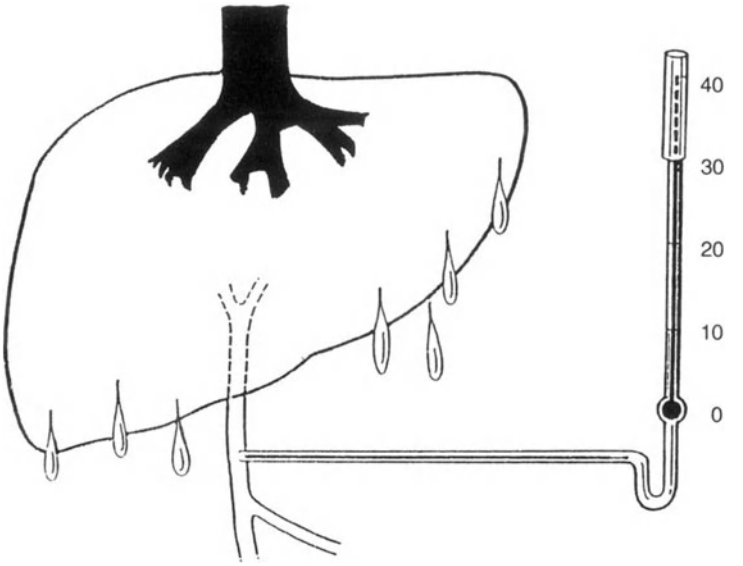


FIGURE 38.10. Diagram showing pathogenesis of BCS: occlusion of IVC-HVs junction and "weeping liver."

seen during laparotomy of patients with BCS; it is one of the main reasons for the usual intractability of ascites formation. Hepatomegaly, splenomegaly, and varices of the esophagus and the cardiac fundus of the stomach are caused by portal hypertension. Simultaneously existing occlusive lesions of the IVC often cause renal dysfunction (reduced renal output) because of renal congestion, dysdigestion because of intestinal congestion, and marked varicose veins on the thoracoabdominal wall, especially on the loin and back, which differs from cirrhotic portal hypertension; also, edema, ulcer(s), and pigmentation of the lower limbs are presented bilaterally. Caput medusae is occasionally seen. Since venous blood is congested in the lower body, returning volume is markedly reduced and heart size is diminished. On X-ray, the patient usually has a small heart, the so-called "rat heart." For this reason, the patient with BCS always has a compromised heart function because of low cardiac output, causing palpitation and shortness of breath on even slight exertion. Tables 38.6 through 38.8[35,39]

TABLE 38.6. Change of cardiac function (N = 32).

Balloon dilatation	SV ml/pulse	CO L/minute
Before	50.3 ± 4.1	4.7 ± 1.9
After	61.4 ± 3.2	5.9 ± 1.4
P	<0.05	<0.05

TABLE 38.7. Change of cardiothoracic ratio before and after mesoatrial shunt.

Time (day)	X ± SD	n
Pre-Op.	0.41 ± 0.06	8
Post-Op. 1	0.49 ± 0.05	8
2	0.54 ± 0.06	6
3	0.56 ± 0.06	6
5	0.57 ± 0.03	5
7	0.58 ± 0.05	5
4	0.59 ± 0.06	3

show changes of stroke volume, cardiac output, cardiothoracic ratio, and parameters of Swan-Ganz catheter before and after balloon dilatation of the IVC web or mesoatrial shunt. Fig. 38.11 shows hemodynamic changes before and after mesoatrial shunt, cavoatrial shunt, and membranotomy.

Tables 38.9 and 38.10[39] demonstrate that blood in the stagnated IVC is rheologically of hypercoagulability and hyperviscosity. At the end stage, due to a dysfunctional digestive system and a constant albumin loss after repeated abdominal paracentesis, patients entered a state of severe nutritional depletion and debilitation (cachexic-like), the limbs as thin as sticks and the abdomen as huge as a big drum. We called it a sign of "spider man" (Fig. 38.12). So long as no measure for correction was undertaken, the patient usually died from massive hematemesis because of ruptured varices of the esophagus, cardiac fundus of the stomach, cachexia due to intractable ascites and malnutrition, or hepatorenal failure. Investigation of 43 liver biopsies of BCS established 4 pathological types: dilatation, congestion, necrosis, and mixed,[40] which are basically correlated with clinical severity.

The webs of IVC were confirmed to be composed of multiple layers of collagenous fibers with mucous degeneration and endothelial cells covering each side.[40]

TABLE 38.8. Changes of cardiac function examined by Swan-Ganz catheter.

Membranotomy	Before	After
IVCP	45	29
RAP	12	18
PAP	8	36
SWIRL	2.27	13.70
SWILV	28.40	59.40
CO	5.50	12.81
CI	3.18	7.42
HR	120	139

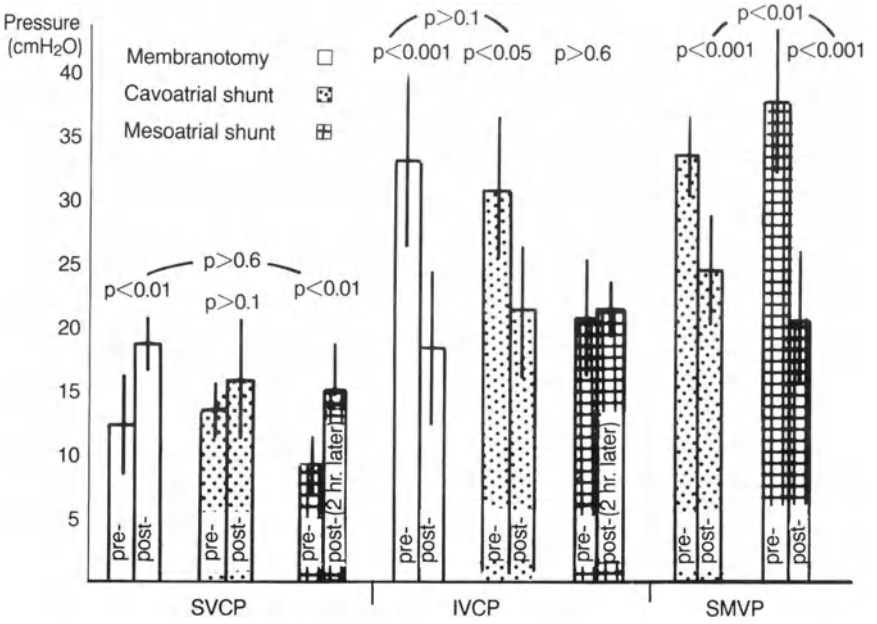


FIGURE 38.11. Histogram showing pressure changes in the IVC, SVC, and SMV pre- and postshunt.

TABLE 38.9. Rheological changes of blood in IVC (N = 32).

Balloon dilatation	Before	After	p
Blood viscosity (mPas)			
307^{-1} (low)	15.67 ± 0.34	13.67 ± 0.2	<0.05
9.6^{-1} (high)	5.86 ± 0.23	4.51 ± 0.17	<0.05

Diagnosis of BCS

For those patients with clues of portal and IVC hypertension, portal hypertension with a relatively normal liver function, or varices on the thoracoabdominal wall (especially on the back, bilateral loins, and lower limbs),

TABLE 38.10. Changes of blood gas in the IVC (n = 32).

Balloon dilatation	Before	After	p
Hb (g/dl)	14.2 ± 0.8	11.7 ± 2.5	<0.05
PVO_2	21.2 ± 6	50.5 ± 17.8	<0.05
$PVCO_2$	44.5 ± 5.5	40.1 ± 13.3	<0.01
SAT (%)	47.4 ± 21.8	74.6 ± 11.5	<0.05

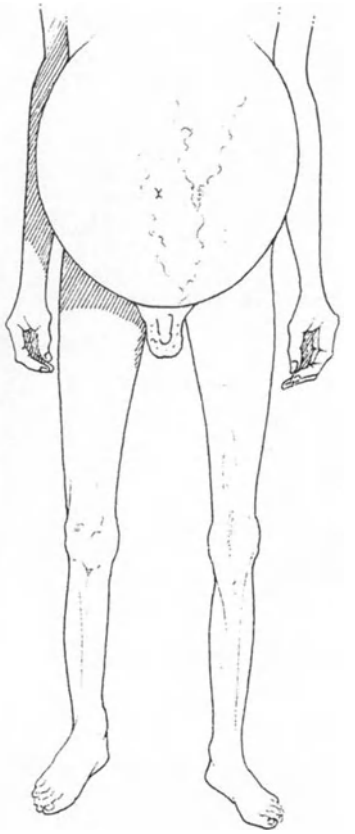


FIGURE 38.12. Artist's drawing showing a "spider man," a clinical sign presented at the late stage of BCS.

diagnosis of BCS should be highly suspected. Screening them with B-mode ultrasonography, a simple, reliable, and convenient non-invasive method, a correct diagnosis rate of more than 90% can be expected.[41,42] However, diagnosis is usually made much later, after a fully clinical picture has emerged.

In order to find patients still in the early stage, a B-ultrasonographic check-up of the IVC and HVs is suggested during any routine health examination. The best means for diagnosis has been cavography, in which a catheter is introduced into the IVC through the femoral vein by a Seldinger technique and, if necessary, another catheter is placed in the supradiaphragmatic IVC by catheterization via the brachial vein. While the contrast is injected simultaneously, the occlusive lesion location and extent can be nicely visualized[43] as demonstrated in Figs. 38.13 and 38.14. Patency of the HVs is always of paramount significance in selection of a suitable sur-

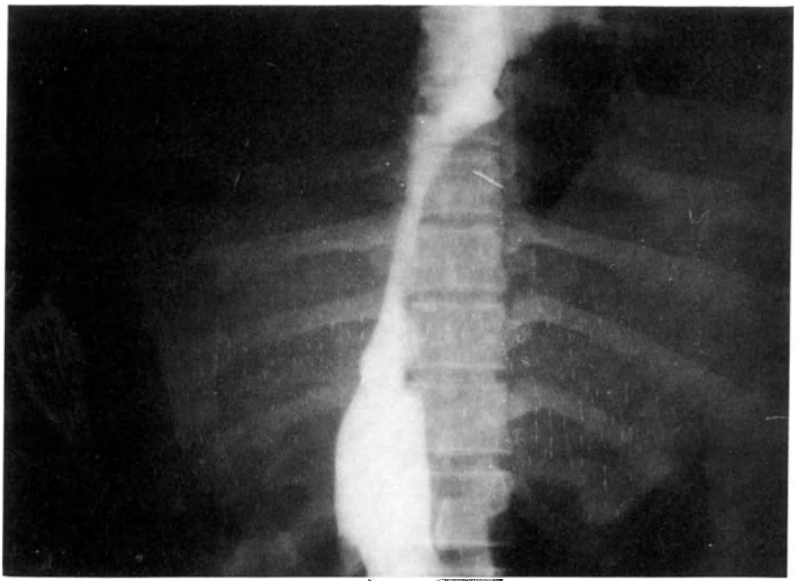


FIGURE 38.13. Cavography showing long-segment constriction of the IVC (Type V).

gical procedure for patients with different pathological configurations. Percutaneous transhepatic hepatophlebography is specially devised for this purpose,[44] of which Fig. 38.15 is an example. CT and MRI have been claimed to be useful in diagnosis of this entity, but they are not as critical as angiographic studies.

The importance of reaching a final diagnosis and a clear pathologic classification is in facilitating selection of an operative procedure. Fig. 38.2 shows this information and Table 38.4 shows a gradation of BCS that helps to predict surgical outcome and operative risk.[3]

Preoperative Support Means

The patient's general condition may be very poor, such as under massive hematemesis, intractable ascites, and/or severe nutritional depletion (cachexic appearance), and/or hepatorenal failure. For the former, all necessary means for emergency surgery should be prepared in addition to employing sclerotherapy for esophageal varices. Our experience includes repeated transfusion of autogenous ascites. After 3 to 10 preoperative transfusions from 500 ml to 2500 ml of ascites (filtered twice under strict sterilized condition) in each of 40 cases, all patients had their intractable ascites relieved and in 3 of them, the ascites almost disappeared before

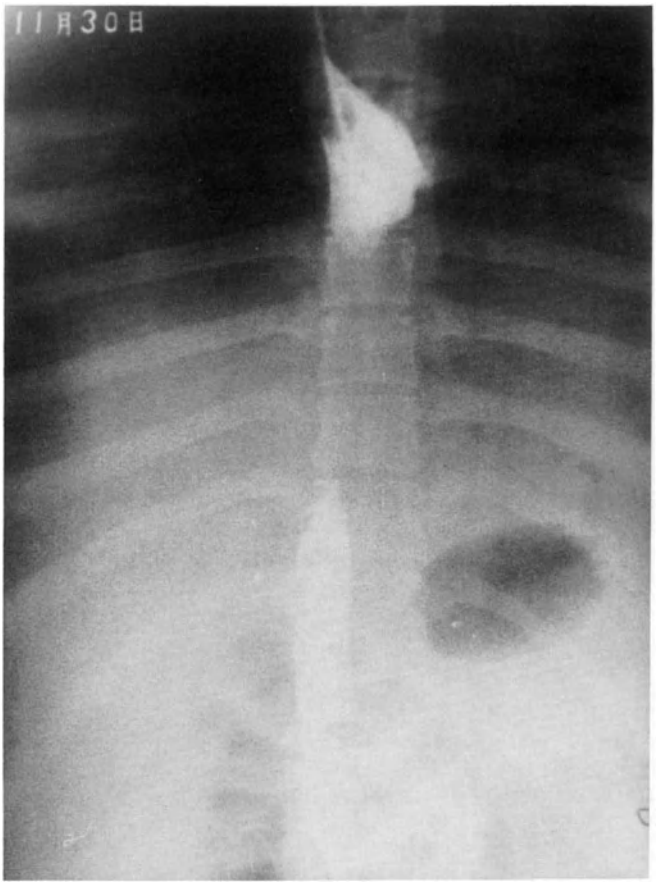


FIGURE 38.14. Cavography showing long-segment constriction of the IVC (Type VI).

surgery. For cachexic patients, gastrointestinal nutrition was adopted and marked beneficial effect was obtained in 4 cases. Chinese herbal medicine was also used in order to improve liver function and general condition of the patients. Thus the preoperative treatment always took a month for severe cases and was effective in reducing operative mortality and promoting the postoperative course of recovery.[45]

Conservative Therapy

In the author's series, a mortality of 87% was recorded in Stage IV patients.[3,23] An additional 48 cases who received conservative therapy were collected before early 1988 in Chinese literature. Among them, 39.6% died, 43.8% were unimproved, and 16.7%, who were treated

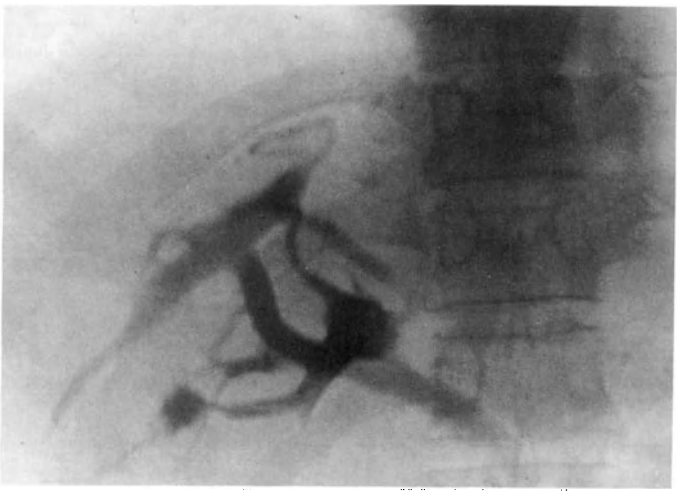


FIGURE 38.15. Hepatovenography showing marked dilated HVs with collateral formation.

chiefly by Chinese herbal medicine, claimed to be improved.[19] In addition to using fibrinolytic therapy for acute onset of BCS due to thrombosis, any means of conservative therapy seemed only to provide patients with time for development of compensating collaterals; but the remaining occluded lesions were unrelieved, which is definitely a clear reason for the bad results of conservative therapy. Nearly 80% of this author's effective results were achieved by surgery. Surgical treatment is definitely superior to conservative treatment. So long as surgical conditions are available for both medical teams and patients, the surgical approach (including transluminal balloon technique) should be adopted as early as possible.

Surgical Means

Often, only major surgical procedures can eliminate or bypass lesions in HVs or retrohepatic IVC. The complicated vascular anatomy in this area, including a profusion of strikingly dilated and thin-walled, frail collateral vessels, which present a strong risk of bleeding, creates an especially intolerable circumstance for patients who are also high operative risks and have various pathological configurations and complications.

Minor Surgery

Thus, in the early period of our work, facing severe cases of BCS with a great quantity of ascites (including one whose abdominal girth was 159 cm and whose fingers were unable to touch his own umbilicus), we tried minor

operations that theoretically might be tolerated, such as the thoracic duct-jugular vein reanastomosis, so as to create a wider orifice for enhancing lymphatic clearance from the thoracic duct and possibly to relieve portal hypertension. However, among the 5 cases who underwent such surgery, only one obtained marked relief. Peritoneojugular shunt or LeVeen shunt was also used at the beginning of this study. However, portal hypertension was not relieved, the shunt could not function for long, and symptomatic relief did not last long. In one case, the subcutaneous tube was partially exposed to the exterior from the thoracic wall as an infected foreign body waiting for extraction. Cephalic vein–peritoneum or saphenous vein–peritoneum anastomosis served as another minor alternative. These anastomoses were very nicely and smoothly created, but the ascites reduction effect lasted only for a few days. The autogenous vein graft is thought to be much better than any kind of artificial shunting tubes, but an omental blockage might easily occlude the vein–peritoneal anastomosis and cause failure. The cephalofemoral venous shunt, in which the cephalic vein was isolated and brought to the groin under the thoracoabdominal wall subcutaneously, was employed in one patient with predominantly IVC hypertension, resulting in a slight relief of edema of the lower limb. It seemed that all the minor, or relatively "safe" procedures, were not able to cure or even markedly relieve severely ill BCS patients.

Membranotomy

Membranotomy (Fig. 38.16) is a relatively simple procedure. A right anterior thoracotomy through the 4th intercostal space or a median sternotomy is employed; the pericardium is incised anterior to the right phrenic nerve, and the right atrium can be better exposed by pericardial traction and proper shift of bed position. The intrapericardial IVC segment is first isolated and encircled with a tape for traction. Inasmuch as it may be a size-diminished heart with an incompletely filling right atrium, a properly sized, side-walled clamp should be selected and applied on the right atrium close to its bottom without compressing the SVC inlet opening, the right coronary, and the sinoatrial node. A purse string suture of a size allowing finger insertion using 4-0 Prolene is prepared on the clamped part of the atrium. Two ends of the suture, after detachment of needles, are trapped into a vascular keeper. The atrial wall encircled by the suture is properly opened without damaging the suture, otherwise a bigger string purse suture is substituted. The left middle finger of the operator is inserted while the clamp is withdrawn and the vascular keeper is properly tightened. No bleeding was caused while doing this. By traction of the tape encircling the IVC, the finger is guided into the IVC rather than the tricuspid valve. The fingertip progresses further and the membranous obstruction or IVC web (be sure it is not the bottom wall of the atrium) can be touched at a depth of about 5 centimeters. The web felt by fingertip was like a layer of strong

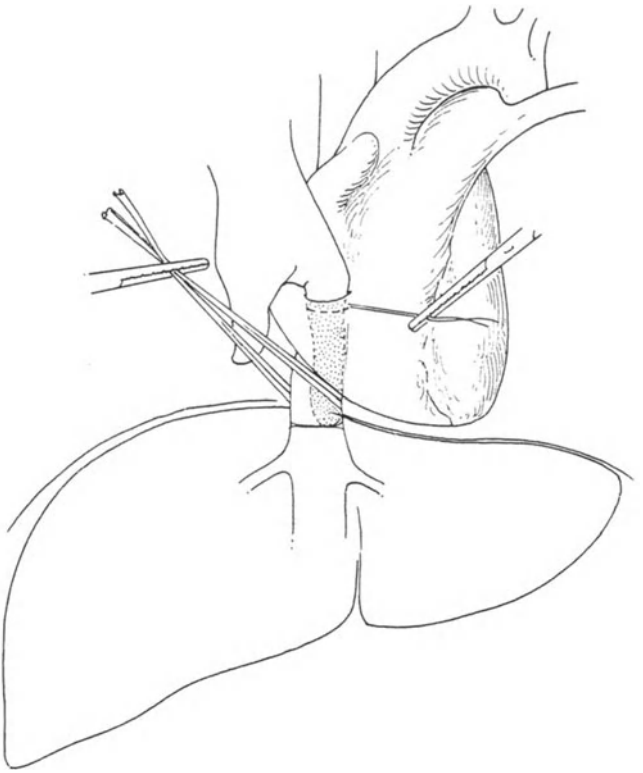


FIGURE 38.16. Artist's drawing showing the transcardiac technique for membranotomy.

cloth with fairly good elasticity which prevents it from being broken. The penetrating maneuver is repeated until the web is fractured. The full finger is then inserted into the IVC and a dilatation manipulation is also repeated if localized stenosis of the IVC is also present. The orifices of the HVs can be perceived in the majority of our patients, and membranous obstruction of the HVs were found in 7 cases during this procedure. All were broken and dilated by fingertip. This technique succeeded in most of our patients. However, in those with "dome-like" or thick web, or those with marked accompanying stenosis of the IVC, a dilatation maneuver is sometimes assisted by a large size (>2 cm ID) balloon catheter or a self-made balloon catheter inserted from the right atrium or the femoral vein by Seldinger's technique, which is always beneficial. Sometimes, after a successful membranotomy, an immediate failure will be perceived during the reclosing process of the involved part of the IVC, following immediately after withdrawal of the operator's finger. In these cases, other alternatives should be adopted. Membranotomy is not indicated in those with fresh thrombus in

the IVC distal to the lesion in order to prevent life-threatening pulmonary embolism following membranotomy. The disadvantage of this procedure is that after a successful membranotomy, the broken and irregular residual membrane is still there, the coexistent stenosis of the IVC may also remain, and the external compression may not be changed. All of these factors are likely to cause reocclusion of the IVC, a condition usually much more difficult to handle than before. There were 3 membranotomy failures and 7 membranotomy recurrence cases in this series. As demonstrated in Fig. 38.3, 1 year following membranotomy, the patency curve seems stable.

Transluminal Balloon Membranotomy

This is a nonsurgical means to treat localized lesion of the IVC. Because of the difficulty in obtaining high quality Grunzig or Inoue catheters, this procedure has been conducted in only 21 cases in this series. This method has the same disadvantage as in the finger membranotomy. In addition, complications, such as cardiac tamponade, fracture of the catheter, and pulmonary embolism, might be incurred. We suggest this procedure for patients with localized lesions, but it is not indicated for those with fresh thrombus distal to the lesion. The author was informed of 2 mortalities occurring during 10 such procedures conducted in another hospital. The method and its results are shown in Fig. 38.17.[46]

Transluminal Laser-Assisted Angioplasty

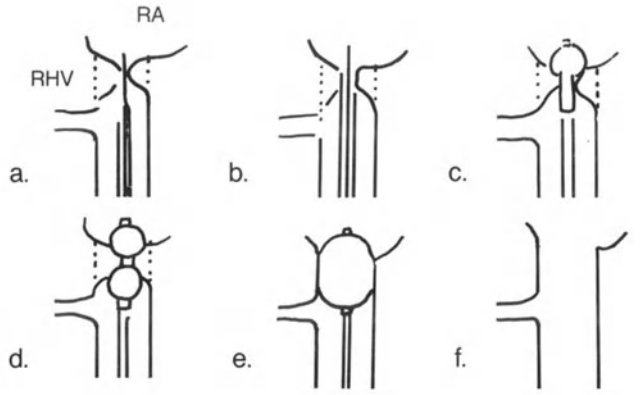
This procedure was reported to be used in 3 cases in whom conventional means were not successful.[47] This procedure was carried out in one case in Jinan, China. However, the fiberoptic laser tip was broken and retained adjacent to the IVC lesion, which required the author to perform a radical operation to extract it.

Percutaneous Transhepatic Recanalization and Dilatation

This procedure was reported recently in one case with BCS.[48] The author considered it useful in treating some selected cases with occlusion near the orifices of the HVs and tried it once.

Cavoatrial Shunt

For the failed membranotomy cases, an immediate decision to shift to use of a cavoatrial shunt (Fig. 38.18) should be made during surgery. The high failure rate of venous prostheses due to thrombosis is an obviously substantial obstacle. The reasons for the operation, in addition to patient requirements, are: 1) large caliber grafts are better at preventing thrombus



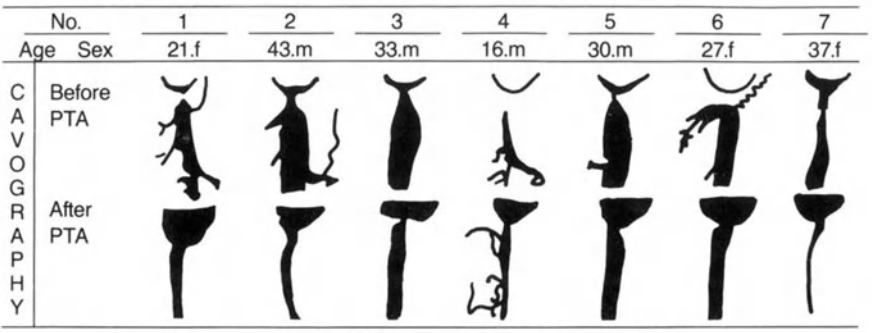


FIGURE 38.17. Diagram showing the balloon dilatation (PTA) technique (above) and its resolving of 7 cases (below).

formation than small ones; 2) pressure gradient between the IVC and the right atrium is significant; 3) pressure in the abdominal cavity is positive while that in the thoracic cavity is negative; and 4) venous flow in the cavoatrial graft may be accelerated during regular inspiration, which enhances intrathoracic negative pressure due to expansion of the thoracic cage. All these factors are accredited to initiation of a 1-way blood flow in the shunting graft. In the first case, we used a locally made, rather stiff Dacron prosthesis 22 mm in diameter. The patient had a good recovery after overcoming a very severe heart failure, presumably due to the sudden return of a large quantity of venous blood congested in the lower body of the patient. Later on, the prosthesis diameter was reduced to 20 mm, 18 mm, 16 mm, and even 14 mm. By personal communication, the Gore-Tex graft, in 10 mm and 12 mm internal diameter (ID), was used as cavoatrial shunt material in another hospital but resulted in an early occlusion of the prosthesis. Thus, we advocate the use of 16 mm or 14 mm ID as the suitable prosthesis diameter for this purpose.

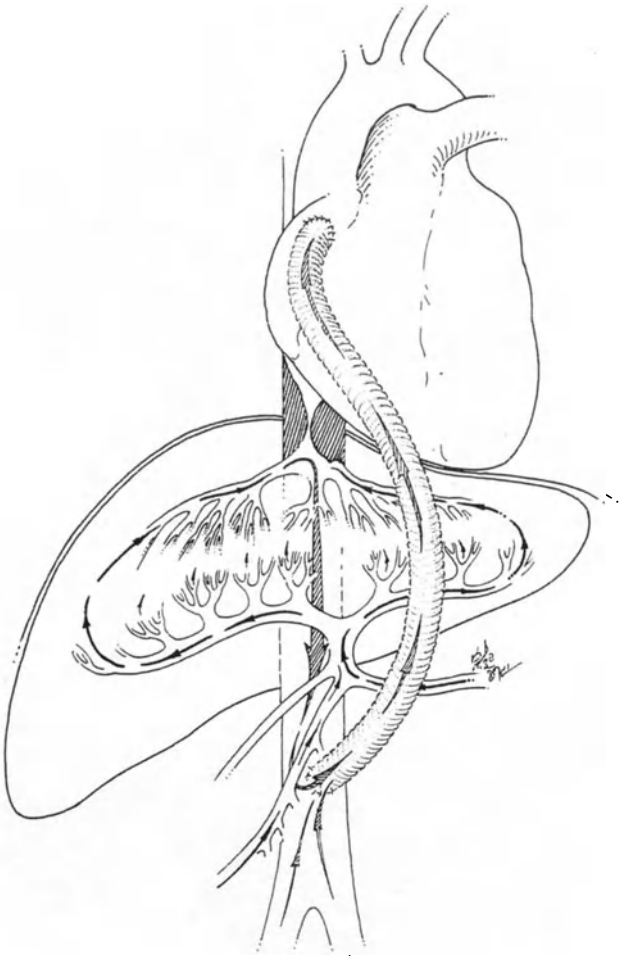


FIGURE 38.18. Diagram showing cavoatrial shunt and its hemodynamic changes (arrows).

Two methods have been used for construction of the cavoatrial shunt: anterior and posterior approaches. For the anterior approach, a midline abdominal or right paramedian incision is made. The IVC is isolated through one of the following routes: 1) Kocher incision, making access from the right lateral peritoneum, and mobilization and traction of the duodenum and ascending colon to the left; 2) dissection of the retroperitoneum from the right to the upper part of the mesentery (part of the IVC is isolated from beneath and below the transverse portion of the duodenum while the transverse colon is elevated and the intestine pushed to the left); or 3) isolation from the left to the mesentery and from the right to the

abdominal aorta while pushing the intestine to the right. As many vein collaterals and lymphatics are diffusely distributed in the retroperitoneum because of longstanding IVC occlusion, the dissection maneuver on the retroperitoneum should be carried out very carefully, suture-ligation should always be used, and some of the lumbar branches need to be ligated and severed. Preparation of a segment of the IVC greater than 4 cm in length is necessary for anastomosis. For those with a large quantity of ascites, a median sternotomy is advised; otherwise a right anterior thoracotomy at the 4th intercostal space with division of the 3rd anterior rib is used. The pericardium is incised and fully tracked for better exposure and manipulation. If a thoracotomy is selected, a hole in the diaphragm is made at a proper position for passage of the graft. The preclotting process, using autogenous blood conducted from outside the graft so as to reduce the intraluminal clot, is completed (if it is required). The graft is tailored into a cobra shape. A suitably sized Satinski Clamp is applied on the IVC, 1000 units of heparin are injected into the IVC distal to the clamp. An end prosthesis-to-side IVC anastomosis is completed using 5-0 Prolene suture in an everted running fashion. If technical difficulty is expected, a "parachute" anastomotic method is selected. A spiral stainless steel external support is used if the graft has no external support. The graft is usually brought to the thoracic cavity or mediasternum posterior to the transverse colon and anterior to the stomach and liver. The upper end of the graft is tailored and an end prosthesis-to-side atrium anastomosis is completed with 4-0 Prolene suture in an everted running fashion. When the anastomosis is performed, the self-made anastomotic retainer is applied in each anastomosis. Two needles are inserted into the graft, the distal one used for installing heparin normal saline solution and the proximal one for evacuating entrapped air in the graft. The clamps applied on the IVC and atrium are withdrawn respectively. The graft is thus functional. An immediate relief of the hepatosplenomegaly and reduction of portal pressure will result if the HVs are patent to the IVC. Thoracic drainage is instituted, bleeding stopped, and the incisions are closed in layers. Figure 38.19 shows the follow-up study by cavography.

Posterior Approach for Cavoatrial Shunt

A left-sided position is undertaken and a posteroanterior thoracotomy through the 7th costal bed is made. The pericardium is incised, the intracardial IVC segment exposed, and the right diaphragm with the underlying liver is pulled to the left by traction. The right phrenic nerve is protected. Along with the IVC, the diaphragm is carefully incised, beginning from the aperture of the IVC down to expose the dilated or relatively normal segment of the IVC, usually over 6 cm in length. The bypass is constructed between the distal dilated segment of the IVC and the right atrium or supradiaphragmatic IVC. The shunting graft is definitely shorter than the

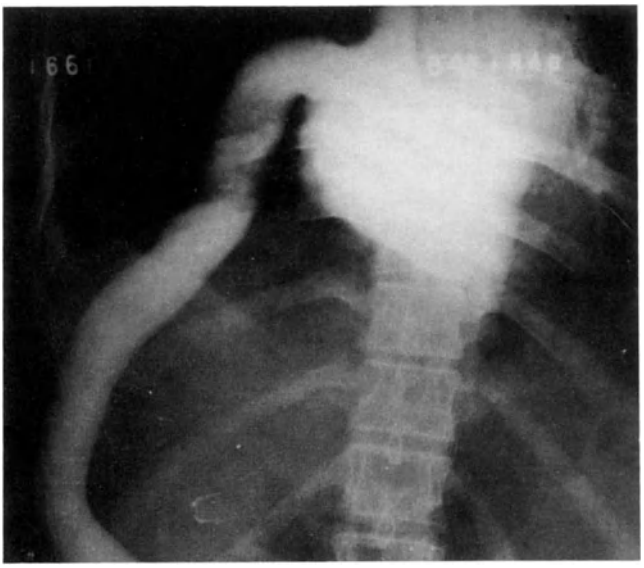


FIGURE 38.19. Cavography showing good patency of a cavoatrial shunt 3 years following surgery.

anterior approach, but the posterior approach is technically more difficult, the chance for cumbersome bleeding is more common, and the incidence of postoperative chylous thorax is relatively higher. Once again, it is important to confirm patency of HVs to the IVC (at least an obviously enlarged short hepatic vein, usually the one coming from the right lower part of the liver to the IVC) in order to obtain a good result. As demonstrated in Fig. 38.3, long-term patency of the cavoatrial shunt is not better than membranotomy.

Mesoatrial Shunt

When occlusion of the IVC involves most or all the IVC, or goes even beyond the common iliac veins, the mesoatrial shunt (Fig. 38.20) is the most suitable indication and the cavoatrial or ilioatrial shunt is precluded. Thus our candidates for this operation usually have poorer general condition, higher risk for surgery, and more predominant clinical manifestations of portal hypertension than those for cavoatrial shunt. Surgical manipulation is also more difficult than the cavoatrial shunt. The main problem is preparation of the superior mesenteric vein (SMV) located beneath the distinctly thickened, tender, and edematous retroperitoneum full of venous and lymphatic collaterals so that a proper anastomosis may be constructed on it. The upper midline abdominal incision is made, viscerae ex-

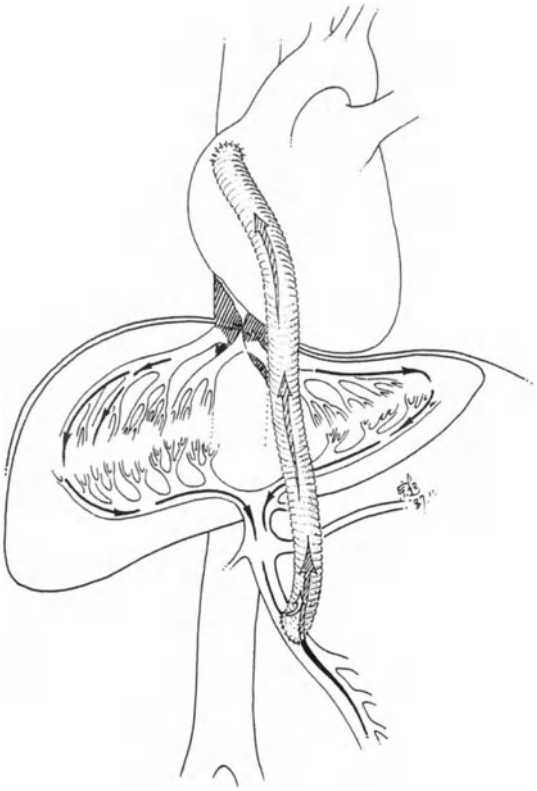


FIGURE 38.20. Diagram showing a mesoatrial shunt and its hemodynamic changes (arrows).

plored, ascites aspirated, portal pressure measured, and SMV isolated at the base of the mesocolon through the retroperitoneum right to the Treitz's ligament while the transverse colon is in traction upward and the intestine pushed away from the operative field. Dissection of at least 4 cm of the SMV (usually with a large right branch included) below the pancreatic body is required. The graft needs to have an external right support and should be 14 mm or 16 mm in diameter. The right atrium is exposed through the right anterior thoracotomy at the 4th intercostal space if the patient had slight ascites or through the medial sternotomy if the patient had remarkable ascites. Graft end-to-side SMV and graft end-to-side right atrium anastomoses are completed as is done in the cavoatrial shunt. After establishing blood flow through the graft, rapid relief of hepatosplenomegaly can be expected, i.e., reduction of liver size and shrinkages on the liver surface can immediately be noticed, a remarkable reduction in spleen size can also be felt easily, and usually a 40% to 50% reduction of

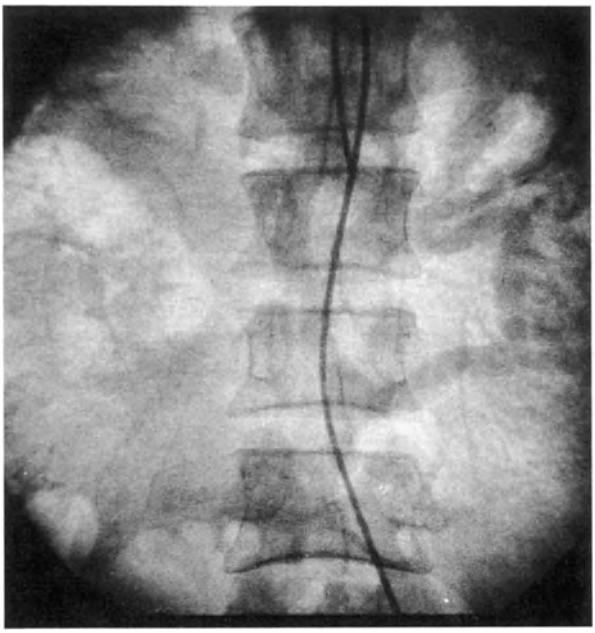


FIGURE 38.21. The venous period of superior mesenteric arteriography showing a patent mesoatrial shunt (arrow) 4 years following surgery.

portal pressure can be measured. There is a 10% chance of controllable encephalopathy and a higher operative mortality compared to the cavoatrial shunt, which is related to the different portal flow pattern (Figs. 38.18 and 38.20) and to the selection of patients. However, this procedure is highly recommended since it directly and effectively relieves portal hypertension, a leading cause of deterioration or death of patients, and it usually brings very severe patients a surprisingly nice recovery.[49] Attacks of encephalopathy can easily be avoided by dietary caution. As demonstrated in Fig. 38.3, this long-term patency is the best among the 3 procedures; however, the operative mortality is higher than the others because patients selected for this operation are in more severe condition. Figure 38.21 shows graft patency at the venous stage of superior mesenteric arteriography.

Splenoatrial Shunt

This serves as an alternative when use of the SMV is unavailable. In one case in this series, a mesocaval side-to-side anastomosis had been performed 2 years prior to admission to our hospital with a chief complaint of repeated massive hematemesis. Venacavography demonstrated a long-

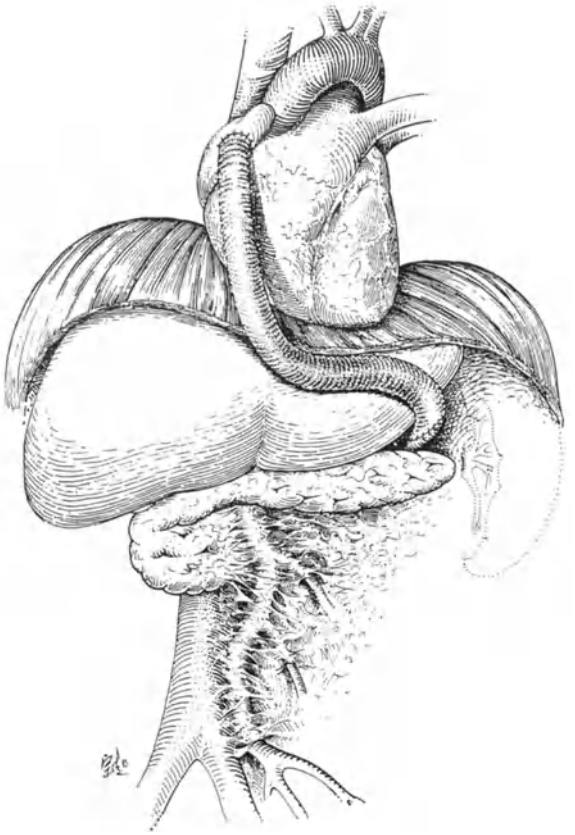


FIGURE 38.22. Artist's drawing showing the splenoatrial shunt.

segment, occlusive lesion of the IVC. During surgery in June 1985, it was found that the SMV could not be dissected because of extensive tense adhesions. A splenectomy and a splenoatrial shunt (Fig. 38.22) with a Dacron graft 16 mm ID were carried out, resulting in an excellent outcome that was followed up for more than 5 years. This operation was performed in 7 more cases in other hospitals, resulting in 5 good results and 2 operative deaths.[50] Splenoatrial shunt is definitely worth recommending and promoting. The portoatrial shunt, based on the same principle, has already been reported in the literature[16] and can be reserved as another option for future use.

Mesocavoatrial Shunt

Everyone likes the lesions to be corrected thoroughly and completely. It is therefore not surprising to consider an idea which might simultaneously

solve both the portal hypertension and the IVC hypertension, both of which existed in most patients with BCS in this series. Chapman reported an iliac-mesenteric shunt procedure.[51] Three of our patients who were in the very-end stage underwent this combined procedure. All died shortly following surgery, probably due to an intolerably sudden return of an extremely large quantity of venous blood that had been congested in the lower part of the body in addition to their very poor condition generally. We have found from the results of our cavoatrial shunt or our mesoatrial shunt that they basically fulfill the operative goal. During follow-up we found that less than 10% of patients had symptoms due to an insufficient decompression of the other system in which no shunting was constructed. We suggest that, when necessary, an additional shunting can be done at a later time, for what we call a staged operation, which is much safer and more feasible.

Mesojugular Shunt

The most severe patients, with distinct malnutrition, massive ascites, and aggressive pleural effusion requiring thoracoparacentesis once or twice a week, cannot tolerate any of the major procedures. We developed a mesojugular shunt (Fig. 38.23) that does not require a thoracotomy or median sternotomy. Only a midline abdominal incision and a small, low, transverse neck collar incision are necessary. The SMV is isolated by the first maneuver and the right internal jugular vein is then isolated; the externally ring-enforced graft, 14 mm or 16 mm ID, is brought into the neck incision retrosternally (in 4 cases) or subcutaneously (1 case); the graft-SMV or graft-jugular end-to-side anastomoses are constructed, respectively. After establishment of blood flow, a portal decompressive effect is also immediately achieved. The ascites and pleural effusion disappeared in 2 to 3 weeks postoperatively in all cases. This outcome, even though still a short-term one, has been surprisingly better than that of those who underwent the mesoatrial or caval–atrial shunt, for whom the recovery period has usually been 3 to 8 weeks. It appears that there is a constant pumping mechanism, produced by a continuously rhythmic and strong cardiac action, which keeps the blood flowing only in an upward direction, i.e., through the mesojugular shunt, because of a significant pressure gradient between the portal and jugular veins. To install a pump in a mesojugular graft by putting it retrosternally seems a unique and effective way to improve performance of the graft. To our knowledge, this method has never been used before. The fifth case (in fact, she was our first case) died of hepatorenal failure 2 months after surgery, presumably because this mesojugular graft was set subcutaneously without establishment of a pumping mechanism, as compared to the retrosternal approach, and without aspiration mechanism created by the thorax cavity during the constantly rhythmic inspiration as seen in cases with the mesoatrial or cavoatrial shunt. Therefore, the subcutaneous mesojugular shunt should be abandoned.

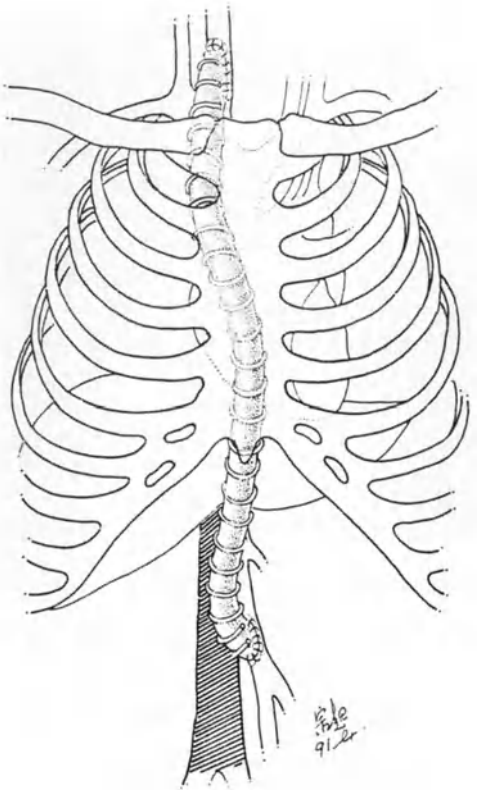


FIGURE 38.23. Artist's drawing showing the retrosternal mesojugular shunt.

Mesocaval and Splenorenal Shunts

These shunts are indicated for patients with classical BCS, i.e., those with intrahepatic venous occlusion and patency of the IVC. Actually, there is no substantial difference from the treatment of portal hypertension caused by liver cirrhosis. Graft patency was nicely demonstrated by cavography through graft catheterization via femoral vein (Fig. 38.24).

Innominate-Atrial Shunt

This can be supplemental for patients with BCS associated with SVC occlusive syndrome. There were 3 successful cases in the author's series including 2 combined with mesoatrial shunt and 1 with transcardiac membranotomy. However, if the BCS is caused by IVC web, a pure transluminal balloon dilatation or finger membranotomy can first be instituted because, after resolution of the IVC webs, the SVC syndrome might be markedly relieved through collaterals, mainly the connection between the azygous system and the IVC. Two patients who received balloon dilatation and

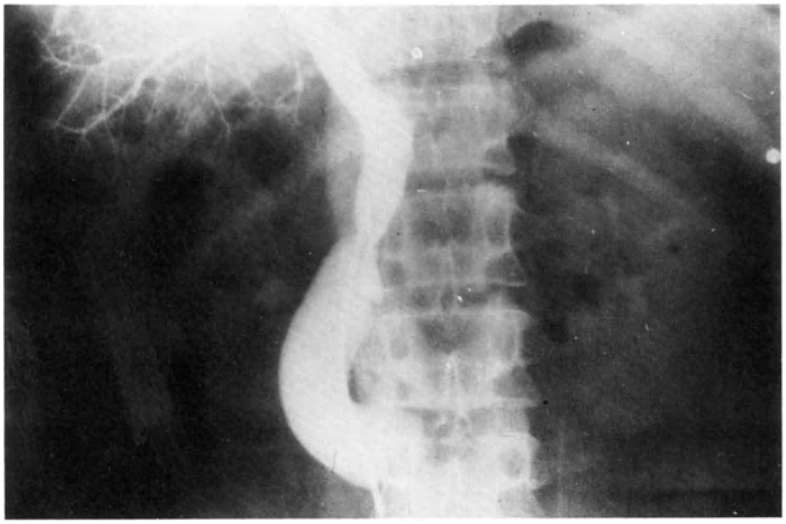


FIGURE 38.24. Cavography showing a patent mesocaval shunt 5 years after procedure.

another 2 who underwent transcardiac membranotomy in this series experienced a marked relief that was quite acceptable to them. If such relief fails to occur, a definitive operative procedure can then be scheduled when it is necessary.

Radical Procedures

None of the above mentioned methods thoroughly eliminate the original lesions, wherever they are located in the IVC or HVs, or both. In order to treat the disease in its original location radically, a radical approach is suggested. So far, we have treated 20 cases radically. A typical right standard posteroanterior thoracotomy via the 7th costal bed is employed. Then, one of 3 methods is used: 1) For patients with a high membranous lesion, the intracardiac IVC and a short segment of infradiaphragmatic IVC are isolated (Fig. 38.25). The web can be felt at that time and after proximal and distal controlling, the IVC is opened longitudinally. The web is clearly identified and completely resected, and the IVC incision carefully sutured. There were 3 cases in this series all resulting in good outcome. 2) The incision extends to the abdomen. A tape encircles the upper part of the IVC. The retrohepatic IVC is isolated and a large size balloon catheter prepared. As soon as the upper IVC and the hepatic triad are blocked, the retrohepatic IVC is quickly opened and the balloon catheter, with a diameter of more than 2 cm, inserted into the distal IVC and inflated with

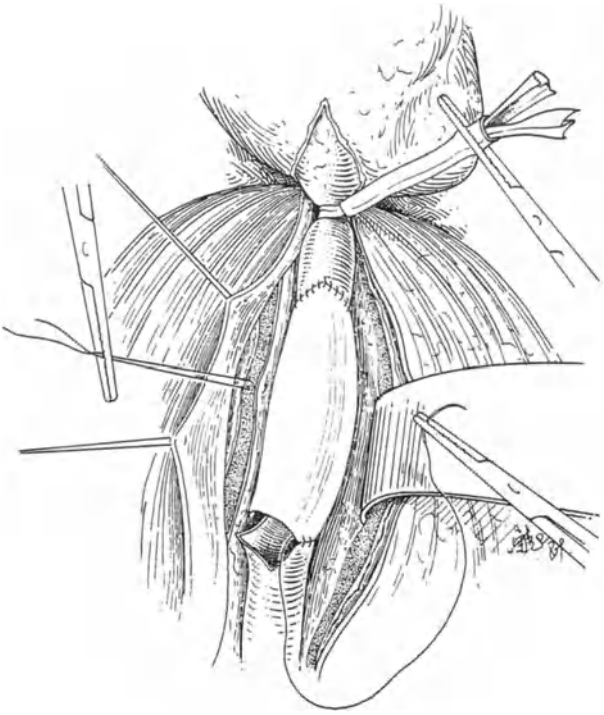


FIGURE 38.25. Diagram showing the radical resection of IVC lesion and patching graft.

heparin saline. The escaping blood is rapidly aspirated for autotransfusion and the operative field becomes clear. The web is carefully and thoroughly resected. The IVC is repaired with Gore-Tex patching and the balloon catheter is withdrawn. It is suggested that the process of web resection and IVC repair be accomplished within 25 minutes so as to avoid hepatic ischemia. We have had only 1 success. 3) For lesion resection under hypothermia and circulatory arrest, extracorporeal circulation is installed in the following way: intubation of the right atrium serves as venous outlet and catheterization of the right iliac artery as the arterial inlet. Until the retrohepatic IVC is nicely exposed, the heart–lung machine functions with cooling. When the temperature is reduced (22°C to 18°C), circulatory arrest begins; the retrohepatic IVC is incised longitudinally, webs of occlusive tissue or thrombus are resected, and the orifices of HVs are explored. Membranous occlusion of the HVs were found in 3 cases in this series (Fig. 38.26) and even a pin-fine foreign body, a broken fiberoptic laser tip located between the diaphragm and the liver, was extracted through radical resection. After a thorough clearing of the IVC, the incision is sutured or patch-grafted with Gore-Tex or Dacron prosthesis, or pericardium if

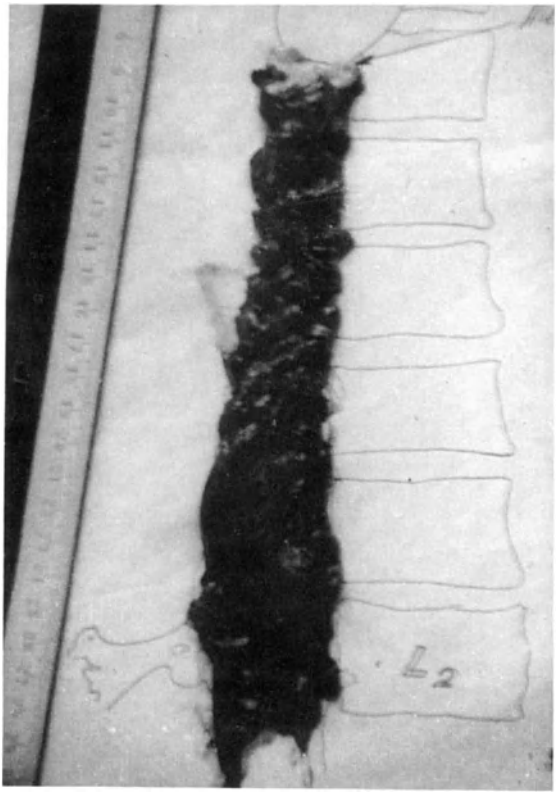


FIGURE 38.26. Photograph showing a thrombus of 19 cm length well established in the IVC between T9 and L2, which was resected with a web under hypothermia and circulatory arrest.

necessary. The heart–lung machine resumes with rewarming until 37°C is reached. The pump stops and intubations are weaned. After bleeding is well controlled, drainages are positioned and the incision is closed. We find now that the radical operation enables us to resect lesions completely. However, it is sometimes unable to prevent recurrence, especially in dealing with a relatively long or constricted IVC with chronic inflammation, in using the pericardial tissue as patching material, and in a situation of local tension usually caused by an external compression of the enlarged liver. In the latter case, use of an appropriate cutting from a ring-supported graft as patching material is helpful in preventing the graft from being compressed due to external lesions. B-mode ultrasonography demonstrates changes in IVC occlusion before and after surgery (Figs. 38.27 and 38.28).

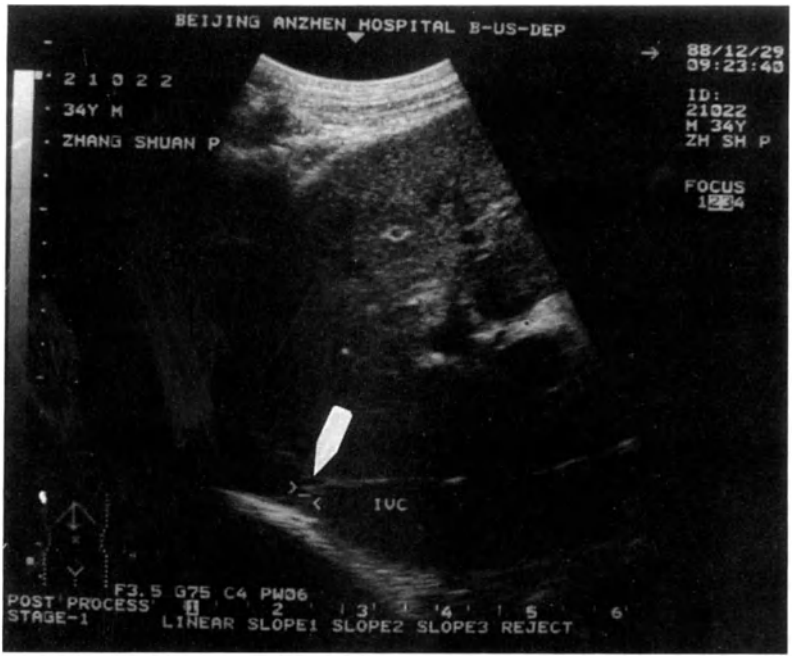


FIGURE 38.27. B-mode ultrasonography showing a marked localized stenosis of the retrohepatic IVC (arrow).

Cavoazygous Shunt

This procedure might serve in some situations when the surgeon is unwilling to do an operation involving the heart. Only one case is available in this series. In the literature, only Yamamoto reported this procedure.[9]

Graft Materials and Other Supplements

Obviously, we are dealing with large-caliber venous prostheses. A rigid graft is required, as was used in early work of this study. The external ring-support graft, including Gore-Tex, Vascuteck, and locally made PTFE are suitable. Otherwise, the spiral stainless steel external supporting device we designed is required to cover the outside of the graft in order to prevent it from being compressed. The EC seeding technique is of benefit,[37] and so far, the author has employed this technique in 10 shunting grafts for treating patients with BCS. The anastomotic retainer we designed is helpful in enlarging the anastomoses and in maintaining their patency; thus they are recommended for venous reconstruction.

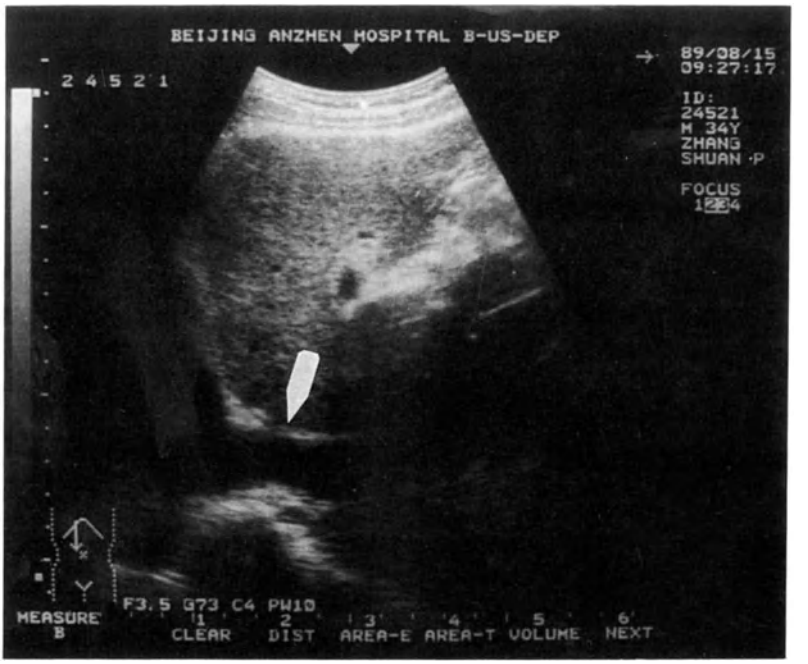


FIGURE 38.28. The stenosis shown in Fig. 38.27 has disappeared after radical resection of the lesion and the affected IVC is widely patent (arrow).

Summary

This paper presents the author's epidemiological and etiological studies, the creation of a dog BCS model and methods of relief, high density EC seeding of prostheses, and clinical experience in 250 patients with BCS.

BCS seems less rare than we previously thought.[52] A geographical prevalence is found in some areas of China. An animal model, with dogs, can be reliably created and relieved by the proper means. IVC webs are congenital and are the main cause of BCS in China. Eight pathologic classifications and 4 gradations are suggested.

Suggested therapeutic approaches: 1) Balloon dilatation is suggested as the first treatment for those with IVC webs, localized stenosis, or IVC occlusion, but without new thrombus distal to the lesion. 2) Transcardiac membranotomy is employed for those with the same indications, especially for failure cases. 3) Cavoatrial shunt is used for those with HVs patency to the IVC. 4) Mesoatrial shunt is indicated for those with diffuse stenosis or occlusion of IVC and occlusion of HVs. 5) Portosystemic shunts are used for those with intrahepatic venous occlusion. 6) Mesojugular shunt is suitable for those with intractable ascites, pleural effusion, and high operative

risk. 7) Innominate-atrial shunt can be supplemental for those with accompanying SVC syndrome. 8) Radical resection is best indicated for those whose lesion requires resection. 9) Liver transplantation is reserved only for end-stage patients.

Suggested venous graft materials: The externally ring-enforced, internally autogenous EC-seeded, anastomotic retainer-armed, knitted Dacron and PTFE grafts are the best shunting materials in venous reconstructive surgery.

Acknowledgment. This work is partially supported by the Chinese Natural Sciences and Technology Foundation. Prostheses are partially provided by the Gore-Tex, Meadox, and Vascuteck companies.

References

1. Budd G. Diseases of the liver. Churchill, London, 1845:147;1857:194–196.
2. Chiari H. Ueber die selbstandige Phlebitis obliterans der Haupstamme der venae hepaticae als Todesursache. Beitr Path Anat 1899;26:1–18.
3. Wang ZG. Recognition and management of Budd-Chiari syndrome. Chin Med J 1988;102:338.
4. Zhang HD, Wang ZG. Nomenclature, classification, and gradation of hepatophlebo-vena caval occlusive syndrome. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari syndrome. Jinan, China, 1988:127–130.
5. Kimura C, Shirotani H, Hirooka M, et al. Membranous obliteration of the inferior vena cava in the hepatic portion. J Cardiovasc Surg 1963;4:87–98.
6. Ohara I, Ouchi H, Takahashi K. A bypass operation for occlusion of the hepatic inferior vena cava. Surg Gynecol Obstet 1963;117:151–155.
7. Hirooka M, Kimura C. Membranous obstruction of the hepatic portion of the inferior vena cava. Arch Surg 1970;100:656–663.
8. Victor S, Jayanthi V, Kandasamy I, et al. Retrohepatic cavoatrial bypass for coarctation of inferior vena cava with polytetrafluoroethylene graft. J Thorac Cardiovasc Surg 1986;91:99–105.
9. Yamamoto S, Yokoyama Y, Takeshige K, et al. Budd-Chiari syndrome with obstruction of the inferior vena cava. Gastroenterology 1968;54:1070.
10. Putnam CW, Porter KH, Weil R, et al. Liver transplantation for Budd-Chiari syndrome. JAMA 1976;236:1142.
11. Akita H, Sakoda K. Portopulmonary shunt by splenopneumopexy as surgical treatment of Budd-Chiari syndrome. Surg 1980;87:85.
12. Cameron JL, Herlong HF, Sanfey H, et al. The Budd-Chiari syndrome. Treatment by mesenteric-systemic venous shunt. Ann Surg 1983;198:335.
13. Senning A. Transcaval posterocranial resection of the liver as treatment of the Budd-Chiari syndrome. World J Surg 1983;7:632.
14. Eguchi S, Takeuchi Y, Asano K. Successful balloon membranotomy for obstruction of the hepatic portion of the inferior vena cava. Surg 1974;76:837–840.
15. Yamada R, Sato M, et al. Segmental obstruction of the hepatic inferior vena

cava treated by transluminal angioplasty. Radiology 1983;149:91–96.
16. Franco D, Vons C, Lecomte Y, et al. Portoatrial shunt in Budd-Chiari syndrome. Surg 1986;99:378.
17. Mishima Y. Current status of Budd-Chiari syndrome in Japan. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome . Jinan, China, 1988:6–10.
18. Sun YQ, Xhu AL. Surgical treatment of Budd-Chiari syndrome through the thoraco-retroperitoneal route. Chin J Surg 1982;20:107 (Chinese).
19. Wang ZG, Li GD. Current status of Budd-Chiari syndrome in China. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:13–17.
20. Simpson IW. Membranous obstruction of the inferior vena cava and hepatocellular carcinoma in South Africa. Gastroenterology 1982;82:171–178.
21. Wang ZG. Congenital defects of the inferior vena cava causing Budd-Chiari syndrome (personal experience in 50 cases). In: Belov S, Loose DA, Weber J (eds.), *Vascular Malformation*. Ehnhorn-Press Verlag. Hamburg, 1989:138–145.
22. Wang ZG, Chu Y, Wang SH, et al. Recognition and management of Budd-Chiari syndrome: Report of one hundred cases. J Vasc Surg 1989;10:149–156.
23. Wang ZG. Experience on management of Budd-Chiari syndrome in 143 cases. Angio Arch 1989;17:147.
24. Sen PK, Kinare SG, Kelkar GB, et al. Congenital membranous obliteration of the inferior vena cava: Report of a case and review of literature. J Cardiovasc Surg 1967;8:344–357.
25. Kilman JW, Williams TE, Kekos GS, et al. Budd-Chiari syndrome due to congenital obstruction of the hepatic portion of inferior vena cava. J Thoracic Cardiovasc Surg 1971;62:226.
26. Wang ZG. Membranous obstruction of the inferior vena cava. Proc CAMS & PUMC 1987;2:76.
27. McDermott WV, Stone MD, Bothe A, et al. Budd-Chiari syndrome: Historical and clinical review with an analysis of surgical corrective procedures. Amer J Surg 1984;147:463–467.
28. Panchanadam M, Jayanthi V, Vijayalakshmi CS, et al. Chronic Budd-Chiari syndrome due to filariasis. Indian J Thorac Cardiovasc Surg 1988;7:6.
29. Ahn S, Yellin A, Shong FC, et al. Selective surgical therapy of the Budd-Chiari syndrome provides superior survivor rates than conservative medical management. J Vasc Surg 1987;5:28.
30. Ualla D, Le MG, Poynard T, et al. Risk of hepatic vein thrombosis in relation to recent use of oral contraceptives. Gastroenterology 1986;90:807–811.
31. Das M, Carroll SF. Antithrombin III deficiency: An etiology of Budd-Chiari syndrome. Surg 1985;87:242–245.
32. Valla D, Casa DN, Lacombe C, et al. Primary myeloproliferative disorders and hepatic vein thrombosis. Ann Internal Med 1985;103:329.
33. Disney TE, Sullivan SN, Haddad RG, et al. Budd-Chiari syndrome with inferior vena cava obstruction associated with systemic lupus erythematosus. J Clin Gastroenterol 1984;6:253–256.
34. Bras G, Jeliffe DE, Stuart KL. Veno occlusive disease of the liver with nonportal type of cirrhosis, occurring in Jamaica. Arch Pathol 1954;57:285–300.
35. Wang ZG, Du W. Hemodynamic change in reconstructive surgery for Budd-

Chiari syndrome. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:36–40.
36. Wang ZG, Du W, Li GD, Pu LQ, Sharefkin JB. Rapid cellular luminal coverage of Dacron inferior vena cava prostheses in dogs by immediate seeding of autogenous endothelial cells derived from omental tissue: Results of a preliminary trial. J Vasc Surg 1990;12:168–179.
37. Li GD, Wang ZG, Wu JD, et al. Improvement of patency and inhibition of neointimal hyperplasia of vena caval Dacron prosthesis by seeding of microendothelial cell derived from omentum in canine. (To be published in *Modern Vascular Surgery*, John B. Chang, ed.)
38. Wang ZG, Wu JD, Li GD, et al. Microvascular endothelial cell seeding to the dog vena caval prostheses made by different materials and configurations. (Submitted.)
39. Zhang CS, et al. Clinical research on balloon dilatation of inferior vena caval web. (To be published.)
40. Bai YQ, Wang ZG, Wang F, Xu WC. Pathologic changes of the liver in Budd-Chiari syndrome in 45 cases. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:206–210.
41. Wang ZG, Chen SL, Lui M, Wang SH, Wu W. Comparison of ultrasonography to cavography in diagnosis of Budd-Chiari syndrome in 80 cases. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:156–158.
42. Wu W, Zhang AP, Wang ZG. The significance of ultrasonography in Budd-Chiari syndrome: Analysis of 51 cases. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:214–218.
43. Wang ZG. Budd-Chiari syndrome: Report of 50 cases. Natl Med J China 1987;67:72 (Chinese).
44. Wang ZG, Wang SH. The significance of percutaneous transhepatic hepatovenography in the diagnosis and treatment of Budd-Chiari syndrome. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:162–167.
45. Wang ZG, Zhong Chen. Preoperative management and postoperative care for patients with Budd-Chiari syndrome. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988.
46. Young XL. Membranous obstruction treated by transluminal balloon catheter dilatation. Personal communication.
47. Furui S, Yamauchi T, Kuni O, et al. Hepatic inferior vena cava obstructions: Clinical results of treatment with percutaneous transluminal laser-assisted angioplasty. Radiology 1988;166:673–677.
48. Lois JF, Hartzman S, McGlade CT, et al. Budd-Chiari syndrome: Treatment with percutaneous transhepatic recanalization and dilation. Radiology 1989;170:791–793.
49. Wang ZG. Treatment of Budd-Chiari syndrome by mesenteric or spleno-atrial shunt: Report of 18 consecutively successful cases. Chin J Thorac & Cardiovasc 1988;4:17 (Chinese).
50. Xu PQ, Ye XX, Qin BM, et al. Spleno-atrial shunt as a new surgical treatment of Budd-Chiari syndrome. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:41–43.

51. Chapman JE, Ochsner JL. Iliac-mesenteric-atrial shunt procedure for Budd-Chiari syndrome complicated by inferior vena cava thrombosis. Ann Surg 1978;188:642.
52. Liu ZY, Wang ZG. Initial investigation of epidemiology of Budd-Chiari syndrome. Vascular Surgery. Proceedings of the First International Symposium on Budd-Chiari Syndrome. Jinan, China, 1988:73–74.

39
Budd-Chiari Syndrome—Pathogenesis and Treatment

KUO-HUA ZHANG and ZHUO-YUN GU

Summary

Occlusion of hepatic venous outflow due to obstruction of inferior vena cava (IVC) is the major problem to be solved for Budd-Chiari syndrome in China. Sixty patients with such an entity had been admitted in our hospital since 1964 to 1990, and 58 cases had changes of IVC. Research of autopsy in six cases showed that congestion of liver and sequential cirrhosis might be reversible (similar to the changes in congestive heart failure), and since medication is intractable, surgery is the treatment of choice. Our study included three categories of IVC obstruction, according to cavovenography, i.e., stenosis of IVC (Type I), occlusion of IVC at hilum of diaphragm (Type II), and extended obliteration of IVC (Type III). Forty-three cases were treated surgically, with three kinds of operation for the three Types: 1) portosystemic shunt was performed in two patients with Type I; 2) cavoatrial, azygoatrial, and mesoatrial bypass procedures were used for Type II in 14, 1, and 1, respectively; 3) splenopneumopexy was elected for 15 patients with Type III. Eight patients had thoracic duct and internal jugular vein anastomosis to manage ascites (including one patient who also had a shunt procedure in the second stage), and two had exploration only. Angioplasty (PTA) was done successfully in one case with web obstruction. There was no operative mortality and seldom any morbidity, other than a short term of hydropneumothorax after bypass procedure. Follow-up studies of two months to 19 years (most were over years) showed that a shorter graft for bypass procedure had a better treatment result with cavoatrial shunt through right thoracotomy. All but one of the patients lived well.

Introduction

Budd-Chiari syndrome is a rare condition, its etiology obscure, and elective treatments are numerous. Five hundred such cases were collected from

the world literature by Orloff in 1978.[1] Obstruction of inferior vena cava accounted for 238 (45.4%) of cases, however, this did not include cases from the Chinese literature. As a result of our studies of this condition and its special characteristics, we would like to introduce our experiences with this syndrome and to discuss its characteristics in China, especially pathogenesis and treatment.

Materials and Method

Since 1964 to 1990, 60 cases of Budd-Chiari syndrome (not including conditions originating from tumors) were admitted in our hospital. There were 46 males and 14 females, and age ranged from 8 to 56 with a mean of 31.6 years.

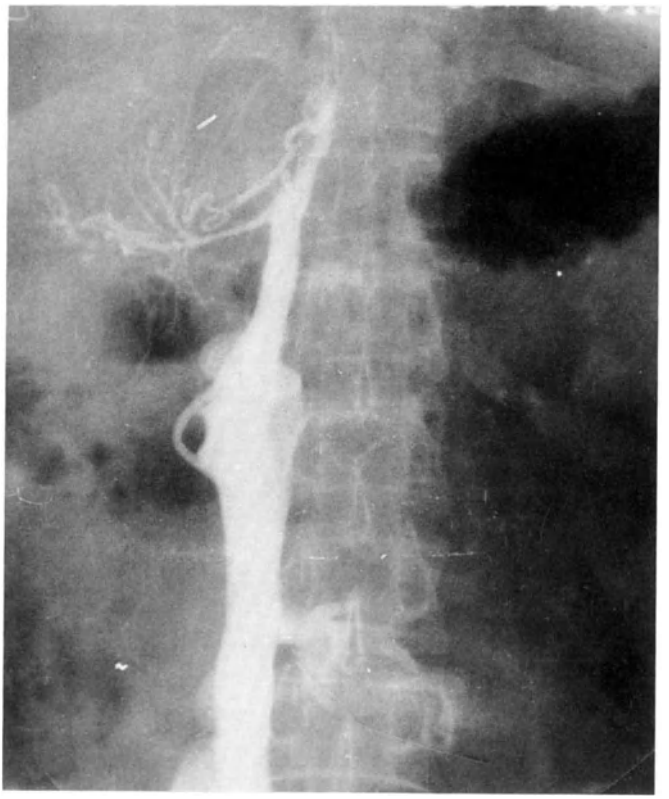


FIGURE 39.1. Type I stenosis of inferior vena cava is presented.

All diagnoses were confirmed by symptoms and signs of hepatomegaly, esophagus varicosis, venous varicosis of abdominal wall and trunk, ascites, splenomegaly, edema of the lower extremities with venous varicosis of legs, and sometimes pigmentation. Cavovenography and CT scan and/or ultrasound were performed in most patients, and additionally, six autopsies showed that four of the six had stenosis, obstruction, or obliteration of hepatic inferior vena cava. Cavovenography and wedged hepatic venography were done in our surgical group for diagnosis as well as selection of procedure. Four categories were found:

1. Hepatic veins obliterated without changes of inferior vena cava in two cases.
2. Partial obstruction or stenosis of inferior vena cava appeared at the diaphragm hilum or extended and regular narrowing of hepatic inferior vena cava in 20 cases (Fig. 39.1).

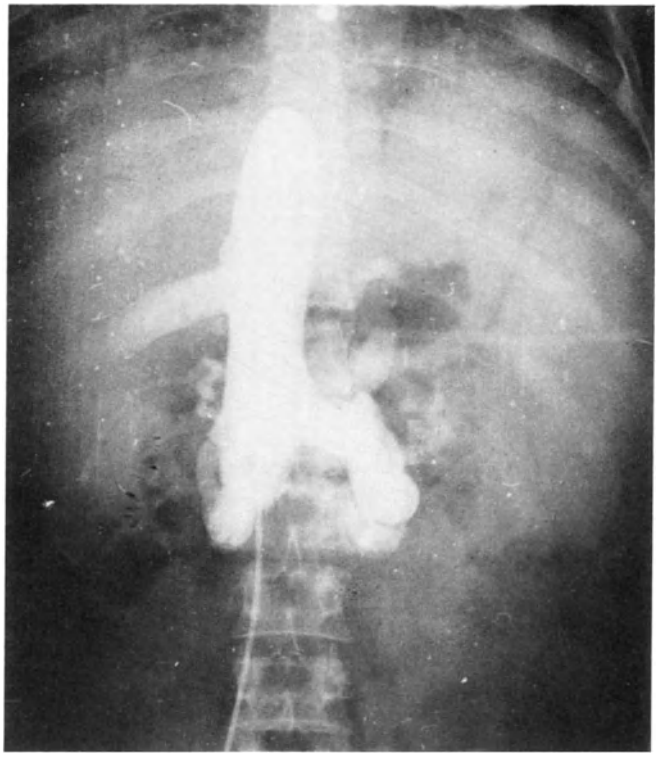


FIGURE 39.2. Type II occlusion of inferior vena cava found at the second hepatic helium.

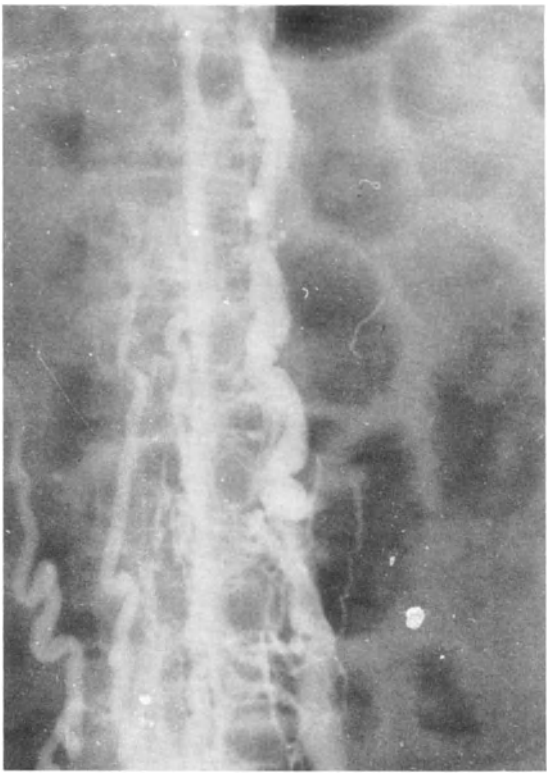


FIGURE 39.3. Type III. Besides collateral vein, inferior vena cava is totally obliterated.

3. Occlusion or membranous obliteration of inferior vena cava found at the diaphragm in 16 cases (Fig. 39.2).
4. Extended or total inferior vena cava obliterated in 6 cases (Fig. 39.3).

Etiology of the disease could not be clearly described. Some factors might be responsible for pathogenesis—one patient had symptoms and signs immediately the day after intravenous pyelography, with high pressure on his abdomen. Chemotherapy might have been another factor that induced obliteration of inferior vena cava in a patient treated for lymphoma. Unfortunately, no evidence could be found to support the diagnosis of tumor, and finally he had retroperitoneal fibrosis due to changes in the inferior vena cava. A couple of patients had a history of thrombophlebitis of the lower extremities, and the syndrome appeared following an insidious period.

Pathology

Six cases were found to be Budd-Chiari syndrome due to benign diseases in autopsies performed in our hospital from 1973 to 1980. All of them had intimal thickening and fibrogenesis of hepatic veins and inferior vena cava, the lumen became stenosed or obliterated. Chronic congestion hepatic cirrhosis, with varicosis of the esophogus and stomach, and congestive splenomegaly where revealed in all six cases.

Thrombus was found in the inferior vena cava of two patients. One of these cases was described as having the nutmeg liver seen with congestive heart failure; the liver was enlarged, congested, and fatty and its cut surface bulged and was greasy to the touch. The other five cases had enlarged or atrophied cirrhotic livers, with marked congestion and with or without necrosis.

Hepatic sinuses were dilated microscopically. A fibrous replacement was seen in the destroyed central zones of the lobules, and necrosis was seen in most areas, or the central vein was narrowed because of the thickened wall. Congestion associated with hemorrhage also could be seen around the central vein.

Changes to the hepatic vein and inferior vena cava were seen as veno-occlusive diseases, which underwent progressive intimal fibrosis and thrombosis.

In 9 biopsied cases involving degenerative inferior vena cava and liver disease, the changes noted were similar to those seen in the autopsied cases.

Treatment

Seventeen of the 60 Budd-Chiari syndrome cases were treated medically, with on favorable response, and six of these died in a few months or years of hepatic failure or massive GI bleeding. Forty-three cases were operated on using the following procedures: portosystemic shunt in 2 patients; cavoatrial, azygoatrial, and mesoatrial bypass procedures in 14, 1, and 1, respectively; splenopneumopexy in 15 cases; 8 thoracic duct and internal jugular vein anastomoses including one who also had a shunt procedure; and angioplasty was done in 1 patient. Additionally, there were 2 exploratory operations.

Results

There was no operative mortality and seldom any morbidity, other than a short term of hydropneumothorax after bypass procedure. Follow-up study from 2 months to 19 years (the majority over a year) showed that the best

procedure was the cavoatrial shunt with a short graft bypass procedure through a right thoracotomy. The longest follow-up included 14 patients who underwent this procedure and who are alive and well 9 years later. The other 11 are also in good condition over 1, 2 and 3 years, although 1 patient has recurring symptoms due to a kink in the graft. This was an operational technique defect seen in the early 1980s. A long graft through a tunnel behind the liver between the inferior vena cava and below the renal vein and right atrium was done in 2 patients in the 1960s. One lived 19 years postoperatively with a recurrence of symptoms 6 years after the operation. Another died 8 years postoperatively when the graft was collapsed by a huge liver; there was no patency at all.

One patient who had azygoatrial bypass, who was followed up for 2 years, is still in good condition. The mesoatrial bypass of 1 patient did not correct the symptoms and failed a year and a half later due to graft thrombosis. The results of portosystemic shunt (portacaval in 1 and splenorenal shunt in 1) and splenopneumopexy were observed in several patients, all of whom feel better than before, with the huge acites disappearing in 2 cases. Thoracic duct and internal jugular vein anastomoses for this disease were not satisfactory in 8 cases, it seems this can be alleviated only in patients suffering ascites for shorter periods. PTA to treat web obstruction in 1 case had encouraging results.

Discussion

Budd-Chiari syndrome is uncommon clinically, but it seems to occur more frequently in oriental populations. Most reports on this syndrome come from eastern China in the Chinese literature, especially in Shandong, Henan and Hebei provinces. In the English literature, it is usually presented as case reports in western countries. Although etiology of this disease is not clear, it is commonly associated with obstruction of the inferior vena cava. In Chinese literature, there were 139 cases collected until November 1987.[2–8] with the exception of 4 cases, all had obstruction of the inferior vena cava. Many factors might contribute to the disease, such as thrombophlebitis, contraceptive pills and polycythemia, listed in the literature. We found that trauma and chemotherapy might also contribute to the disease. Congenital factors may play an important role in formation of the disease, since the inferior vena cava is formed as building blocks in the embryo, and most patients are younger than 30 years old.

Autopsy results showed that major changes to be obstructed outflow of the hepatic vein and hepatic congestion similar to the changes of congestive heart failure. Since these changes are reversible, early management could lead to better results.

Mechanical obstruction of the blood stream from the inferior vena cava to the atrium and sequential portal hypertension are the major problems to be solved. The clinical aspects are: abdominal distension, venous varicosis

at trunk, ascites, edema with or without pigmentation of the lower extremity, hepatomegaly, splenomegaly, etc. Laboratory findings were not significant, other than hepatic dysfunction and hypersplenism. The most useful imaging procedure is cavovenography. According to the level, degree and extent of the obstruction, many surgical procedures have been designed and performed as reported in the literature. Basically, procedures are aimed at decreasing either the elevated pressure of the inferior vena cava or portal hypertension, such as membranotomy, cavo-, meso- or splenoatrial shunt, portocaval shunt, etc. However, in the case of extended or total inferior vena cava obliteration, splenopneumopexy could be used.

Two pathways of cavoatrial shunt may be chosen, pre- or posthepatic bypass. A graft between the inferior vena cava and the right atrium through a tunnel behind the liver (it is easily compressed by a huge liver) or a prehepatic one needs to be quite long. It would be better to use a short graft between the hepatic and the inferior vena cava below the obstructive site and right atrium. Mesoatrial, splenoatrial and portoatrial shunt could be selected for those patients without leg edema. Membranotomy is the treatment of choice for those with inferior vena cava web only.

On the basis of data in this study, we conclude that:

1. Obstruction of the inferior vena cava leading to occlusion of hepatic venous outflow is the major problem evolving from Budd-Chiari syndrome in China, with surgery the treatment of choice.
2. Cavovenography is the most useful imaging procedure for diagnosis, especially for showing the site, extent and degree of obstruction of the disease for selection of surgery.
3. This entity may be divided into 4 groups according to the situation of obstruction: obstruction of hepatic veins only, partial obstruction or stenosis of inferior vena cava around the diaphragm, occlusion of inferior vena cava at the hilum of diaphragm, and extended or total obliteration of inferior vena cava.
4. Many procedures might be selected, but no one procedure is suitable for all circumstances. Generally speaking, a rational choice of surgical procedure for this disease is a bypass shunt.
5. A shorter graft for bypass procedure leads to better treatment results and a cavoatrial shunt through a right thoracotomy is recommended.
6. Membranotomy or PTA may be used for those with inferior vena cava web only.
7. In those cases not suitable for shunt procedure, the alternative is splenopneumopexy.

References

1. Orloff MJ, Johansen KH. Treatment of Budd-Chiari syndrome by side-to-side portocaval shunt: Experimental and clinical results. Ann Surg 188:494, 1978.
2. Gu ZY et al. Budd-Chiari syndrome—A report of 8 cases. Chinese J Surg 19:754, 1981.

3. Zhang, KH. Occlusive disease of inferior vena cava—A report of 32 cases. In press.
4. Wang ZG. Recognition and management of Budd-Chiari syndrome—Report of 70 cases. International Symposium on Current Practice in Vascular Surgery in Beijing, 1987.
5. Treatment of the Budd-Chiari syndrome with prehepatic graft bypass shunt in 14 cases, Ibidem, 1987.
6. Sun KX, et al. Surgical treatment of Budd-Chiari syndrome—A report of 8 cases. Chinese PLA J Med 6:485, 1985.
7. Sun KX, et al. Surgical treatment of Budd-Chiari syndrome—A report of 13 cases. Chinese J Surg 25:520, 1987.
8. Sun YQ Sun. Cavotrial shunt for the treatment of Budd-Chiari syndrome. Chinese J Surg 20:107, 1982.

40
How Well Does the Informed Consent Inform?

STEVEN P. SCHMIDT and FREDERICK I. FIELD

Abstract

Obtaining the informed consent can be especially difficult in vascular surgery patients where the operative procedure is complex and the patients are often elderly. We interviewed 90 vascular surgery patients regarding their ability to recall key information about the surgical procedure and possible complications. The consent material had been presented to the patient and family members in detail by the attending and/or resident surgeons in the form of an oral discussion. When asked if they were well informed and understood the surgery, 97% answered in the affirmative, yet only 56% of patients had an adequate knowledge of basic, simple questions regarding their surgery. Eighty-seven percent of patients felt they were well informed of the risks and possible complications, yet only 9% had an adequate knowledge about possible major risks and complications of their surgery. Furthermore, only 26% wished that they understood their surgery better, and only 25% desired more knowledge of the possible risks and complications. This raises questions whether the average vascular surgery patient understands the information given during consent, and whether they consciously or unconsciously suppress the information about operative risks and complications.

Introduction

The educational value of the informed consent has been a subject of considerable debate in recent years. It can be questioned whether the average patient has the background knowledge necessary to understand these aspects of a surgery: why the operation is necessary, what basic operative procedures will be performed, and what the potential risks and complications are. The informed consent can be especially difficult in vascular surgery patients where the operative procedure is complex, prosthetic materials are often used, and the majority of patients elderly. To study this issue we surveyed our vascular surgery patients in the following manner.

Materials and Methods

We interviewed those patients undergoing major vascular surgery during a 15-month period at the Jerry L. Pettis Memorial Veterans Administration Hospital. Excluded from the study were those patients who: 1) could not be located, 2) had expired, 3) were too deaf or senile to be interviewed, or 4) refused to be interviewed. Major vascular surgery was defined as those patients having one of the following operations: 1) carotid endarterectomy, 2) aortic surgery, 3) lower extremity vascular reconstruction, and 4) miscellaneous major vascular reconstructions. Prior to each operation the procedure and its major complications were explained in detail with an oral discussion by the attending surgeon and/or chief resident.

Ninety patients were contacted and interviewed. The following questions were asked in this order:

1. Name.
2. Social security number.
3. Type of surgery.
4. Date of surgery.
5. a. "Who explained the procedure to you? chief resident? attending?"
   b. "Did you feel you were well informed about the surgery?"
   c. "Did you feel you were well informed of the possible risks and possible complications?"

Questions regarding the surgery:

6. a. "Did they operate on an artery, vein, or both?"
   b. "Did they bypass, repair, or replace the vessel?"
   c. "What material was used?"
   d. "Does an artery take blood away from or toward the heart?"

Questions regarding complications:

7. "What are the potential complications of this operation?"

Questions regarding patient satisfaction with the information received in the consent process:

8. a. "Do you wish you understood the operation better?"
   b. "Do you wish you knew more about the risks and potential complications?"
9. "Did you experience a complication?"

Acceptable answers for question 7 (regarding risks and possible surgical complications are listed in Table 40.1.

Adequate knowledge regarding the operation was defined as correctly answering three or more parts on question 6. Adequate knowledge of complications was defined as accurately identifying 60% of the possible responses as defined in Table 40.1.

TABLE 40.1. Complications.

Type of surgery	Possible complications
Carotid	stroke, M.I., bleeding, infection, restenosis
Aorta	M.I., bleeding, infection, renal failure, leg ischemia, impotence, lung problems
Lower extremity	M.I., occlusion, infection, bleeding, amputation

Results

All patients were male and 50% (45/90) were over age 65 (Table 40.2). There were 21 carotid endarterectomies, 26 aortic operations, 31 lower extremity revascularizations and 12 miscellaneous major vascular procedures (Table 40.3). Ninety-six percent (86/90) of the patients answered that they felt "well informed about the operation," but only 56% of the patients had an adequate knowledge of the surgical procedure. Similarly, 88% (79/90) of the patients considered themselves "well informed about the surgical risks and complications," but only 10% (9/90) had an adequate knowledge of the risks and possible complications (Table 40.4).

TABLE 40.2. Demographic data.

% men	100% (90/90)
age range	40–82
<45	1% (1/90)
45–65	49% (44/90)
>65	50% (45/90)

TABLE 40.3. Types of surgery performed.

Carotid endarterectomy	23% (21/90)
Aortic surgery	29% (26/90)
Lower extremity	35% (31/90)
Miscellaneous	15% (12/90)

TABLE 40.4. Comparison of patients' perception of being informed and their performance of the basic test.

Patients who felt "well informed about the operation"	96% (86/90)
Patients with an adequate knowledge of the operative procedure	56% (50/90)
Patient "well informed of potential risks/complications"	88% (79/90)
Patients with an adequate knowledge of the risks/complications	10% (9/90)

TABLE 40.5. Time interval after surgery vs. score.

Months	N	% of correct answers regarding surgery	% of correct answers regarding complications
>9	16	53	12
7–9	6	79	32
4–6	14	71	22
0–3	54	69	28
Mean score		67	24

After being asked these questions about the surgical procedure, 27 patients stated that they wished they understood their surgery better. The average score of these patients was 63% as compared to 67% for those patients who felt they understood their operation adequately. In a similar comparison, 26 patients stated that they wished they had understood the risks and complications better. These patients correctly answered an average of 14% of the complication questions as opposed to a score of 24% for those patients who felt they understood the risks and complications. Table 40.5 shows the time interval between surgery and the interview vs. score.

Discussion

The informed consent of patients to surgical treatment has been a subject of concern within the medical and legal community as early as the 1900s.[1] At first the issue was whether any consent should be obtained. Then later, the importance of the quality of the informed consent was emphasized. The legal community originally required physicians to provide their patients with similar information to that which other physicians were providing—the "community of physicians' peers" standard.[2] This later evolved into the consent, including those facts that "a reasonable man would want to know."

From our data only 56% of the patients had an adequate understanding of the operative procedure, but 96% felt they were well informed! This obvious discrepancy may be due to our technique of interviewing the patients, but is more likely due to the patients consciously or subconsciously ignoring the facts. Especially if the operation and recovery were uncomplicated, the patient has no stimulus to recall what was done. That patients selectively listen is further demonstrated by the large discrepancy between 88% of the patients who thought they were well informed about the complications and the 10% who had an adequate knowledge of the complications. Learning about the possible risks and complications of an operation is not a pleasant task and this information was undoubtedly suppressed or just ignored.

Several studies have examined the informed consent process. Alfidi[3] studied the effects of disclosure on angiography patients. He found an overwhelming majority of patients consented to the procedure following comprehensive written and oral explanation, however, a third of patients admitted to being disturbed by this information. Cassileth and associates[4] of the University of Pennsylvania Cancer Center examined reasons why 200 cancer patients could not recall major portions of their consent form or discussion. They found three factors associated with poor recall: education level, medical status, and the care with which the consent was read.

Conclusion

Should the consent process be changed? Mazur[1] has defined the negative aspects of informed consent to be: "overloading patients with detailed information they may not want, presenting patients with numerical information they may not understand, and patients' lack of an overall framework to compare the risks of the procedures they are facing now with other risks in their own life (relative risk). The goal of increasing a patient's understanding of the procedure, the alternatives, and complications should be maintained. Possible methods of improving patients' understanding and recall include drawing diagrams, writing key points, and including other family members in the discussion. Since the informed consent is now an integral part of patient care in the United States, the limitations of both the patient and the physician in achieving this goal need to be recognized.

References

1. Mazur DJ. What should patients be told prior to a medical procedure? Am J Med 1986;81:1051–1054.
2. Kaufman CL. Informed consent and patient decision making: two decades of research. Soc Sci Med 1983;17:1657–1664.
3. Alfidi RJ. Informed consent: a study of patient reaction. JAMA 1971;216:1325–1329.
4. Cassileth BR, Zupkis RV, Sutton-Smith K, et al. Informed consent—why its goals are imperfectly realized. NEJM 1980;302:896–900.